# Overuse Tendonitis and Rehabilitation

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**SUMMARY**

**Tendon injuries are often caused by overuse during sport or day-to-day activities. Knowledge of these injuries has grown greatly during the last several decades. We review recent literature on the structure and mechanical properties of tendons, the cause of overuse injury, and rehabilitation.**

**RÉSUMÉ**

**L'abus d'activités quotidiennes ou sportives peut souvent causer des blessures tendineuses. Depuis les dernières décennies, on assiste à un développement marqué des connaissances entourant ces blessures. Les auteurs passent en revue la littérature récente sur la structure et les propriétés mécaniques des tendons, la cause des blessures par surutilisation et la réadaptation.**

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Increased attention to healthy lifestyles during the last several decades has led Canadians to spend more time participating in sport and physical recreation. More individuals are active well into their adult years, and some have taken up exercising again after many years of relative inactivity. Consequently, family practitioners are noticing a large increase in the number of overuse injuries they treat. These physicians are seriously concerned about preventing and appropriately treating such injuries.[1,2]

Overuse injuries occur when the body cannot adapt to the cumulative stress of a repetitively applied force.[3] Depending on the duration and severity of symptoms, patients with overuse injuries might have to give up recreational pursuits or even alter their daily activities. Overuse injuries can affect many tissues and anatomic sites, but all share common origin: repetitive trauma that overwhelms the ability of tissue to repair itself.[4]

Researchers estimate that 50% of all sport injuries are due to overuse. The tissue most commonly affected is a musculotendinous unit.[2,5,6] Injury can occur anywhere along the unit: in the muscle belly, in the tendon, at the muscle-tendon junction, or where the tendon enters the bone. However, during physical activity, stress and force are focused on the tendon.

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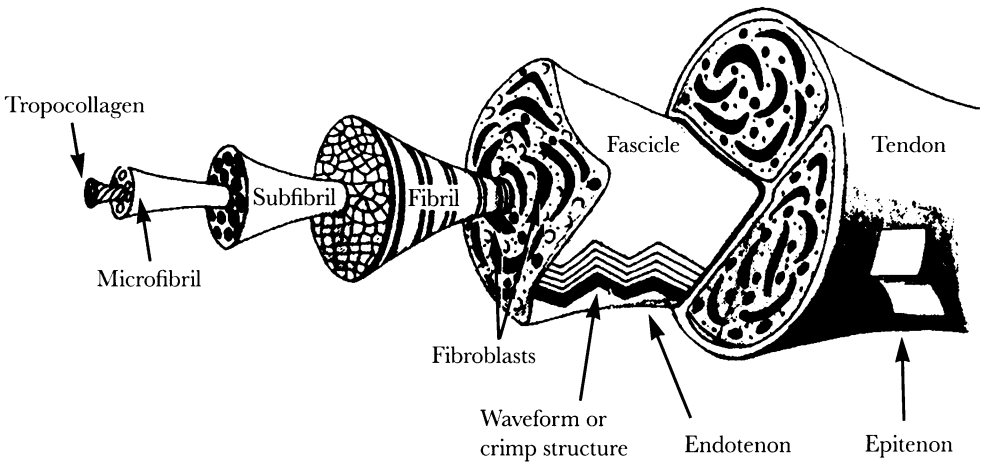
## Structure and anatomy

Tendon is extremely strong; its tensile strength is 49 to 98 N/mm$^2$.[7] Tendons are composed of 30% collagen and 2% elastin embedded in an extracellular matrix containing 68% water. Collagen, which accounts for 70% of the dry weight, is the main structural component of tendon. Collagen resists the force applied to tendons and has a breaking point similar to that of steel.[8]

Elastin helps make tendons flexible. Elastin can stretch up to 170% of its initial resting length in some circumstances without rupture. Collagen, however, can elongate only 4% before it fails. The ground substance is a gellike material composed of proteoglycans and water that surrounds the collagen fibres. It provides structural support and a medium for diffusing nutrients and gases. The ground substance

*Figure 1.* **Hierarchial organization of a tendon.**

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allows the collagenous proteins to aggregate into the shape of fibrils.[8]

Collagen, elastin, and ground matrix are all produced by fibroblasts and are organized in a complex hierarchical manner to form the tendon proper.[3] Fibroblasts are found throughout the tendon among collagen bundles. They are motile and highly proliferative, increasing in number during injury repair. The structural unit of collagen is tropocollagen, and researchers think that five soluble tropocollagen units cross-link to form microfibrils. These insoluble molecules aggregate into more progressively definable groupings (*Figure 1*[3]).

