The Foot in Running

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Abstract

Injuries to the foot and ankle are often encountered in runners, be they high-level competitors or recreational joggers. Many of these injuries are due to overuse syndromes and training errors; others are related to the running surface or the athlete's footwear. Only with a rational approach to diagnosis can the primary underlying cause be identified so that appropriate treatment can be prescribed. Conservative measures, which include rest, cross-training, orthotic changes, and altering training methods, are often curative. Surgery is usually indicated only after conservative measures have been exhausted. Careful preoperative planning is needed to minimize dissection, thereby optimizing the chance of a return to the preinjury activity level. Preoperative counseling of the patient is also important, so that expectations about the outcome and the rehabilitation requirements are realistic.

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Many of the problems seen with recreational jogging and competitive running occur in the foot and ankle. This article will focus primarily on providing a rational approach to the diagnosis of running injuries to the foot, with an emphasis on determining the underlying cause. Only by understanding the primary predisposing factor can a physician institute training alterations, prescribe appropriate orthotic devices, and determine which surgical interventions will allow the patient to return to running.

Orthotic Devices

Indications

An orthotic device is used to relieve pressure from areas of the foot, to distribute loads to a particular area, or to prevent specific motion. Selection of the orthosis is dependent on the amount of control that is needed and on whether the foot is hypermobile or rigid. Rigid deformities of the foot, such as a rigid cavovarus deformity, need a well-constructed, custom-molded orthosis made from a plaster mold. Flexible deformities, such as flexible flatfoot, respond to less expensive semirigid, over-the-counter orthoses. We usually use foam mold boxes in making orthotic devices for flexible deformities. For most runners, a flexible or semirigid three-quarter-length orthotic device with a full-length covering is used.

It has been proposed that if an orthotic device helps correct problems with the runner's gait, it can be assumed that there will be improvement in running economy. However, Burkett et al[1] found that the use of orthoses actually increases oxygen consumption, which they attributed to the weight of the orthoses. They estimated an increase in oxygen consumption of 1.7% with the addition of 200 g of shoe mass. Catlin and Dressendorfer[2] found an increase in oxygen consumption of 1.9% with the addition of 200 g of shoe mass (100 g per shoe). This probably is of no concern for the recreational runner, but for the elite long-distance runner, a 1.7% increase in absolute oxygen consumption could reduce running speed as much as 5 m/min.[1]

These results should not be construed as a recommendation to forgo wearing orthoses while running a race. That decision should based on the runner's ability to run pain-free, independent of the energetic consequences of wearing orthoses.

Types of Orthoses

The ideal orthosis is durable, inexpensive, and easy to fabricate and distributes loads evenly while controlling forefoot, midfoot, and hindfoot motion. Since no single material can fulfill all of these requirements,

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one must choose from the three types of materials available—soft, semirigid, and rigid—on the basis of the needs of the patient. These materials are often combined to achieve the desired effect.

Soft orthoses are usually inexpensive and provide the most cushioning. They can be made in the office or obtained ready-made, as they do not require precise design and fit. Materials used are polyurethane, polyvinylchloride, and latex foam. This type of orthosis can be used either as a temporary measure or on a long-term intermittent basis.

Semirigid orthoses are the most frequently used. They may be made in the office but must be fabricated by someone who understands the biomechanics of the foot and which functions are being altered. One type is constructed out of Plastazote, a heat-sensitive, semiflexible material that becomes quite pliable when warmed. It can then be molded onto the foot or onto a cast made with the foot in a corrected position. Additional height of the longitudinal arch can be fabricated by autoadhesion of the Plastazote in layers. To ensure total foot contact, a foam pillow should be applied directly to the foot. Other types of semirigid materials include leather, cork, felt, cellular rubber, and viscoelastic polymers.

Rigid orthoses may be made of steel, thermoplastics, acrylic, polypropylene, or composite carbon fiber. They require precise design and offer maximal tensile strength and durability. They are also thinner (usually less than 3/16 inch), which allows use in most footwear. Rigid orthoses are molded over a positive model of the foot that has been obtained with the subtalar joint as close to neutral as possible. Care must be taken when rigid orthoses are used by patients participating in high-impact activities because of minimal reduction of impact shock.[3] These orthoses are usually expensive, and potential problems with their use include added pressure on bony prominences and development of lower-extremity stress fractures, neuromas, and sesamoiditis.

Injuries Related to Surface Type

Synthetic surfaces have replaced cinders and grass over the past 30 years. These surfaces have allowed athletes to achieve higher levels of performance at the expense of greatly increasing the number of injuries.[4] With the elimination of the skid phase of running that is seen with imperfect surfaces such as grass and cinders, there is a more powerful push-off and more efficient running; however, there is also more torsional and shearing stress transferred to the lower-extremity joints. Synthetic surfaces accentuate vibration to an extent not seen on cinder or grass surfaces, with a greater potential for damage to the ankle, leg, and knee.

