Disruption of the hamstring musculotendinous unit in athletes tends to be a devastating injury, one that frequently heals slowly. More often than not, the athlete, impatient for complete healing, returns to training and competition before he or she has fully recovered.

The muscle never completely heals although the symptoms appear to subside, at least until the first attempt at a burst of speed. The injury may persist throughout a sport season and sometimes even throughout the athlete's career (Christensen & Wiseman, 1972; Garret et al., 1983; 1984; 1989).

This article looks at the potential causes of hamstring muscle strain and presents a rational basis for the use of various therapeutic techniques from the time of injury to the return to sports.

**Etiology**

There is probably no single factor that predisposes an athlete to hamstring strain. Common proposed causes are poor flexibility (Ekstrand & Gillquist, 1983; Liemohn, 1978; Worrell et al., 1991) and inadequate muscle strength (Burkett, 1970; Christensen & Wiseman, 1972; Hage, 1981; O'Neil, 1976; Sutton, 1984).

Muscle fatigue has been linked to hamstring muscle injury (Dornan, 1971; Heiser et al., 1984). Data obtained on rabbit muscle has shown that fatigued muscle absorbs less energy before reaching the level of stretch that correlates to muscle failure (Mair et al., 1996). Therefore, training should include proper conditioning to avoid fatigue, and when fatigue is a factor, activities should be modified.

Faulty technique in sports is attributed to injury. Hamstring muscle injury among water skiers may be more common than previously thought.

Sallay et al. (1996) looked at such injuries among water skiers and found evidence of complete or partial avulsion of the proximal hamstring origin in all 12 skiers. The mechanism of injury was cited as improper body position which resulted in severe hip flexion while the knee was maintained in extension.

Hamstring injury is likely to occur in any sport that requires running. Overstriding while running is considered a major technique fault that stretches the hamstring muscle beyond normal lengths. Hamstring strains are thought to occur primarily in two phases of the running cycle: the swing and the takeoff.

In the late forward swing, an excessive antagonistic force of the quadriceps may lengthen the hamstring muscle and force it to overstretch. In the takeoff phase, paradoxical extension of the knee occurs via the synergistic action of the gastrocnemius and soleus. Muscle integrity may be compromised as the hamstring suddenly changes from stabilizing the knee in flexion to assisting in extension.

Individual analysis of running technique may be sought to prevent reinjury. “Toe up, heel up, knee up” is a common coaching cue aimed at improving sprinting performance (personal communication, Bo Schexnayder, track and field coach, U. of SW Louisiana, April 1993).

The “toe up” cue can also prevent hamstring injury by encouraging the gastrocnemius to serve as a knee flexor. This prevents the hamstring from overworking to flex the knee during the recovery phase.

Analysis of pelvic tilting may also shed light on the problem. An anteriorly tilted pelvis will lengthen the hamstring by displacing the muscle origin at the ischial tuberosity further from its insertion. This will decrease the amount of muscle excursion available until the point of muscle fail-
ure. Training techniques can be implemented to address pelvic stabilization.

Not all factors are so easily addressed. Histologically, injury may be related to the predominance of Type II muscle fiber in the hamstring. Type II fibers have a less developed endomysium structure, making them more susceptible to injury (Stauber, 1989).

Another structural factor is the dual innervation of the biceps femoris. Since the long head is innervated by the tibial portion of the sciatic nerve and the short head by the peroneal branch, it is thought that asynchronous stimulation of the two heads may occur (Burkett, 1970).

A study by Speer et al. (1993) imaged hamstring strains and found that in each patient, only one of the three muscles was injured. In 11 of the 17 patients it was the biceps femoris muscle that was injured.

The reinjury rate of the hamstring musculature has been proposed to be as high as 77% (Bailey & Bremiller, 1981). This may be related to areas of inflammation and calcification in the hamstring muscle group following the initial injury (Garrett et al., 1989).

Computed tomography (CT) is recommended for imaging ectopic deposition of calcium in muscle, but the calcification may also be visible on plain radiographs (Speer et al., 1993). Although imaging studies are not routinely warranted, further diagnostic follow-up of the chronic hamstring “muscle strain” is indicated to rule out other pathology such as neoplasm when the patient does not respond to conventional treatment.

**Medical Management**

**Immediate Care**

Initial treatment should consist of PRICE: Pain relief, Rest, Ice massage, Compression with an elastic wrap, and Elevation.

For pain relief, NSAIDS or acetaminophen can be used for 7 to 10 days (Best & Garrett, 1996). Varying degrees of rest should be imposed depending on the extent of injury. This may range from partial or complete cessation of athletic activities to non-weight-bearing ambulation on crutches.

Cryotherapy via ice massage, ice packs, or cold whirlpool immersion is essential for reducing the swelling by causing vasoconstriction. It decreases muscle spasm by decreasing muscle spindle activity and reduces pain by decreasing nerve conduction velocity.

Compression with an elastic wrap and elevation will further reduce swelling, thereby helping the body to heal itself.

**Physical Modalities**

Physical modalities can be used to address the soft tissue changes associated with muscle strain. Speer et al. (1993) found that radiographic imaging can localize disruption in a strain injury to the myotendinous junction. However, the same study showed that distant muscle tissue also demonstrates extensive injury as the fluid at the disruption site migrates along the epimysium and subcutis.

An effective adjunct to the use of PRICE immediately after injury is the use of microcurrent electrotherapy. Low-voltage current is administered at pulsed microamperage that strongly mimics the body’s own physiological electrical currents. This restores homeostasis to the body, facilitating healing and recovery.

Optimal healing effects occur at the subsensory level at frequencies under 1 hertz and amperage of 10-80 uA (Manley, 1991). Patients in the acute stage should be treated within 24 hours of each treatment to “recharge” the tissue and increase the electrochemical energy available for healing.

Dyson (1985) proposed that the healing process could be accelerated in the acute inflammatory stage of tissue repair using a single dose of pulsed ultrasound within a few hours of the injury. He recommended a 20% duty cycle at 0.5 W/cm² for 5 minutes to stimulate the release of histamine from mast cells by degranulation.

Ultrasound is more commonly used for its thermal effect following the acute period of injury. Continuous ultrasound effectively improves tissue extensibility by altering collagen molecular bonding (Ziskin & Michlovitz, 1989). This makes it a powerful adjunctive tool when it immediately precedes a stretching regimen. The “micromassage” effect of ultrasound helps to break up adhesions.

If the hamstring muscle heals with contracted scar tissue, normal muscle excursion will be prevented and the athlete’s condition may become chronic.