Fibrils are the basic units of a tendon; several parallel fibrils surrounded by the matrix are known as fibres. Fibres group to form fascicles, which are surrounded by the endotenon, a sheath of loose alveolar connective tissue. Blood vessels, nerves, and lymphatic vessels are carried in the endotenon, which allows movement between individual fibres. The fascicles are enclosed by the epitenon, a fine connective tissue sheath surrounding the entire tendon. The paratenon is the outermost sheath. A small amount of fluid is found between the paratenon and epitenon, preventing friction and damage to the tendon. In areas of increased friction, a complex synovial sheath replaces the paratenon.

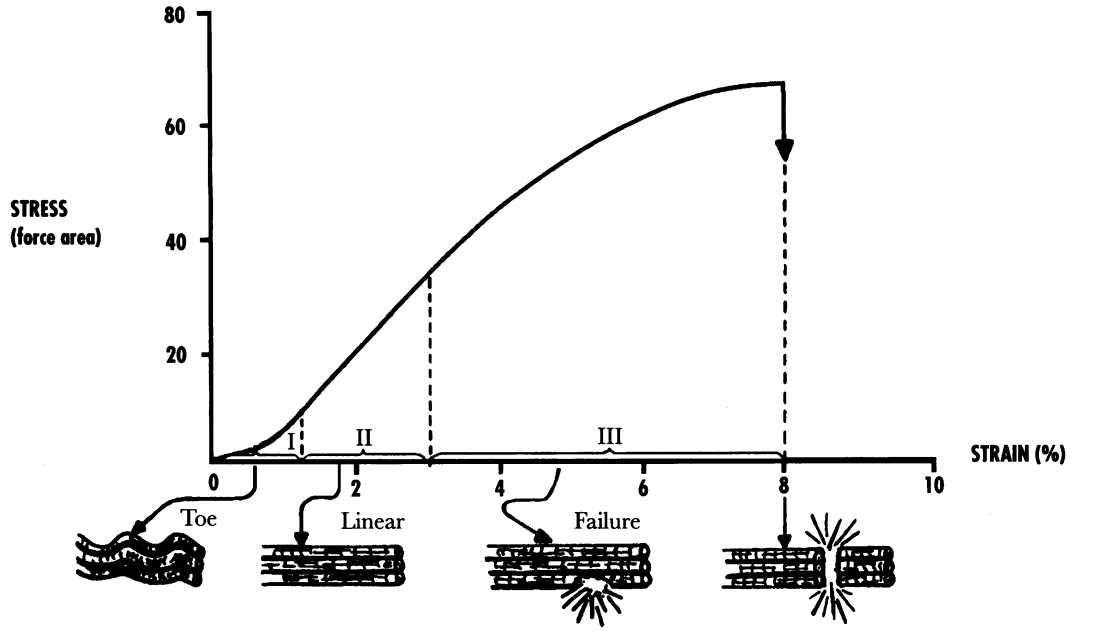
Tendon is a well vascularized tissue, although less vascularized than muscle and skin. A tendon receives its blood supply three ways: through the musculotendinous junction, along the length of the tendon, and at the bone-tendon junction. However, the vasculature of the tendon is variable and inhibited in areas of friction, compression, or wear. This poor perfusion can contribute to the development of overuse syndromes.[3,8]

## Mechanics and function

The mechanical behaviour of tendon depends on its structure. Because a tendon's function is to transfer the force of muscle to bone, it is subjected to great tensile stress. A high collagen content makes it very strong and capable of elongating and returning to its original state. However, tendon is less able to resist shear and compressive forces than isometric tension or strain. Tendons must be flexible to allow unhindered movement and to maintain the ability to redirect forces "around corners."[3]

