RUNNING INJURES AND TREATMENT

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There are few individuals who have not suffered some form of chronic injury from running. Typically running injuries have been attributed to "overuse". This term may be inaccurate and in fact misleading. For example, many people run many miles per week year after year and do not report injuries. On the other hand, some individuals run very few miles per week and suffer some type of injury. If overuse was the sole source of injury then individuals who ran longer distances would inevitably incur a greater number of injuries. There is no evidence to support this notion. In fact, there is evidence that runners with low mileage may be injured more often than those with high mileage (Pagliano and Jackson, 1980).

Multiple contributing causes for a diseased state have been discovered in several areas of medicine. These multiple causes are referred to as risk factors. One risk factor alone may be insufficient to cause a disease but the greater the number of risk factors, the more likely the person is to suffer from the disease. This concept can also be used for running injuries. It would appear that the primary risk factor in chronic running injuries results from structural abnormalities of the lower extremity. Thus. runners who have performed over a long period of time in a relatively injuryfree state have satisfactory structural lower extremity biomechanics. Overuse is also a risk factor but certainly one of less importance than rhe structure of the lower extremity.

HYPERPRONATION (OVER-PRONATION)

There have been several self-reporting and clinical studies that have described the types of injuries that runners incur (Runner's World, 1971 and 1973; James et al., 1978; Clement et al., 1981). In each of these studies it was apparent that the majority of lower extremity injuries to runners occurred extrinsic to the foot; that is, the injuries were essentially above the level of the sub-talar joint. For example, James et al. (1978) reported 29% of injuries to runners could be generally described as knee pain and 13% as shin splints. Clement et al. (1981) reported injury rates of 50.6% for patello-femoral pain, 12.0% for ilio-tibial band syndrome and 10.8% for patellar tendinitis. It is logical to assume that, since the foot is the primary contact with the running surface, what occurs at the foot/ground interface may cause these injuries. The culprit of these injuries is generally labeled "hyperpronation" or "over-pronation" although the mechanism of the relationship between injury and hyperpronation is poorly understood.

During the support phase of running, the foot contacts the ground in a slightly supinated or inverted and dorsiflexed position. The foot then rapidly rolls medially to a pronated or everted position. Maximum pronation is reached at about 45% of the support period (Hamill et al., 1992). At about 75% of the support period there is a significant increase in the rate of re-supination. A certain amount of pronation is necessary to accomplish the attenuation of impact forces.

Clarke et al (1983) suggested that 7 to 13o of pronation could be considered normal. However, when the angle of pronation exceeds this maximum, hyperpronation occurs. It has been suggested that hyper- or over-pronation results in injury. This mechanism is often referred to as the "Hyperpronation Model" of lower extremity injury.

Bates and associates (1978) suggested a mechanism for injury that incorporates the sub-talar and the knee joint actions. Knee flexion is a synchronous complimentary

motion with sub-talar joint pronation: Likewise, knee extension is complimentary synchronous with sub-talar joint supination (Figure 1).

Both knee flexion and pronation cause the. tibia to internally rotate while knee extension and supination cause the tibia to externally rotate. It was hypothesized that a disruption of the normal timing of these joint relationships may result in injury. For example, if the sub-talar joint is still pronating while the knee is extending, an inappropriate torsion around the tibia could occur resulting in injury to the structures of the knee or foot but most probably to the knee. Hamill et al. (1992) suggested that this mis-timing of the lower extremity joint actions should be defined as hyperpronation.

INJURY MODELS: STRUCTURAL MODEL VS HYPERPRONATION MODEL

While there is some evidence to support relationships between hyperpronation and certain types of injuries such as patello-femoral pain syndrome (Soderburg. 1986). hyperpronation alone cannot be used as a general model for lower extremity injury. Clinical observation reveals a limited correlation between the amount of pronation and the severity or even the occurrence of injury. Recent treatment of running injuries have stressed the importance of relating the foot structure to the amount of pronation (Root et al.,1971; Gould, 1990). From a mechanical standpoint, structural abnormalities may or may not lead to hyperpronation but may instead lead to the inappropriate timing relationships in the joint actions of the lower extremity that may in turn lead to torsional stresses on tissues.

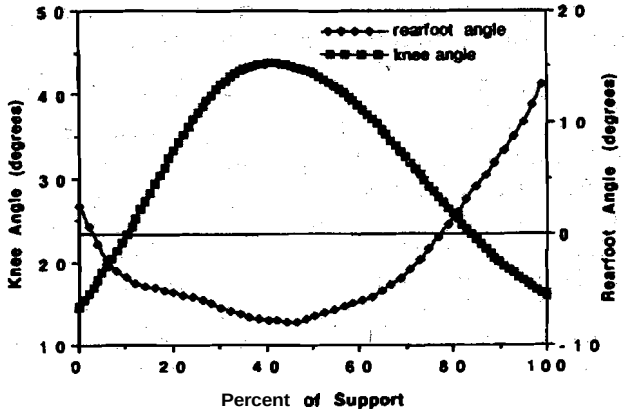

Figure 1. Knee and rearfoot angles during support.