Torsional Joint Injuries

In running, minimal emphasis is placed on rotational flexibility. Most emphasis is placed on linear stretching of the hamstrings and Achilles tendons. If there is limited hip, pelvic, or back rotation, more torque is placed on the knee, leg, and ankle during running. For instance, if there is an external rotational deformity of the hip, more torque is placed on the knee during running as the runner increases speed and attempts to rotate the lower extremity inwardly. If the runner does internal rotational stretching of the hip and runs on the foot with the extremity slightly externally rotated, knee pain can be lessened. Rarely does femoral anteversion or internal tibial torsion cause problems, since both are more anatomic positions for running.

Short-Leg Syndrome

Leg-length discrepancy is often associated with a history of repeated injuries to the shorter leg. These injuries may include stress fractures, medial knee strain, hyperpronation of the foot with resultant plantar fasciitis, patellar subluxation, or iliotibial-band tendinitis.[5] Short-leg syndrome can develop from a functional, a structural, or an environmental leg-length discrepancy. Structural short leg is an anatomic difference resulting from congenital short tibia or femur or old fractures. Functional short leg may be due to a fixed pelvic obliquity from scoliosis or a flexion contracture of the knee or ankle. In the experience of the senior author (D.E.B.), environmental short-leg syndrome is caused by running at only one tilt on a road or always in the same direction on a circular track.

Regardless of causation, it is hypothesized that the inequality increases the mechanical stresses in the affected extremity by both increasing the load borne by the shortened extremity and decreasing the area of load distribution (mechanical stress = force/area).[6] The short leg is considered to be at higher risk for stress-related injuries.

In symptomatic runners, a discrepancy of more than 20 mm usually requires correction.[7] In runners who have repetitive injuries of the extremity, a buildup may be put in the running shoe even for a discrepancy as small as 5 mm, provided no other causes can be found.[6] Total correction of the inequality should not be made initially; a gradual buildup is in order so as not to overcorrect the deformity. The insole of the running shoe can be built up in one-eighth- or one-fourth-inch

increments, increasing the elevation of the shoe to one-half inch. Should more elevation be needed, it must be added to the midsole or outer sole of the shoe. For greater discrepancies of 50 to 60 mm, operative intervention should be considered.

A lift is not indicated for a runner who has a leg-length discrepancy but has had no significant problems. In such a case, putting a lift in the shoe could create, rather than solve, a problem.

Forefoot Disorders

Metatarsalgia

Metatarsalgia, either localized or generalized, can have many underlying causes in the runner. In localized metatarsalgia, a hypermobile first metatarsocuneiform joint or a long second metatarsal can increase pressure under the second or third metatarsal. In Morton's syndrome, the combination of a short first metatarsal, a hypermobile first metatarsal segment, and posteriorly displaced sesamoids results in increased stress on the second and third metatarsal heads.8 More generalized metatarsalgia may be caused by a tight Achilles tendon or anterior ankle impingement that limits ankle dorsiflexion, which leads to increased metatarsal impact and increased weight concentration on the forefoot. The differential diagnosis also includes metatarsophalangeal synovitis, Freiberg's disease in the adolescent, subluxation of the second or third metatarsophalangeal joint, and entrapment of or damage to the interdigital nerves. It is important not to overlook these less common causes of pain.

Treatment of metatarsalgia includes placing a pad just proximal to the second and third metatarsal heads or just beneath the first metatarsal to allow it to bear weight. Stretching of the Achilles tendon and placement of a well-fitting orthosis are often successful for a contracted Achilles tendon. For anterior ankle impingement, an arthroplasty will help increase ankle dorsiflexion. If conservative care fails to provide relief, plantar condylectomy is sometimes necessary. In more severe cases, a metatarsal osteotomy may be indicated.

Sesamoid Injuries

Sesamoid injuries in the runner may be due to overuse. Predisposing factors include a cavus foot secondary to plantar flexion of the first metatarsal, hyperpronation of the foot, and limited dorsiflexion of the first metatarsophalangeal joint. The cause of the pain may be classified as due to sesamoiditis, osteochondritis, or fracture of one or both of the sesamoids. The tibial sesamoid receives most of the weight of the first metatarsal and is, therefore, the more commonly affected.[9]

The injured sesamoid is usually tender locally, and the pain is increased by passively dorsiflexing the great toe. The initial radiograph often appears normal, but a bone scan may be helpful in localizing the site of pathologic change to the sesamoids. When no history of specific injury is reported and a sesamoid stress fracture is suspected, 3-week-interval radiography or bone scanning is indicated.

We prefer an initial conservative treatment program that includes reduction of activities and casting for 2 to 3 weeks, followed by use of an orthotic device incorporating a steel-shank extension and a U-shaped pad to unload the symptomatic sesamoid. As symptoms resolve, activities can be gradually increased over time.