At rest, tendons have a wavy configuration that disappears when they are stretched 2% beyond resting length. Resting collagen fibres are crimped, so the first response to tensile forces is straightening. This toe region of the stress and strain curve is where the elastic properties of the collagen are tested (*Figure 2*[3]). In the second part of the curve, collagen fibres elongate and straighten. The tendon will return to its original length as long as it is not stretched more than 4%.[3]

*Figure 2.* **Typical stress-strain curve of a tendon.**

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Its size and the number of fibres it contains will determine the force the tendon can withstand. At 4% to 8% strain, the collagen fibres slide past one another as cross-links start to fail. As cross-links are broken, the weakest fibres rupture at microscopic level. In the fourth part of the curve, macroscopic failure occurs because of tensile failure of the fibres and shear failure between the fibres. Although most physiological forces or loads are reported to cause less than 4% strain,[7] certain activities, especially in sport, can exceed this level and lead to macroscopic injury.[9]

## Mechanism of injury

Tendon is susceptible to both excessive tensile loads and compressive forces. Injury represents a failure of the cell matrix to adapt to load exposure, which can be either acute or secondary to cyclic overuse. Thus tendon injuries can be divided into two broad categories according to rate of onset and mechanism: macrotraumatic (acute tissue destruction) and microtraumatic (chronic overuse).

Although Elliott[7] stated that tendons in vivo are never stressed more than one quarter of their ultimate strength, the literature has reported greater stresses.[9,10] Furthermore, repetitive strain can result in cumulative microtrauma that weakens the structural and vascular elements. Once the ability of tendon tissue to repair itself is outpaced by the repetition of insult, overuse injury occurs.[3]

Chronic microtrauma is associated with subclinical injury or damage that has been accumulating for an extended time before the moment of conscious insult. The gradual, degenerative nature of the chronic overload indicates that overt clinical symptoms are the result of a process, rather than an acute event. This is distinct from acute injury, where the onset of symptoms and injury occur simultaneously and treatment can be initiated early in the process. In microtraumatic injuries, an accumulation of repetitive scar adhesions and degenerative changes imply that recovery will be slower.[11]

## Classification

Tendon injuries can range from mild inflammation of surrounding tissue to more extensive structural alteration. Historically, clinicians have used the term tendonitis to describe virtually all painful tendon structures, including synovial sheaths and adjacent bursa. This tradition is currently being challenged by mounting pathological evidence that acute traumatic inflammatory response and the more insidious process of chronic

tendon degeneration are distinct processes.[11] This problem of terminology has caused much confusion in describing the inflamed tendon unit.

Similar observations by Puddu et al[12] and Clancy[13] have led to the development of a new classification of tendinous injury (*Table 1*[11]) that describes four pathological conditions based on the anatomy of the tendon and its surrounding tissues. These authors introduced the term tendinosis to describe an area of intratendinous degeneration that is initially asymptomatic. Thus tendon disease can manifest as paratenonitis, tendinosis, or a combination of the two. Most overuse injuries are thought to fall into the paratenonitis with tendinosis category, because microscopic examination reveals degenerative changes in the tendon and increased inflammatory infiltrates and fibrosis in the paratenon.[11]

These pathophysiological classifications are useful for appreciating different microscopic alterations caused by injury, but might be impractical for clinical diagnosis. However, physicians need to recognize the clinical presentation of the different tendinous injuries in order to be able to recognize mild forms of overuse syndromes and understand the principles of their treatment.

*Table 1.* **Classification of tendon injury**

| TERMINOLOGY | PATHOLOGY | CLINICAL SIGNS AND SYMPTOMS |
|---|---|---|
| Paratenonitis | Inflammation of paratenon only (might be lined by synovium) | Cardinal inflammatory signs: swelling, pain, crepitation, local tenderness, warmth, dysfunction |
| Paratenonitis with tendinosis | Paratenon inflammation associated with intratendinous degeneration (collagen fibre disorientation, hypocellularity, scattered vascular ingrowth) | Same as above, with often palpable tendon nodule, swelling, and inflammatory signs |
| Tendinosis | Noninflammatory intratendinous degeneration due to atrophy (ie, aging, microtrauma, vascular compromise) | Often palpable tendon nodule that can be asymptomatic, but might also be point tender. No swelling of tendon sheath |
| Tendonitis<br>Acute (< 2 weeks)<br>Subacute (4-6 weeks)<br>Chronic (> 6 weeks) | Symptomatic degeneration of tendon with vascular disruption and inflammatory repair response Three subgroups range from purely inflammatory with acute disruption, to inflammation on top of existing degeneration, to calcification and tendinosis in chronic conditions | Symptoms are inflammatory and proportional to vascular disruption, hematoma, or atrophy-related cell necrosis. Symptom duration defines each subgroup |