An alternative model may be referred to as the "structural model". This model is based on the concept that the structural abnormality should be identified and its effect on the timing of the lower extremity joint should be clarified in order to understand the mechanism of injury and thus treat the injury. Figure 2 illustrates this model.

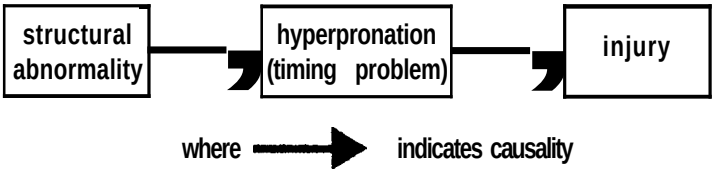

Figure 2. Structural Model of injury.

In the structural model, hyperpronation is seen as one possible effect rather than a cause. In rehabilitation, the crucial difference between the hyperpronation and structural models is that in the latter model the foot structure is treated and not the hyperpronation per se. The structural model also implies that if hyperpronation is present, it is a progressive problem in that the occurrence and severity will be dependent on the structural abnormality.

FOOT STRUCTURE ABNORMALITIES

There are many foot structure abnormalities that have been associated with injuries to the lower extremity. Two such foot structures, varus and valgus abnormalities, are clinically defined by the relationships between the leg, rearfoot, and forefoot in a non-weight bearing situation. Rearfoot varus and valgus are defined by the angle formed by a line that bisects the leg and one that bisects the calcaneus. Forefoot varus and valgus are defined by the angle formed by a line that bisects the leg and one that runs from the first to the fifth metatarsal joint. In the neutral foot the angle between the leg and rearfoot is 0" and 90° between the rearfoot and forefoot. This foot structure is illustrated in Figure 3a. Various combinations of rearfoot and forefoot varus and valgus abnormalities exist. The most common seen clinically are: combined rearfoot and forefoot varus (Figure 3b), forefoot varus (Figure 3c), and rearfoot varus (Figure 3d).

A forefoot varus deformity that causes the sub-talar joint to pronate excessively and the rearfoot to move into a valgus position during weight acceptance is referred to as a compensated forefoot varus. If the forefoot varus abnormality does not cause the foot to move into valgus, it is referred to as a non-compensated forefoot varus. There is a spectrum of states between these two extremes known as partially compensated forefoot varus. Generally the varus conditions are seen clinically more often than valgus deformities.

Two other foot structures that are associated with lower extremity injury are the equinus foot and the plantar flexed first ray foot. In the equinus foot the forefoot is more distal to the body than the rearfoot when the foot is dorsiflexed at 90°. The forefoot is plantar flexed relative to the rearfoot (McGlamry and Kitting, 197.3). If only the first metatarsal head is plantar flexed relative to the rest of the foot, then this condition is called a plantar flexed first ray. A rigid plantar flexed first ray is particularly important due to increases in the pressures on that structure during weight bearing activities. Both of these conditions are associated with poor shock absorption during ground contact.

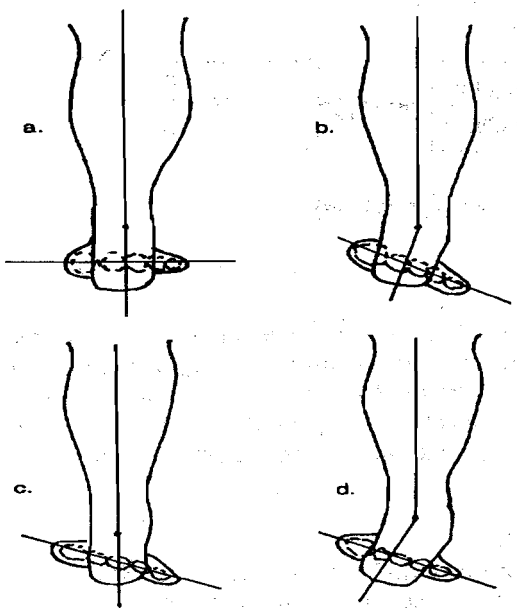

Figure 3. Diagnosis of the foot in a non-weight bearing state: a) neutral foot; b) combined rearfoot/forefoot varus; c) forefoot varus; and d) rearfoot varus.