If the use of conservative measures for more than 6 months has failed, surgical intervention may be indicated. Shaving the plantar half of the tibial sesamoid has provided good results in patients with a discrete intractable plantar keratosis over the tibial sesamoid without evidence of a plantar-flexed first metatarsal.[10] If the sesamoid disorder is due to plantar flexion of the first metatarsal, a more diffuse callus is seen clinically beneath the metatarsal head; in this setting, a proximal dorsal closing-wedge osteotomy of the first metatarsal can be effective.[10] For displaced or nondisplaced sesamoid fractures that are persistently symptomatic despite conservative treatment, surgery may be considered.[9,11] Partial or total sesamoid excision or bone grafting is recommended, depending on the size and number of fragments present.

Hallux Rigidus

Hallux rigidus frequently occurs in runners, with resultant limited and painful motion of the first metatarsophalangeal joint. Examination reveals palpable marginal osteophytes and limited flexion and extension. Radiographic findings of degenerative arthrosis in the first metatarsophalangeal joint, lateral and dorsal osteophytes on the metatarsal head, and occasionally loose fragments within the first metatarsophalangeal joint are diagnostic of this condition.

Conservative treatment consists of using a device that limits motion of the first metatarsophalangeal joint by providing rigidity to the forefoot. A metatarsal bar or a rigid-sole running shoe may be used.

If conservative measures have failed and the osteoarthritic changes are mild to moderate, a cheilectomy of the first metatarsophalangeal joint is used. Because approximately half of the joint motion gained intraoperatively is lost postoperatively,[12] it is important to gain as much motion as possible at the time of the cheilectomy. Our goal is to achieve 80 to 90

degrees of intraoperative dorsiflexion by removing the dorsal 20% to 30% of the metatarsal head. A dorsal closing-wedge osteotomy (Moberg procedure) of the proximal phalanx can be included to decompress the first metatarsophalangeal joint if this motion cannot be achieved. In competitive sprinters, however, we believe some push-off strength may be lost, and we try to avoid a Moberg procedure in these individuals.

Arthrodesis is the definitive procedure for advanced osteoarthritis. We try, if possible, to avoid the Keller procedure or prosthetic replacement in the running athlete to avoid stress-transfer lesions and patient dissatisfaction with loss of push-off strength.

Hallux Valgus

The hallux valgus deformity has been associated with improperly fitting footwear, ligamentous laxity, and a hereditary predisposition. Runners with pronated feet are at risk due to increased valgus stresses on the hallux during toe-off, which cause repetitive microtrauma and the resultant hallux valgus deformity.[11] Initially, hallux valgus deformity produces pain at the medial eminence. As a bunion worsens, the first metatarsal joint drifts into more valgus deviation, causing increased transfer of weight to the second and third metatarsals. Hallux valgus can eventually lead to secondary disorders, such as metatarsalgia, subluxation of the second metatarsophalangeal joint, claw-toe deformities of the lesser toes, and neuromas.[13]

The objective of treatment must be to provide a stable and durable correction, as well as to maintain adequate range of motion of the first metatarsophalangeal joint. Conservative treatment consists of training alterations and shoe modifications. A shoe with a wide toe box and a well-padded forefoot may be helpful. Orthotic devices, such as a metatarsal lift, may alleviate symptoms due to metatarsalgia, as well as secondary claw toes and neuromas. A lightweight medial arch support may relieve the valgus stress on the first metatarsophalangeal joint in the runner with hyperpronation.

If the hallux valgus deformity progresses and the functional capacity of the athlete suffers, a precise, well-planned operation should be performed. The procedure chosen must be individualized to the degree of deformity, the patient's needs, and the underlying pathologic changes. In the world-class athlete, the goal of surgery should be the functional return and restoration of a congruent joint, which often means a less definitive procedure until the athlete retires. In the weekend athlete, the more definitive procedure is used, knowing that some function will be lost. In our experience, a distal chevron osteotomy has resulted in satisfactory outcomes and appears to be a good alternative in the adult athlete with mild to moderate deformity.[14]

Interdigital Neuroma

For the runner with evidence of an interdigital neuroma (Fig. 1), initial treatment should consist of a metatarsal bar or pad placed behind the metatarsal head in an attempt to decrease the traction on the nerve. If this is not successful, an injection of corticosteroid should be considered. For recalcitrant cases, nerve excision through a dorsal incision with release of part of the intermetatarsal ligament may be indicated.

Callosities and Blisters

Callosities and blisters are frequently seen in running athletes. These lesions occur in the forefoot about the toes and under the metatarsals. Helpful treatment measures include well-padded shoes, a rigid stretching exercise program, and topical foot keratolytic agents (e.g., salicylic acid and urea agents). The use of a metatarsal pad or foot liner may also be beneficial to relieve friction and pressure on the metatarsal heads. If callosities persist, radiographs should be obtained to identify possible exostoses or bone spurs that may require surgical excision.