*Adapted from Leadbetter.*[11]

## Contributing factors

Several intrinsic and extrinsic factors have been suggested as predisposing conditions for developing overuse injuries. Adding the high number of repetitions involved in endurance exercises to the abnormal stresses and loads placed on the soft tissues by intrinsic problems can lead to tissue breakdown and injury.

The role of intrinsic factors in the etiology of overuse injuries (especially those related to malalignment of the lower limb) has been extensively studied.[2,14] Malalignment could be the result of excessive pronation or supination, femoral anteversion, underlying degenerative joint disease, or other structural abnormalities. Leg length discrepancy, muscle imbalances, and poor flexibility have also been cited as factors that predispose individuals to overuse problems.

Combined with the intrinsic mechanical determinants of overuse injury, extrinsic factors contribute significantly.[2] The principle of "transition," as described by Leadbetter,[15] states that sports injury is most likely to occur when the athlete experiences any change in mode or use of the involved part.

Extrinsic factors associated with overuse syndrome arise primarily from inappropriate training and thus are easily corrected once recognized. Training errors (excessive mileage, increased intensity of training, improper technique, or sudden changes in training routines) are a significant cause (up to 75%) of overuse tendon injury.[2,6,14] Improper equipment selection, poor environmental conditions, and unforgiving training surfaces have also been associated with overuse injuries.

In summary, physicians should consider many factors because usually more than one factor is at work. The best approach is to keep an open mind and allow all possibilities, recognizing that an obvious cause could be compensatory or represent only one of many problems.

## How Pain Affects Performance

Pain affects athletic performance. Although pain is a subjective assessment, a classification of tendonitis severity can be a criterion for evaluation. We use the degree of reported pain, assessed both before and after exercise, to determine the efficacy of the exercise program and to assess the rate of progression of the program.

The intensity of the exercise should be such that pain, or discomfort, is experienced in the last set of 10 repetitions. This discomfort indicates that the tendon is slightly overloaded, which is necessary to increase its strength. We have observed that no pain indicates insufficient loading, and so no improvement in symptoms occurs. However, extreme pain, especially throughout the entire 30 repetitions, is a sign that too much force is being applied. This might act to worsen the patient's condition. *Incorrect evaluation of the level of pain or discomfort is the greatest cause of program failure.*

**Classification system for the effect of pain on athletic performance**

| CLASSIFICATION LEVEL | DESCRIPTION OF PAIN | LEVEL OF SPORTS PERFORMANCE |
|---|---|---|
| 1 | No pain | Normal |
| 2 | Pain only with extreme exertion | Normal |
| 3 | Pain with extreme exertion and 1 to 2 hours afterward | Normal or slightly decreased |
| 4 | Pain during and after any vigorous activities | Somewhat decreased |
| 5 | Pain during activity forcing termination | Markedly decreased |
| 6 | Pain during daily activities | Unable to perform |

*Sidebar material reprinted with permission from Curwin and Stanish.*[3]

### Diagnosis

Chronic overuse tendon injuries can present a diagnostic dilemma. The classic signs of inflammation following an acute injury that were defined by Celsus in the often quoted phrase "rubor et tumor cum calore et dobre" (redness and swelling with heat and pain) are not always present or identifiable.[11] Complaints might be vague and nonspecific and performance unaffected in mild cases. Hence diagnosing a patient with an overuse injury must start with an accurate history and an anatomically based, systematic physical examination.

To identify the injured structure it is important to characterize the discomfort and establish etiological factors that might have preceded tissue injury. An awareness of contributing intrinsic and extrinsic factors greatly facilitates diagnosis.

During physical examination, the suspected musculotendinous unit must be isolated, palpated, and stressed. The classic signs of inflammation in addition to tenderness and crepitus might or might not be present. Sound knowledge of the anatomical area involved is important for differentiating overuse injury from other musculoskeletal problems, some of which might accompany the overuse syndromes (eg, bursitis).