MECHANISMS OF INJURY

Injuries to the lower extremity can be categorized as intrinsic, extrinsic, or intermediate. This categorization refers to the location of the injury. Intrinsic injuries are those to the foot structures. Intermediate injuries are those to structures that originate on the leg, cross the ankle joint, and insert on the bones of the feet. Extrinsic injuries are those to structures above the sub-talar joint. The most common injury associated with running, in the order of frequency, are: 1) knee pain; 2) shin splints; 3) Achilles tendinitis; 4) plantar fasciitis; and 5) stress fractures (James et al., 1978).

Intrinsic injuries include plantar fasciitis, metatarsalgia, sesamoiditis, bunions, and stress fractures. The major foot structural abnormalities associated with these injuries are non-compensated or partially compensated forefoot varus deformities. In both cases the rearfoot cannot compensate for the varus deformity. Since the forefoot must make contact with the ground during support, this deformity results in torsioning of the intrinsic foot structures. For example, plantar fasciitis may result from two mechanisms. First, there is increased deformation of the longitudinal arch resulting from the increased torque when a varus forefoot makes contact with the ground. This will cause the arch to flatten and stress the fascia more than normal. Secondly, there is a "wringing" effect when the forefoot torsions about the rearfoot. Plantar fasciitis may also result from late pronation during the push-off phase of support. As the heel rises, the plantar fascia tightens. This places very high forces on the fascia and inflammation may occur.

Intermediate structure injuries include shin splints and Achilles tendinitis. To use as an example, shin splints is a term used to describe tendinitis of the posterior and anterior tibialis, flexor digitorum longus, and flexor hallucis longus muscles at the origin on the tibia. These muscles cross the ankle and insert on the medial side of the foot. The mechanism of shin splints may also be the result of foot structural abnormalities. There are several suspected mechanisms but one such mechanism

involves the eccentric actions of these muscles. The eccentric action of these muscles is thought to decelerate the foot to counteract pronation during impact. Injury may result from an inability to eccentrically contract fast enough to match the high velocity of pronation. Any foot structure such as a forefoot varus structure will result in both greater pronation and greater velocity of pronation.

Extrinsic injuries include knee pain, particularly patello-femoral pain syndrome, and medial and lateral collateral sprains. These injuries may potentially be caused by foot structural abnormalities. The disruption of the timing of the functional relationships between lower extremity joint actions described earlier is a suggested mechanism for this type of injury. Patello-femoral pain syndrome is one injury that can be explained by this mechanism. As the sub-talar joint pronates. the knee flexes. the tibia internally rotates and the femur externally rotates. If the timing of these actions is disrupted, the patella may track more laterally out of the condylar groove causing an inflammation of the patella. Greater pronation values are evident in soft midsole shoes versus hard midsole shoes. The differences in the external/internal rotation values can be seen in Figure 4. In this graph it can be seen that there is less external rotation in the soft shoes where there is greater pronation. For example, in a forefoot varus deformity. the individual may pronate much later than normal. The resulting effect is that the knee extends while internal tibial rotation continues. External femoral rotation may not proceed to the extent that would occur normally, resulting in the vastus lateralis pulling the patella more laterally.

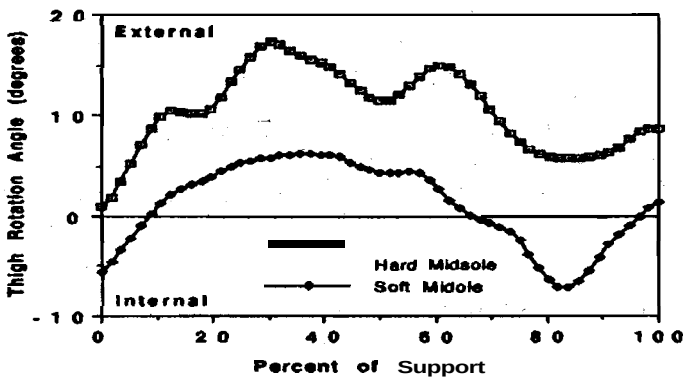

Figure 4. Internal/external rotation of the thigh during support.