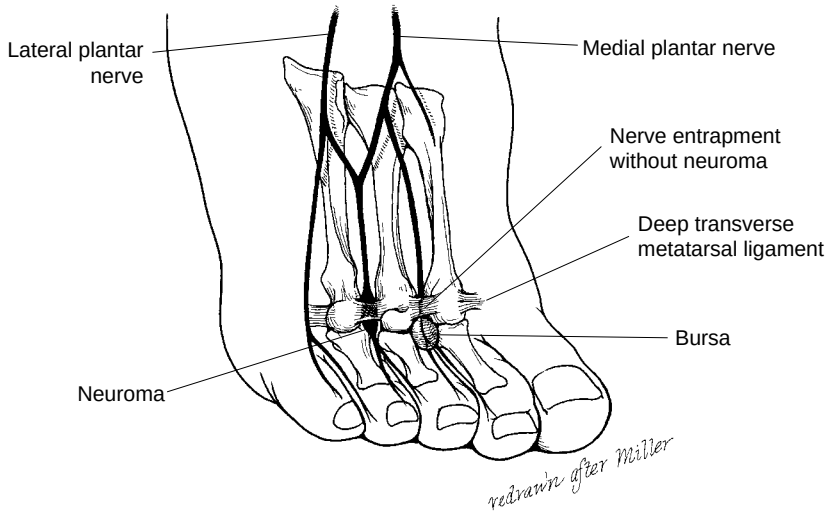

Fig. 1 Position of the foot at push-off. Formation of a neuroma due to compression of an interdigital nerve can be the cause of forefoot pain in the runner.

Toenail Disorders

Running athletes develop toenail disorders from repetitive trauma, commonly caused by hyperextension of the great toe and contact of the toenail with the dorsum of the shoe. Nail loosening, ingrown toenail, and subungual hematoma are common. Poorly fitting shoes can be another cause. A shoe that is too short will not allow enough space for the toes. A shoe that is too wide allows the foot to slide forward within the shoe and causes the toes to strike against the distal end of the shoe.

Treatment consists of metatarsal pads for hyperextension deformities and properly fitting shoes with a snug midfoot and adequate toe box. Taping of the toenails also prevents friction and loosening.

Stress Fractures

A stress fracture results when the natural intrinsic reparative processes fail to keep up with microdamage. Normally, physiologic bone remodeling takes place in response to physical stress and leads to increased strength at the site of stress.[15] When increased forces cause too sudden a change in stress concentration or excessively high stresses are incurred, the microdamage rate overwhelms the reparative processes. If this imbalance continues, the microscopic changes eventually lead to the macroresponse seen as a stress fracture.[7,15]

There are a variety of reasons why stress fractures occur. An injury in one leg will result in increased stress on the uninjured extremity. The uninjured leg supports more of the load during running, and a stress fracture may occur. The combination of poor conditioning and a high level of motivation also seems to predispose an athlete to a stress fracture.[16] Other causes include training errors and a sudden change in running routine, such as beginning incline running. In excessively long runs, the muscles fatigue, placing additional force on the bones, and the normal cushioning effect is lost, increasing the stress at focal points in the bone.[17] Runners who run on hard surfaces, wear poorly cushioned shoes, or have a heavy heel strike are also at risk for stress fractures.

The initial symptoms are often insidious and difficult to diagnose. Radiographs are often normal for 3 or 4 weeks after the onset of symptoms, and in some cases may never reveal the stress fracture. A bone scan can be obtained if the diagnosis is uncertain, especially in the high-profile athlete, in whom early diagnosis and treatment are vital.

The cornerstone of treatment of stress fractures in the running athlete is reduction of activities. Cast immobilization is usually not warranted unless there is an intra-articular fracture or a fracture of the navicular or the base of the second metatarsal. In cases of delayed union or nonunion, an external electric bone stimulator may be beneficial. To avoid recurrence, the underlying cause of the stress fracture must be addressed before resumption of running.

Biomechanical causes of foot disorders can usually be corrected with shoe modifications. However, if it is believed that the stress fracture occurred because of training errors, these must be addressed, and the runner must be willing to change his or her routine. Also, a softer, more forgiving running surface should be sought.

If no specific biomechanical imbalances or training errors can be found and training is appropriate for the runner's age, metabolic disorders should be considered. Hormonal imbalance and nutritional and hematologic disorders may contribute to stress fractures, especially if they are recurring. Oligomenorrheic runners have been noted to have significantly decreased bone mineral and trabecular bone density than their eumenorrheic counterparts.[18] It appears that the positive effects from weight-bearing exercises are offset by the adverse effect of low estrogen levels. If metabolic disturbances are suspected, routine laboratory studies, such as determination of calcium and phosphorus concentrations and a complete blood cell count, should be performed as an initial screen.

Tendinitis

Achilles Tendinitis

Achilles tendinitis is a continuum of disease, presenting initially as an inflammation of the peritendinous structures and bursa and progressing to involvement of the tendon itself (tendinosis). Common training errors responsible for this condition include abruptly increasing or restarting the training program, running on uneven or slippery terrain, and performing inadequate stretching exercises.