Because alterations in strength or biomechanics of other parts of the kinetic change in which the affected tendon participates might occur, the examination must be thorough and include all the potentially affected tissues. Radiographic evaluation is usually not warranted for diagnosing overuse tendon injury.

### Tendon healing

The literature reports extensively on how the acutely traumatized tendon heals, but has little information on how the chronic overuse or nontraumatic injury heals.[11] Only recently have models been constructed to simulate chronic overuse injuries commonly seen in sport.[16] A sound understanding of the stages of healing will help physicians conceptualize the healing process going on within the tissue. Such understanding will help them make rational decisions about rehabilitation.

Tendons heal in essentially three phases[17]: the cells react to injury; fibrous protein synthesizes with collagen; and scar remodeling occurs. Because healing

occurs continuously, the phases are arbitrarily chosen for convenience. All three phases are essentially occurring at the same time, although inflammation obviously takes place first and the lengthy remodeling phase finishes last.

***Cellular reaction***. Inflammation lasts 5 to 7 days. Although the tendon is unable to synthesize collagen immediately following injury, other pleuripotent cells begin to develop into fibroblasts, thus increasing the number of fibroblasts in the area. Infiltrative inflammatory cells also increase, and later in this phase, scattered collagen fibrils of varying levels of maturity and increased ground substance are observed.

***Collagen synthesis***. Production of collagen begins by 3 days after injury. During this fibroblastic phase there is increased vascularity and relatively little inflammation. Mucopolysaccharides and collagen fibrils increase in number and the extracellular matrix appears wavy. By 21 days after injury, the fibrils are coalescing into bundles.

***Remodeling***. This phase, which continues for life, is most pronounced at 17 to 28 days after injury. From the 14th to the 42nd day, the total amount of collagen remains stable because collagen synthesis equals collagen degradation. Healing scars increase in strength as more collagen becomes stable. Longitudinal tension might aid the remodeling process. Mechanical stresses applied to tendons 5 to 7 days after injury yield stronger tendons than those applied earlier in the inflammatory phase.

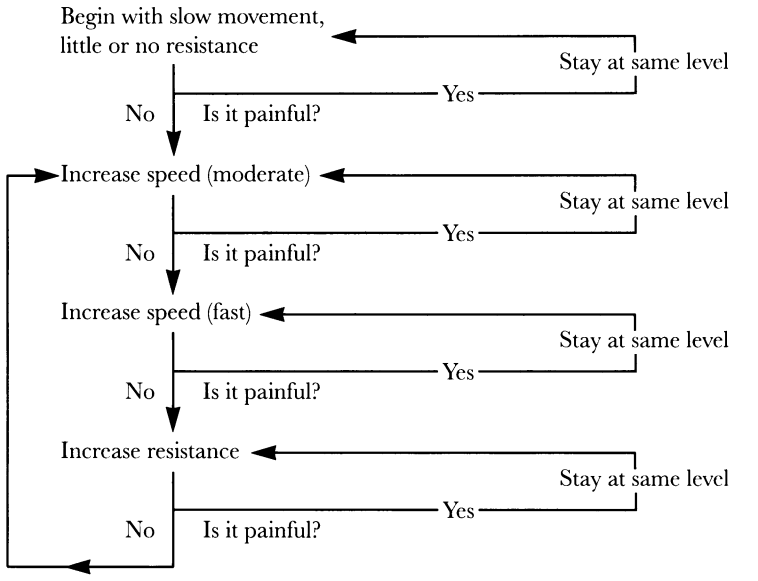
***Overuse injuries***. It is important to remember that this healing process reflects the response of an acutely traumatized tendon and that recovery from an overuse injury is slightly different. A chronic overuse (microtraumatic) injury can be distinguished histologically by observing a degeneration of both fibrocytes and matrix components. Furthermore, the inflammatory cell infiltration and orderly wound repair that is observed following macrotrauma is often diminished or absent.[11]

## Epigenetic and genetic influences on tendon healing

The tendon cell matrix response to injury might be affected by both epigenetic and genetic influences. Epigenetic factors are defined as factors that can influence tissue healing by altering the phenotypic expression (ie, protein production) of the cell without altering the genome.[11] Microvascular insufficiency, diminished estrogen levels in postmenopausal women, diabetes, and excessive rest following injury could all exert a detrimental epigenetic influence on tissue injury healing.

