CHAPTER TWO:

REVIEW OF LITERATURE
INTRODUCTION

The increased incidence of stress fractures to collegiate athletes competing today has resulted in a vast amount of clinical research. In an effort to better understand the underlying causes of stress fractures to prevent future injury, researchers have examined numerous factors that play a role in stress fracture formation. Most of the research to date has focused primarily on specific anatomical sites and type of activity associated with the formation of stress fractures, as well as, dramatic changes in training routine prior to injury.

Studies have also suggested that female athletes may be more susceptible to stress fractures than their male counterparts. For example, Sallis and Jones (1991) reported that the incidence of stress fractures was approximately 10-20% in females and 0.9-2.0% in males who participated in sports training. In the last decade, an increased amount of research has focused on the unique medical concerns of the female athlete. In particular, a condition referred to as the “female athlete triad” (disordered eating, amenorrhea and osteoporosis) and it’s subsequent affect on the health and performance of the female athlete. In particular, inquiries concerning bone health in the athletic woman have reached a heightened level of knowledge (Nattiv & Armsey, 1997). Current research has shown that physiological factors, such as decreased bone mineral density and improper biomechanics, may place women athletes at higher risk for stress fractures (Goldberg et al, 1994). Although the mechanisms underlying the complex interactions of mechanical, hormonal and nutritional factors on bone health have not been fully made clear, most scientists agree that an evaluation of bone health and stress injury to bone encompasses all of these factors as well as genetic predisposition (Nattiv & Armsey, 1997).
This review of literature will examine the nature of stress fractures as they occur in the athletic population, as well as, discuss the documented conditions and factors that are involved in the formation of stress fractures.

**DEFINITION OF STRESS FRACTURE**

A stress fracture is best defined as a partial or incomplete fracture resulting from an inability to withstand nonviolent stress that is applied in a rhythmic, repeated, subthreshold manner (Dugan & D’Ambrosia, 1983). The continuous pounding of running on a hard surface is one such example of this type of nonviolent stress. Stress fractures can occur in arms, hands, legs, feet and vertebrae and are not limited to individuals of a certain age, gender or level of physical activity. Among athletes, the incidence and location of stress fractures depends largely on the athlete’s particular sport or activity.

An important concept of stress fractures is that the injury itself is not the result of a single event, but rather, a gradual process of bone breakdown. Bone is a dynamic tissue that responds to changes in strain. Such changes may occur in both the magnitude of the load placed on the bone, as well as, the frequency with which the load is applied (Hough & Ray, 1994). The bone structure will generally adapt favorably when there is a gradual increase in strain. When different loads are applied to a bone in different ways, the bone gradually remodels itself when osteoblasts deposit additional bone where the strain is greatest and osteoclasts remove bone that is not loaded (Hough & Ray, 1994).

It is believed that stress fractures occur when the bone strain caused by loading patterns during repetitive activity surpasses the ability for the bone to adapt positively.
Several explanations for the phenomenon of stress fractures have been presented. Brahms, Fumich and Ippolito (1980) noted that the muscular system provides shock absorption for the body and when this system becomes fatigued, it loses efficiency and allows the biomechanical forces to be transmitted directly to the bone. As a result of these stresses, microscopic deformities occur in the bone that prompts the body’s normal remodeling process to begin. This is typically when the athlete first begins to notice symptoms of tenderness and pain. Because training usually continues throughout this phase, the development of a stronger bone matrix lags behind. This results in a weakened area of the bone that is then susceptible to fracture (Taunton, Clemmer & Webber, 1981). Another perspective on the etiology of stress fractures is that repetitive actions of muscles on bone increase the muscular tension across the bone, which results in microfractures and ultimately stress fractures (Jackson, 1991; Hough & Ray, 1994).

MEDIAL TIBIAL STRESS SYNDROME

Medial Tibial Stress Syndrome (MTSS) most commonly referred to as shin splints, is one of the most common injuries to athletes and usually the predecessor to tibial stress fractures. Pain associated with MTSS is at the medial aspect of the leg adjacent to the medial tibia. Tenderness is usually found between 3 and 12 centimeters above the tip of the medial malleolus at the posterio-medial aspect of the tibia (Pribut, 1998). When the tibia is palpated the tenderness is not directly medial, but just behind the most medial portion of the tibia, in the mass of soft tissue and at the bone itself. Periostitis sometimes occurs in this location. The sore, inflamed structures usually include the medial muscles and tendons. Most frequently involved is the posterior tibial
tendon and muscle, but the flexor digitorum longus and flexor hallucis longus may also be involved (Pribut, 1998). Clinically, physical examination can be used to differentiate between MTSS and a stress fracture. With MTSS, the tenderness extends along a considerable vertical distance of the tibia. When a stress fracture is present, tenderness is usually noted that extends horizontally across the front of the tibia. In addition, when the muscles over the tender area are stretched, pain usually results if the injury is MTSS.