In the peritendinitis stage, it is the structures surrounding the tendon, including the peritendinous sheath and bursa, that become inflamed; the tendon itself is not involved. Tendinosis can develop gradually when there is damage to the tendon itself in the form of tears, necrosis, or calcification within the tendon.

Achilles tendinitis can occur in two forms: insertional and noninsertional. We have found insertional Achilles tendinitis more difficult to treat, although it appears to occur much less commonly.

Insertional Achilles Tendinitis

The pathologic changes in insertional Achilles tendinitis are localized near the tendon-bone junction. There is often an associated Haglund's defor-

mity (excessive posterosuperior prominence of the calcaneal tuberosity).[19] This bony prominence causes inflammation of the retrocalcaneal and superficial calcaneal bursa, which leads to tendinitis. A cavus foot brings the bony prominence closer to the anterior edge of the distal part of the Achilles tendon, increasing the risk of tendinitis. Symptoms include pain at the posterior aspect of the heel, especially when running uphill. There is often an associated tight heel cord and a palpable bony prominence posteriorly.

Conservative treatment includes rest, intermittent ice massage, Achilles stretching, anti-inflammatory medications, and a heel lift to be used in all shoes, including running shoes. If the pain is more medial, a medial heel wedge should be used. If the pain is more lateral, a lateral heel wedge can be considered. Corticosteroid injections are not recommended because of the risk of tendon rupture.

If conservative measures are not effective after 10 to 12 months, surgery may be considered. If a Haglund's deformity is present, it should be removed, and the tendon debrided. Any detached portion of tendon should be sutured back to the calcaneal tuberosity. This can be done through a single posterior Achilles-splitting incision or through medial or lateral incisions adjacent to the Achilles tendon. The choice of incision is based on the source and location of the pain. If significant disruption of the Achilles tendon occurs, a tendon transfer may be required to provide additional strength to the Achilles tendon. The tendons that may be used for the transfer include the peroneus brevis, the flexor digitorum longus, and the flexor hallucis longus.

Postoperatively, the patient is treated with immobilization and cast braces for 6 to 10 weeks, depending on the amount of disruption of the Achilles tendon insertion and whether a tendon transfer was performed. The runner can usually return to full activities within approximately 6 months.

Noninsertional Achilles Tendinitis

Noninsertional Achilles tendinitis is characterized by pain and tenderness approximately 2 to 6 cm above the insertion. This is the area with the most tenuous blood supply to the Achilles tendon. Except for location, the type of pain is similar to that seen with insertional Achilles tendinitis.

Conservative therapy is identical to that for insertional tendinitis with the exception that an injection of bupivacaine into the tendon sheath may be used to disrupt adhesions and provide relief of symptoms. If the symptoms persist for 10 to 12 months and tendinosis is present, debriding the tendon and weaving the plantaris tendon through the Achilles tendon in the area of damage should be considered. Occasionally, only debridement of the necrotic tissue through a splitting incision is necessary.

Posterior Tibial Tendinitis

Posterior tibial tendinitis occurs on the medial aspect of the ankle, generally behind or slightly distal to the medial malleolus. Less often, the pain is at the insertion, usually associated with an accessory navicular bone.

The initial conservative treatment includes intermittent ice massage, anti-inflammatory medications, and shoe modifications. A medial heel and sole wedge should be used, especially if the patient hyperpronates. If symptoms persist, an injection of bupivacaine into the tendon sheath may disrupt adhesions and provide clinical relief. In some persistent cases, casting for 4 to 6 weeks may be necessary to allow inflammation to subside.

If symptoms persist for more than 4 to 6 months, surgical intervention with tenosynovectomy is indicated to prevent further tendon degeneration. For insertional tendinitis with an associated accessory navicular bone, the fragment is excised, and the tendon is sutured back to the medial side of the navicular. If significant tendon disruption occurs, a tendon transfer using the flexor digitorum longus is attached to a drill hole in the navicular bone.

In chronic noninsertional posterior tibial tendinitis, a release of the tendon sheath with debridement may lead to resolution of the symptoms. If the posterior tibial tendon is torn proximal to its insertion, a tendon transfer should be considered at the time of repair. The patient must be made aware preoperatively of the length of the postoperative rehabilitation period (typically 6 to 9 months).

Peroneal Tendinitis

Tendinitis of the peroneal tendons occasionally occurs in runners, particularly those with a cavovarus deformity, in whom increased stresses are placed on the peroneal tendons. Symptoms occur posterior to the distal end of the fibula or at the point where the tendons are separated by the peroneal tubercle along the lateral border of the calcaneus. Symptoms may radiate up the lateral aspect of the leg or be increased by forced supination of the foot, similar to the situation in a cavus foot with a rigid plantar-flexed first metatarsal and forefoot valgus. Peroneal subluxation during the toe-off phase of gait may be a predisposing factor. Subluxation is assessed by applying resistance to the foot as the patient actively everts against that resistance, causing the peroneal tendons to subluxate.