Aging exerts both epigenetic and genetic influences on tendon tissue and its response to insult. Advancing age has a detrimental effect on tendon structure and thereby increases the likelihood of overuse injury. As tendons age, collagen content increases, but elastin, proteoglycan matrix, and water content all decrease, resulting in less resilience. Furthermore, the metabolic activity of the tendons decreases with advancing age and the tissue adapts less well to physiological stress. Other possible genetic influences, such as blood type O and family history of tendon fragility, are currently under investigation as possible contributing factors to overuse syndromes.[11]

*Figure 3*. **Eccentric exercise program**

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## Rehabilitation

The goals of rehabilitation after overuse injuries are:

- to relieve symptoms,
- to decrease inflammation when present,
- to promote restoration of tissue integrity, and
- to return people to their activities as soon as possible.

Treatment can be individualized to suit particular needs but is based on sound principles using a variety of modalities and exercise. The main difference to be noted in treating overuse injuries is the shift in focus away from the symptoms of the *itis* toward restoring what is lost with the *osis*.[18] Rehabilitation can be broken down into three phases: acute, recovery, and maintenance.

***Acute phase***. The acute phase of rehabilitation is concerned with protecting the injured tissues by splinting, bracing, or other support to allow healing and control of the early inflammatory process. Ice, physical therapy, and prudent use of nonsteroidal anti-inflammatory drugs can help reduce inflammation and pain. As stated previously, the classic inflammatory process might not be present in some overuse tendon injuries during the acute phase. It is inappropriate to use NSAIDs or other modalities for extended periods when there is little evidence of an active inflammatory process. Casting or other rigid forms of immobilization are not considered good medical management today, although local rest and protected range of motion should be instituted.

Injectable steroids should be used cautiously and be reserved for acute inflammatory exacerbations that have not settled with noninvasive measures. For instance, classic lateral humeral epicondylitis, or tennis elbow, normally settles with stress reduction, direct application of ice, and oral analgesics. If the inflammation lingers longer than 10 to 14 days, then corticosteroid injections can be very helpful. During the acute phase of inflammation, local injectable steroids should be used cautiously with tendons that have perilous circulation, such as the patellar and Achilles tendons. Do not be reluctant to use injectable cortisone in and about an inflamed rotator cuff or bursa. This area responds very well to such treatment.

***Recovery phase***. After the acute phase, rehabilitation should emphasize appropriately loading the tendon and its muscle to provide proper stimuli for healing. Healing and recovering muscle strength and flexibility are most important at this stage. Protected motion is gradually increased to full passive then active range of motion. Resisted exercise (as tolerated) then progresses from isometric to concentric to eccentric work. Maximum stress is placed upon the muscle-tendon unit during eccentric exercise (*Figure 3*). Unless the muscle-tendon unit is strengthened to withstand maximum stresses, it will break down. Patients not rehabilitated on an eccentric strengthening exercise program will certainly reinjure themselves, which will frustrate both the patient and the physician.

It is vital to recognize the importance of task-specific exercise. If the individual athlete is preparing to return to a jumping sport, then the Achilles and patellar tendon must be prepared very effectively with a combined concentric and eccentric program to tolerate the challenge of ballistic movement. Fundamental to the rehabilitation program is restoring flexibility to the articulus of the muscle and tendon unit. The program should begin cautiously and within the limits of pain, but before returning to full athletic challenge, the tissues must regain maximum elasticity. Proprioceptive exercises should be included late in rehabilitation. Finally, general conditioning exercises and exercises for uninjured body parts should be incorporated as long as they do not interfere with the recovery of the injured tissue.

***Maintenance phase***. This final phase of rehabilitation is most important for restoring maximal performance and minimizing the risk of reinjury. Strength and flexibility must be fully restored (although inflexibility might be slow to resolve). Sport-specific pylometrics (ballistic stretching) can be added when strength is adequate. Unrestricted return to play should be allowed only after the athlete has undergone sport-specific testing and is deemed able to compete. ■

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