RISK FACTORS FOR INJURY

While structural abnormalities may be considered the ovemding cause of chronic running injuries, the severity of the injury will not necessarily determine either the incidence or likelihood of injury. The severity of injury will be determined by other risk factors that are present for one with a structural abnormality. These factors include but are not limited to: 1) obesity; 2) exercise frequency and duration; 3) prior history of injury; 4) gait pattern; 5) footwear; and 6) nutritional deficits. Certainly these factors interact with the structural abnormalities. For the sake of brevity only two of these factors, gait patterns and footwear, will be discussed. Gait pattern refers to the foot contact pattern during the support phase of running. Mason (1980) identified three such patterns and labeled them as: 1) heel-toe where the initial ground contact in on the lateral aspect of the heel; 2) toe-heel-toe where the initial contact is on the forefoot followed by a rocking back on the heel; and 3) forefoot where the initial contact is on

the forefoot and the heel does not touch the ground. A gait pattern adopted by the individual that is not the result of a structural abnormality may combine with an abnormality and cause an injury. For example, a toeheel-toe runner requires unusually high eccentric contraction forces of the triceps surae in order to absorb the impact of contact and lower the calcaneus to the ground. If this pattern is combined with a rearfoot valgus resulting from a compensated foot, exceptionally high forces may be generated around portions of the Achilles tendon. The forefoot varus alone may be insufficient to produce a problem but the additional stresses on the Achilles tendon due to the gait pattern may result in injury.

Running footwear can also serve to accentuate structural deformities. For example, footwear with very soft midsoles may serve to increase pronation. It has been demonstrated that shoe midsole density influences the degree of pronation and the amount of time the foot is in pronation (Hamill et al., 1992). This effect can also be seen in racing flats versus training shoes with greater pronation evident in racing flats (Hamill et al., 1987). Late pronation may also be caused by foot structural problems. The combination of late pronation caused by a foot deformity and by the structure of a running shoe may be additive. For example, an individual with a forefoot varus abnormality who uses a soft midsole running shoe will probably pronate substantially later in the support phase resulting in a mis-timing of the lower extremity joint actions. The potential for injury thus may be simultaneously increased.

TREATMENT OF RUNNING INJURIES

James et al. (1978) reported methods of treatment used in a clinical study of individuals who had a running injury (Table 1). Two methods, rest and orthotics, dominated the successful methods of treatment. If the structural model of injury is accepted, then the structural problem of the individual must be considered. Rest alone will not be successful. Foot orthotics appear to be the most effective and successful treatment to compensate for structural abnormalities. An orthotic is not designed to cure an injury. It is designed to attenuate biomechanical effects rather than preventing hyperpronation. Theoretically, an orthotic acts mechanically to prevent a great amount of pronation and the possibility of inappropriate timing of lower extremity joint actions.

Table 1. Treatments used for running injuries (James et al., 1978).

1. rest	47%
2. orthoses	46%
3. reduced mileage	26%
4. shoe change/modifications	19%
5. steroid injection	17%
6. anti-inflammatory	14%
7. surgery	5%

The design of an orthotic involves the process of posting the insert. The degree and location of the posting must be made on the basis of the foot structural abnormality that is present rather than on the amount of hyperpronation present. For example, an individual with a fore foot varus condition must be accommodated by a forefoot varus posting and not simply a rearfoot posting to attenuate the amount of pronation.

SUMMARY

A biomechanical model has been presented that focuses on foot abnormalities as the etiology of specific running injuries. This model, called the structural model, differs from the hyperpronation model in that the ultimate cause of the injury is a foot

structural abnormality and not simply a pronation problem. Evidence to support this structural model is based on clinical experience since, unfortunately, there is little research in the scientific literature to support the model.

REFERENCES

Bates. BT et al. Foot function during the support phase of running. Running 4:24-31,1978.

Clarke, TE et al. The effects of shoe design parameters on rearfoot control in running. Med Sci Sports Exerc 15:376-381, 1983.

Clement, DB et al. A survey of overuse injuries. Phys Sports Med 9:47-58, 1981.

Gould, JA. Orthopedic and Sports Physical Therapy. St. Louis, MO: CV Mosby, 1990.

Hamill, J et al. Effects of shoe type on cardiorespiratory responses and rearfoot motion during treadmill running. Med Sci Sports Exerc 20:515-521, 1987,

Hamill, J et al. Timing of lower extremity joint actions during treadmill running. Med Sci Sports Exerc 27:807-813, 1992.

James, SL et al. Injuries to runners. Am J Sports Med 6:40-50, 1978.

Mason, BR. A kinematic and kinetic analysis of selected parameters during the support phase of running. unpublished doctoral dissertation, University of Oregon, 1980.

McGlamry, DE and RW Kitting. Equinus foot, an analysis of the etiology, pathology, and treatment techniques. J Am Pod Med Assoc 63:165-173, 1973.

Pagliano, J and O Jackson. The ultimate study of running injuries. Runner's World 15:42-48, 1980.

Root, ML et al. Biomechanical Evaluation of the Foot Vol. 1. Los Angeles, CA: Clinical Biomechanics, 1971.

Soderburg, GL. Kinesiology. Baltimore, MD: Williams and Wilkins, 1986.

Runner's World. Emmaus, PA: Rodale Press, October, 1971.

Runner's World. Emmaus, PA: Rodale Press, October, 1973.