Conservative treatment includes rest, intermittent ice massaging, anti-inflammatory medications, and, on occasion, cast immobilization. For the runner who has persistent pain in spite of conservative

care, consideration should be given to exploration of the peroneal tendons. These tendons often have longitudinal tears or flap tears, which must be debrided or repaired. For chronically subluxating peroneal tendons, we prefer direct repair of the retinaculum back to the bone and utilization of the groove-deepening technique described by Slätis et al.[20]

Heel Pain

The most common causes of heel pain in the runner include fat-pad trauma, plantar fasciitis, fracture of the os calcis, and compression of the first branch of the lateral plantar nerve. Fortunately, most cases resolve with conservative treatment, although it is common for symptoms to persist for 10 to 12 months.

Plantar fasciitis refers to heel pain produced by microtears in the plantar fascia and chronic inflammation. This inflammation can occur at the medial tubercle of the calcaneus where the plantar fascia attaches or more distally along the course of the plantar fascia. Patients have a history of severe pain in the morning on arising, which decreases as they begin to walk. The pain is often exacerbated by running, especially with hill climbing and sprinting. Tenderness is usually elicited by palpating along the medial tubercle of the calcaneus and distally into the fascia itself. The discomfort may increase with forced toe dorsiflexion. When examining the patient with this constellation of symptoms, it is important to rule out tarsal tunnel syndrome, which is characterized by tenderness not only at the medial tubercle of the calcaneus but also along the course of the posterior tibial nerve.

Treatment of plantar fasciitis consists of decreasing activity, heel-cord stretching, contrast baths, night splints, and anti-inflammatory medications. If symptoms persist, surgery may be indicated. In our experience, however, approximately 95% of patients recover within 1 year without surgical intervention. For patients who do not improve despite prolonged conservative treatment, we prefer a partial plantar fascia release with removal of the inflamed necrotic fascia. We remove the calcaneal spur if it appears large enough to be causing mechanical pain or compressing the first branch of the lateral plantar nerve.

Another cause of heel pain in the runner is a stress fracture of the os calcis. This can be either a fracture through a calcaneal spur or one that extends up into the body of the calcaneus. These injuries are seen on oblique radiographs of the os calcis or a bone scan. The injuries usually respond to conservative care, although they can be symptomatic for 6 to 10 months.

Entrapment of the first branch of the lateral plantar nerve can also cause heel pain.[21] The nerve courses deep to the deep fascia of the abductor hallucis muscle and then passes transversely, superficial to the quadratus plantae, and innervates the abductor digiti minimi (Fig. 2). In the runner, there is often significant hypertrophy of the abductor hallucis muscle, which may lead to compression of the nerve as it passes deep to the muscle. Another site of compression is where the nerve passes over the medial edge of the quadratus plantae to assume a horizontal course. Symptoms of compression of the first branch of the lateral plantar nerve may be similar to those of plantar fasciitis, although the pain is usually described as having a more burning quality with increased radiation. Pain is also often described as radiating up into the leg and across the lateral aspect of the foot.

The conservative treatment of lateral plantar nerve entrapment includes relative rest, heel-cord stretching, contrast baths, anti-

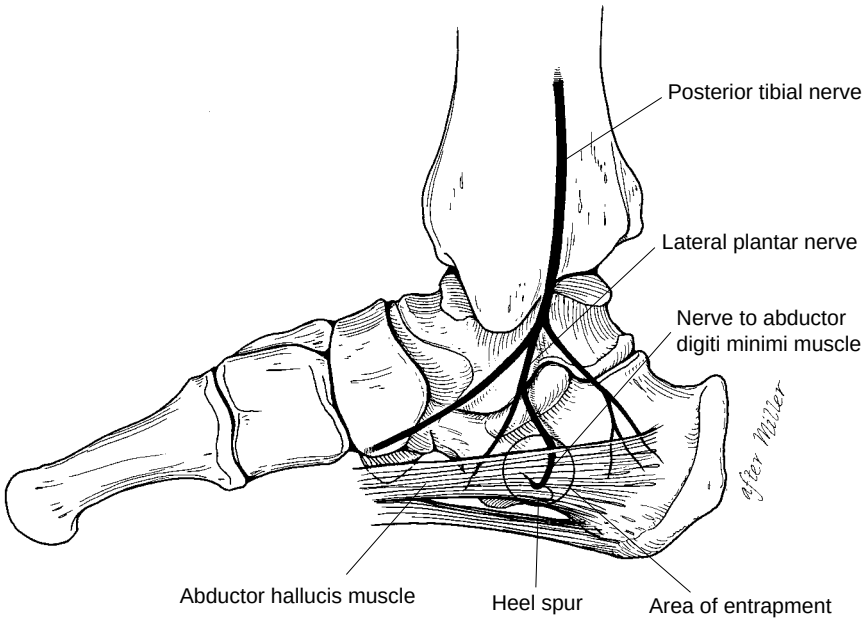

Fig. 2 Entrapment of the first branch of the lateral plantar nerve (the nerve to the abductor digiti minimi muscle) can cause heel pain. The first branch is trapped by the surrounding muscles and the deep fascia of the abductor hallucis muscle.

inflammatory medications, night splints, and occasionally corticosteroid injections. A longitudinal arch support might be beneficial, particularly if the runner pronates.