**ANATOMICAL SITES OF STRESS FRACTURES**

Most stress fractures of track athletes, long distance runners and athletes who participate in predominantly running sports occur in the lower leg. Previous studies have shown the tibia, metatarsus, fibula, and femur to be the sites most frequently involved in stress fractures (Goldberg et al., 1994). Most studies have agreed that the tibia is the most common site of stress fractures in athletes (Matheson et al., 1987; Orava, 1980; McBryde, 1985).

Matheson et al., (1987) examined 320 stress fracture cases that occurred over four years. Their results showed that 49.1% of the cases occurred in the tibia, followed by the tarsals (25.3%) as the second most common site. The metatarsals made up only 8% followed by the femur (7.2%), and the fibula (6.6%). They concluded that the large number of tarsal stress fractures that were found may have been the result of the type of bone scan used to diagnose the fractures.

There have been some disputes in the literature concerning stress fractures of the tibia. Not all of the research supports the idea that the tibia is the most common site of
stress fractures. In his report, Devas (1969) found the most common stress fracture in athletes is of the metatarsal bone followed by the fibula.

Brukner et al., (1996) reviewed 180 consecutive stress fracture cases taken at a sports medicine center. Their findings revealed that among track athletes (n=54), the most common stress fracture sites were the tarsal navicular (32.2%), tibia (25.9%), metatarsals (16.7%) and the fibula (9.3%). As a whole for the entire study, the most common site was found to be the metatarsal bones. However, it was noted that this may have been due to the large number of dancers who were treated at the clinic. Dancers (42.9%) were the most common group who sustained metatarsal stress fractures.

**DRAMATIC CHANGES IN TRAINING ROUTINES**

One of the main causes of stress fractures consistently documented throughout the research is a sudden increase or dramatic change in training routines (Goldberg & Pecora, 1994; Taunton et al., 1981; James et al., 1978). It is estimated that clinical stress fractures stem from training errors 60 to 75% of the time (McBryde, 1985). Typical training errors that have been associated with stress fractures include a considerable increase in mileage, switching from running on flat surfaces to hills, a sudden change in training style or intensity and running on hard surfaces such as asphalt (Goldberg et al., 1994; James et al., 1978).

Improper training is especially important to competitive collegiate athletes since their training routine often changes several times throughout the course of the season. In particular and most importantly freshmen athletes, who are often not compliant with off-season workouts, encounter this sudden dramatic change in workout intensity when they
begin preseason workouts. This, combined with their decreased level of fitness, increases their risk for developing stress fractures.

Taunton and colleagues (1981) especially illustrated this dramatic change in training routine. They studied 62 subjects who had been diagnosed with stress fractures and found that the intensity of the training program was a major cause of stress fractures. They reported that 27% of the cases developed after a rapid commencement of training, 6% after sudden exposure to hills, 8% after a rapid increase in mileage and 10% after a particularly sever single session. In addition to this, they found that a combination of errors in training and changes in footwear were responsible for 44% of the stress fracture cases.

Goldberg and Pecora (1994) examined stress fractures in athletes at a major university and found that an abrupt change in training preceded 86% of the injuries sustained by the athletes. Of these, 69% resulted from a sudden increase in training that was usually after summer break, off-season semester, or a switch to a training program of greater intensity. They also discovered that 17% of the stress fractures followed an abrupt change in surface type, which is important since athletes typically train on different surface types depending upon the part of the season during which they are training.

In another study, James and colleagues (1978) reviewed 180 stress fracture cases and discovered that 60% of the injuries were associated directly with training errors. Specifically, excessive mileage of greater than 49 miles per week accounted for 29% of the stress fractures.

It is important to note that of the studies mentioned above, only one of them addressed errors caused by improper footwear. Most likely this is due to the fact that
today most running shoes have good heel stability and shock absorption. This may be one of the reasons why shoes are not indicated as a cause of stress fractures.