If conservative measures fail after 10 to 12 months, a local nerve decompression can be performed through an oblique medial incision. Neurolysis is performed by removing a small amount of the medial third of the plantar fascia and releasing the deep fascia of the abductor hallucis. This operation has been reported to yield good results in over 90% of patients.[21]

Nerve Compression

There are several common sites of nerve compression in the runner.[7] Entrapment of the first branch of the lateral plantar nerve to the heel has been described in the previous section.

Tarsal tunnel syndrome is characterized by pain along the posterior tibial nerve. It is sometimes referred to as posterior tarsal tunnel syndrome to differentiate it from the less common anterior tarsal tunnel syndrome (deep peroneal nerve compression). The posterior tibial nerve may be compressed behind the medial malleolus, causing a radicular painful, tingling sensation on the plantar aspect of the foot. The compression is caused by excessive pronation and possibly the presence of a prominence on the medial posterior talus. If the tarsal tunnel syndrome occurs because of excessive pronation, simply using a longitudinal arch support to bring the foot out of pronation will often decrease the severity of the symptoms. If a prominent posterior medial talus or a large os trigonum is causing the compression, the bony prominence may have to be removed.

The deep peroneal nerve may be compressed in the anterior ankle or at the dorsal talonavicular joint.[22] Anterior tarsal tunnel syndrome occurs when the deep peroneal nerve is trapped beneath the inferior extensor retinaculum.[7] The nerve can be compressed between a small dorsal bony prominence at the talonavicular joint and the inferior extensor retinaculum extending over the joint. Surgical decompression consists of partial release of the inferior retinaculum and removal of the osteophyte. The deep peroneal nerve can also be compressed at the dorsum of the foot in the area of the first tarsometatarsal joint, by either a bony prominence or an accessory bone.[7,22] Surgical decompression may be required if symptoms persist.

Superficial peroneal nerve entrapment should be considered in a runner with unexplained anterolateral leg pain. Compression of this nerve usually occurs at its exit point from the deep fascia, 6 to 9 cm above the tip of the lateral malleolus (Fig. 3). Entrapment of the superficial peroneal nerve typically causes pain over the lateral border of the distal calf and the dorsum of the foot and ankle. Local tenderness where the superficial peroneal nerve exits the fascia proximal to the lateral malleolus and sensory irregularities should arouse suspicion of nerve entrapment.[23] A positive exercise test and abnormal results of nerve conduction studies help confirm the diagnosis. If the symptoms are chronic and recurring, a fascia release is necessary at the site where the superficial peroneal nerve exits the fascia.

The sural nerve may be trapped anywhere along its course. In several reported cases, entrapment occurred after fracture of the base of the fifth metatarsal or recurrent ankle sprains; it may also occur at a site where a ganglion emerges.[24] On rare occasions, the sural nerve will be irritated in patients with Achilles peritendinitis. This is suggested clinically by pain radiating down the lateral ankle on palpation of the posterior Achilles tendon. In the presence of adhesions or inflammation involving the lateral ankle with associated localized tenderness, a positive Tinel's sign, and paresthesias, the examiner should consider sural nerve involvement. If conservative measures fail, the

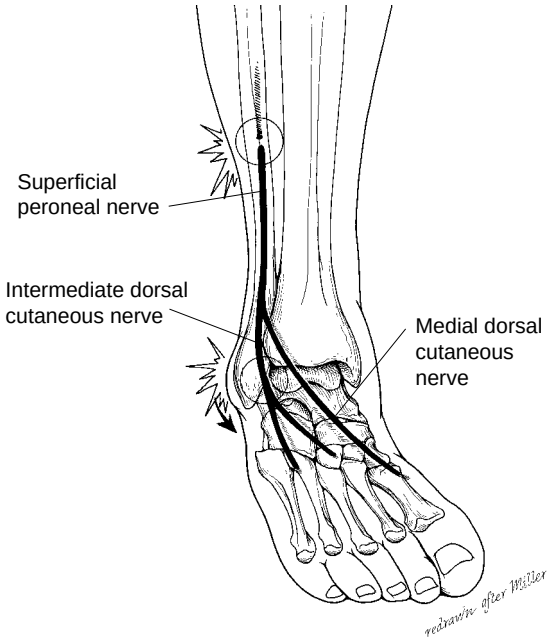

Fig. 3 Entrapment of the superficial peroneal nerve can cause dorsal foot pain. Compression of the nerve usually occurs at its point of exit from the deep fascia, 6 to 9 cm above the tip of the lateral malleolus. In the patient with ankle instability, inversion of the ankle can cause this entrapment.

sural nerve can be decompressed and transposed.