**STRESS FRACTURES IN FRESHMEN ATHLETES**

At this particular point, there is a lack of literature that has looked predominantly at the incidence of stress fractures in freshman athletes compared to other classes. One such study conducted by Goldberg *et al.*, (1994), compared college freshmen athletes to new military recruits who had shown a high incidence of stress fractures. They referred to freshmen athletes as essentially being “new recruits” who, much like the military recruit that is forced into a sudden increased training regimen, are quickly exposed to the demands of college sports. Goldberg and colleagues (1994) designed their study to see if indeed freshmen athletes demonstrated a higher incidence of stress fractures when compared with other college athletes. They discovered that freshmen athletes represented 30-35% of the athletic population and that over a 3-year period 67% of the stress fractures reported occurred in freshmen athletes. Goldberg *et al.*, (1994) reported that because the freshmen athletes are frequently unaware of the increased physical demands of college athletics, and are least compliant with off-season workouts, they are placed at a higher risk for stress fractures. Because freshmen athletes comprise such a large number of college athletes, increased research with this population would be beneficial.

**DIAGNOSIS OF STRESS FRACTURES**

The clinical diagnosis of stress fractures requires a careful patient history, full thorough examination and appropriate x-ray and bone scan studies (Dugan *et al.*, 1983).
It is important to document the onset of pain that is not associated with a particular event. The athlete suffering from a stress fracture will initially present symptoms of deep aching pain over a bony structure (Sallis et al., 1991). Initially, pain occurs only at the end of a workout. As time goes by, the frequency and intensity of the pain increases and it will occur earlier in the workout, as well as, at rest.

It is important to note that the delay in diagnosing stress fractures is usually caused by the symptoms resembling other more common overuse injuries of the leg (Orava and Hulkko, 1984). There are some tests that can be done to help distinguish between soft tissue injury and stress fractures. The most dramatic test during physical examinations of those suffering from stress fractures is palpation over narrowly defined bony surfaces resulting in pronounced exquisite tenderness (Hough et al., 1994). Soft tissue pain typically extends the length of the bone. A positive “hop-test” (pain at the site of a stress fracture reproduced by hopping on the injured leg) is another important test that can be administered when checking for stress fractures. As well as, the use of a percussive palpation technique using a tuning fork placed over the injured site. This test will typically elicit pain that will not be reproduced with soft tissue injuries (Sallis et al., 1991).

The physical examination should include evaluation of extremity alignment, length, knee function, ankle dorsiflexion with the knee extended and flexed, heel-leg alignment, and heel-forefoot alignment (James, Bates & Osternig, 1978).
RADIOGRAPHY VS. BONE SCAN

Radiographic studies have not been shown to be an accurate tool in the early diagnosis of stress fractures. A study by Giladi *et al.*, (1984) revealed that only 22% of the fractures identified were detected by radiography. From this, they concluded that radiography does not constitute a reliable tool for the early diagnosis of stress fractures. Similarly, Matheson *et al.*, (1987) reported that in 43.3% of cases presented, only 9.8% demonstrated abnormal radiographs. In addition, a number of studies including one conducted by McBryde (1985) have shown that it takes at least 2-4 weeks after the onset of symptoms for a stress fracture to appear on a radiograph. In contrast, technetium-99 bone scans can detect a stress fracture within several days after symptoms arise. Technetium-99 bone scans detect the increased osteoclastic and osteoblastic activity at the fracture site when the normal phosphorous in the bony lattice work is replaced by labeled agents (Blatz, 1981). This radionuclitide bone scan has been firmly established as a sensitive modality for the early detection of stress fractures, as well as, a number of other bone lesions (Mills, Marymont III & Murphy, 1980). It must be stressed however, that to effectively diagnose a stress fracture all of the tools mentioned previously must be utilized.

TREATMENT OF STRESS FRACTURES

Most stress fractures heal with rest only. Rest interrupts the osteoclastic activity and allows the osteoblastic activity to catch up resulting in periosteal and endosteal healing (Dugan *et al.*, 1983). Although the treatment of stress fractures is primarily aimed at minimizing the time that the athlete is unable to train and compete, it usually
takes an average of 3-8 weeks of relative rest before the fracture is completely healed. Taunton and colleagues (1981) reported that following the initial diagnosis, athletes were treated with a combination of modified rest, ice massage, anti-inflammatory medication, and physiotherapy for muscle retraining and flexibility. This last point is extremely important because rest tends to rapidly weaken trained muscles. As a result, weakened muscles are less able to absorb shock and prevent the transference of shock to the bone (Taunton et al., 1981). Thus, increasing the risk that the athlete will suffer another stress fracture.

Athletes with stress fractures usually respond well to treatment that includes active rest. Active rest is the use of training that improves or maintains cardiovascular function, muscular strength, flexibility and proprioceptive function during recovery in a manner that insures that weightbearing and impact forces on the injured bone are controlled (Hough et al., 1994). Cross training exercises should be tailored to mimic the type of training that the athlete would be doing if they were healthy. Alternative training methods such as bicycling, aqua jogging and swimming have been utilized successfully. These dynamic exercises do not cause the high momentary tensions on the leg that running does which allows the bone time to properly heal without compromising the athlete’s performance (Taunton et al., 1981).