A common location of nerve compression in the jogger is in the medial arch. Compression of the medial plantar nerve occurs at the talonavicular joint underneath the knot of Henry (Fig. 4). If there is hypermobility of the forefoot with a wide migration of the navicular bone on the talus, the medial plantar nerve may be caught and compressed under the knot of Henry just beneath the talonavicular joint. This causes a peculiar "giving-away" sensation in the foot and a radicular-type pain that radiates throughout the medial arch. Generally, the neuritic symptoms occur only in the distribution of the medial plantar nerve. This syndrome is known as "jogger's foot."

For cases unresponsive to medial longitudinal arch support and other conservative modalities, an isolated release of the medial plantar nerve is indicated. By making a small incision medially on the foot and releasing a portion of the naviculocalcaneal ligament, the site of compression can be identified and released.

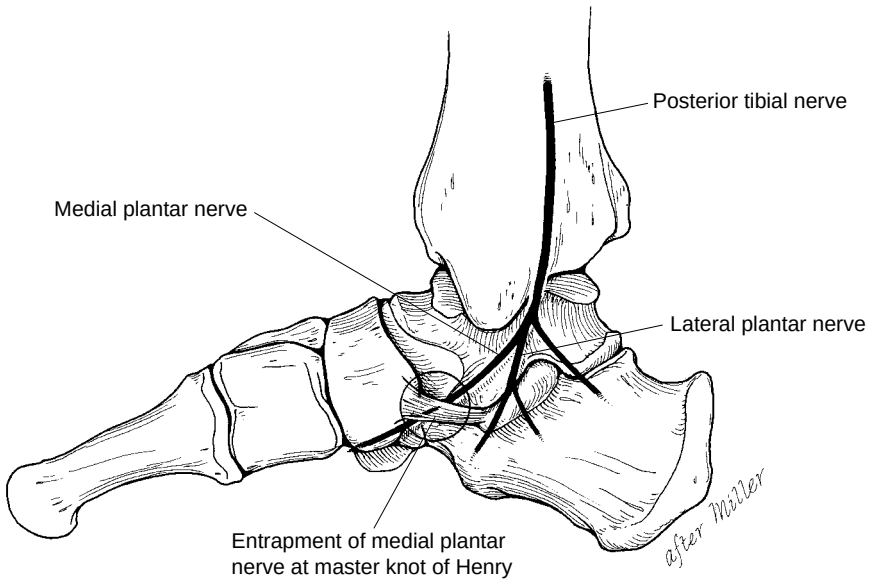

Fig. 4 Entrapment of the medial plantar nerve can cause a "giving-away" sensation in the foot and a radicular-type pain that radiates throughout the medial arch. These neuritic symptoms are known as "jogger's foot." Excessive abduction of the forefoot can exacerbate this condition.

Impingement Syndrome

There are three relatively common sites of impingement in runners: the dorsum of the first metatarsophalangeal joint, the anterior aspect of the ankle, and the lateral aspect of the ankle.

Impingement in the dorsal first metatarsophalangeal joint is one of the components of hallux rigidus. As discussed previously, in cases that fail to respond to shoe modifications or use of an orthotic device, a cheilectomy is the preferred surgical treatment.

Anterior ankle impingement presents a similar problem. When persistent pain is not relieved by a slight heel lift in the running shoes, an anterior ankle arthroplasty can be carried out with a small anterior arthrotomy incision or an arthroscope.[25] At the time of arthroplasty, it is important to evaluate the area of erosion on the neck of the talus and to remove enough of the anterior tibial prominence so that there is no encroachment onto the talar neck.

Impingement in the lateral aspect of the ankle is most often a sequela of trauma, such as a sprained ankle, and is usually seen in patients with excessive pronation. The impingement occurs at the lateral subtalar joint or in the fibulotalar joint. Conservative treatment, by supporting the medial aspect of the foot with a longitudinal arch support or a medial heel wedge, is usually appropriate. Rarely, surgical exploration and debridement is warranted if symptoms persist.

Summary

Many of the problems seen with recreational jogging and competitive running occur in the foot and ankle. These problems are most often due to overuse syndromes, training errors, or use of inappropriate footwear or running surfaces. The initial treatment is to decrease or stop the patient's running. For many athletes, running is an integral part of their lifestyle, and converting them to a nongravity or cross-training program to allow the affected part to heal is important for their mental well-being, as well as to maintain their aerobic conditioning. Occasionally, modifications must be made to running shoes, such as the use of orthotic devices, but more often athletes need to be instructed to alter their exercise routine and change their running surface. If surgery is performed, great care should be taken to minimize soft-tissue injury. Preoperative patient education is essential, so that expectations about the outcome and the length of rehabilitation are realistic.

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