Throughout the literature, it has been stressed that impact producing weightbearing activities, especially running, should be avoided until pain disappears, usually after the first four weeks (Hough et al., 1994). After this time if the clinical signs and symptoms have disappeared, the athlete can begin a gradual return to sports specific activities. However, if this is not the case, additional rest will be necessary. To
emphasize this point, James and colleagues (1978) report that a common mistake among athletes is to resume a vigorous training program too early before the fracture is completely healed only to suffer the reoccurrence of the asymptomatic injury. Many times this is the result of the athlete feeling pressured to return to competition because they are pain free. Most often, this pressure comes from the coaching staff who wants the athlete to return to full competition as soon as possible.

**RISK FACTORS**

*Biomechanical Abnormalities*

Many anatomic factors must be considered as risk factors for stress fractures. One of the main biomechanical problems repeatedly cited in the literature is excessive pronation. Excessive pronation has been linked to tibial stress fractures. During pronation the foot is everted, abducted, and plantar flexed (James *et al.*, 1978). It is normal for some pronation to occur during walking and running. However, as stated by Taunton and colleagues (1981), difficulties arise when pronation continues past midstance and into the propulsive phase of gait (Taunton, Clemmer & Webber, 1981). They go on to say that this occurs with the malalignment conditions of forefoot varus, subtalar varus, and tibial varum. In addition, they suggest that forefoot varus particularly predisposes the leg to internal tibial rotation, which if continually repeated over time, adds excessive stress to the mid and distal tibia. Thus, increasing the risk for stress fractures.

Prolonged pronation is not the only cause of stress fractures. Another commonly cited problem is a cavus foot. Both Taunton (1981) and James (1978) state that the cavus
foot is a relatively rigid, inflexible foot that is not well adapted to the accumulated impact loading of activities such as long distance running (Taunton et al., 1981; James et al., 1978).

**Female Athlete Triad**

The number of female athletes participating in collegiate sports today is increasing year after year. These athletes are faced with demanding training schedules and the pressure of performance expectations that are associated with competitive athletics.

Athletes driven to excel in their sport may develop a condition called the female athlete triad, a recently recognized syndrome consisting of three interrelated conditions. These distinct conditions are disordered eating, amenorrhea and osteoporosis.

Disordered eating includes behaviors of anorexia nervosa, or bulimia, which consists of bingeing and purging (purging may come in the form of exercise). These conditions are usually the result of the athlete’s obsessive fear of fatness and abnormal body image. Athletes believe that if they weigh less, they will perform better. It has been estimated that between 15-60% of female athletes suffer from eating disorders (Dueck, Matt, Manore & Skinner, 1996). The incidence varies greatly by sport. Most incidences occur in sports that focus on low weights for appearance or perceived performance (Bunt, Going, Westfall, Perry & Pamenter, 1989).

Amenorrhea is the second condition in the triad. It is defined irregularities in menstrual function (Bunt et al., 1989). The specific circumstances that initiate the onset and reversal of athletic menstrual dysfunction remain unclear. However, the evidence
that amenorrhea leads to damaging side effects such as musculoskeletal distress is strong. It has been shown that amenorrheic athletes typically show reduced levels of estradiol and progesterone that may prevent the formation of adequate bone density (Bunt et al., 1989). Estrogen has several important functions in promoting bone health. It increases the amount of calcium absorbed from the intestines, and reduces the amount of calcium lost in the urine. In addition, it helps keep more calcium in the bones themselves. The lack of estrogen in an amenorrheic athlete, combined with the lack of calcium in many women's diets, results in loss of bone density. Prolonged periods of low estrogen thus increase the risk for stress fracture.

Osteoporosis is the last part of the triad. This disease is characterized by low bone mass and deterioration of bone tissue. This makes the skeleton more fragile and leads to an increased risk of fracture.

**SUMMARY**

The increasing demand placed on athletes today to perform at higher and higher levels has exposed them more than ever to the risks of developing stress fractures. Freshmen athletes entering a collegiate program are exposed to these risks more so than any other class of athlete because they are not accustomed to such high levels of vigorous training. Identifying and understanding factors that increase the risk for stress fracture formation such as, when in an athlete’s season most injuries occur and in which sports would help tremendously in preventing such injuries. Sports medicine physicians, trainers, coaches and ultimately the entire athletic department would benefit from the
knowledge gained from such research. Fewer athletes would be forced out of competition and thus be able to contribute to the success of their respective teams.