



ELSEVIER
SAUNDERS

Clin Sports Med 25 (2006) 17–28

CLINICS IN SPORTS MEDICINE

Classification and Return-to-Play Considerations for Stress Fractures

Jason J. Diehl, MD^a, Thomas M. Best, MD, PhD^a,
Christopher C. Kaeding, MD^{b,*}

^a*Division of Sports Medicine, Department of Family Medicine, The Ohio State University Sports Medicine Center, 2050 Kenny Road, Columbus, OH 43221, USA*

^b*Division of Sports Medicine, Department of Orthopedics, The Ohio State University Sports Medicine Center, 2050 Kenny Road, Columbus, OH 43221, USA*

"Stress reactions" and "stress fractures" are fatigue-failure injuries of bone that are commonly diagnosed in competitive, occupational, and recreational athletes. These overuse injuries result in a mechanical failure of bone due to the accumulation of microdamage secondary to repetitive strain episodes. A stress reaction precedes a stress fracture in the bone fatigue-failure continuum and is hallmarked by bony microfailure without cortical disruption. These injuries are most common in the lower extremities of endurance and repetitive-motion athletes such as runners, jumpers, skaters, and soldiers [1,2]. Despite a female predilection in military recruits, in athletes, there seems to be no difference in incidence between men and women [3]. Next to prompt and proper diagnosis and treatment, return-to-play decision making can have the greatest impact on an athlete's recovery. It is therefore important that athletes be given the best evidence available to guide their time frame for return to sport and physical activity.

Bone stress-injury treatment and return-to-play decision making is based on the site of the injury and its corresponding potential for healing and risk of significant complication. Most stress fractures heal uneventfully with relative rest and activity modification. These fractures have recently been termed low-risk stress fractures [4]. Overtreatment of low-risk stress fractures can lead to deconditioning and unnecessary loss of training and competition. Conversely, there are anatomic sites that have a predilection for slow or incomplete healing, a high tendency for recurrence, or a significant risk of complication with progression of the fracture. Fractures at these locations are termed high-risk stress fractures [5]. To avoid prolonged or inadequate healing and potentially serious complications, it is important to be cognizant of these high-risk sites. Prompt diagnosis and treatment is key to minimize the impact of high-risk stress fractures on the athlete's career.

* Corresponding author. E-mail address: kaeding-1@medctr.osu.edu (C.C. Kaeding).

Successful treatment of stress fractures should not only focus on bony healing but also address reasons why the injury occurred in the first place. These reasons can be divided into intrinsic and extrinsic risk factors. Intrinsic risk factors include biomechanical, hormonal, and nutritional status. Common biomechanical factors leading to recurrent stress fractures include muscle weakness, low bone density, high longitudinal arches, leg-length discrepancies, and likely forefoot and knee varus [6–9]. Menstrual irregularities have also been linked to increased stress fracture incidence in general [10]. The most common extrinsic risk factor appears to be training errors. For example, training errors have been shown to increase the risk of stress fractures in military recruits who begin training in poor physical condition and have high volumes of training [11,12]. Individuals with a history of a previous stress fracture also are at higher risk of future injuries [13].

Return-to-play decision making is an integral part of the treatment of stress fractures in athletes. The development of the treatment plan should include consideration of intrinsic and extrinsic predisposing factors, classification and grade of the stress fracture, and the point in the athlete's career or competitive season at which he or she presents. The authors stress that each treatment is individualized for best recovery and prevention of future injuries. In this article, the authors briefly discuss the pathophysiology and diagnostic process of stress fractures and expand on the classification of stress fractures and its impact on return-to-play decision making based on the relative risk of the fracture.

PATHOPHYSIOLOGY

Stress fractures were first described in military recruits exposed to new cyclic loading activities. Repetitive loading alters a bone's microstructure through remodeling according to Wolff's law. The result is a stronger bone able to withstand greater loads [14]. When an athlete begins a new or increases a current exercise program, an increase in the number and size of microfractures within the bone occurs. The body responds by increasing osteoclastic and osteoblastic activity. Initially, osteoblastic activity lags behind the resorptive properties of the osteoclasts. This process leaves the bone susceptible to fatigue failure if the area is continually stressed without adequate time for repair [15,16]. An alternative hypothesis is that muscle dysfunction from overuse results in focal bending stresses that exceed the structural and physiologic tolerance of bone.

With continual strain episodes, fatigue failure proceeds through a progression of microfracture initiation, propagation, and eventual macrofracture. If the body does not repair the microscopic crack before additional loads are applied, then the fracture progresses across the bone. This process is termed crack propagation. Continued propagation and coalescence of the microcracks can result in macroscopic failure of the bone.

The balance between the creation/propagation of microcracks and the body's ability to repair them may be influenced by multiple factors. These

factors may include the athlete's hormonal, menstrual, and nutritional status; muscle function; and genetic predisposition. These factors continue to be studied and elucidated.

There is a continuum of fatigue failure of bone. Early accumulation of microdamage is often referred to as a stress reaction of bone. When a clear fracture line is visible on imaging (CT, MRI, or radiograph), a stress fracture has developed. Some consider a stress reaction as an early, low-grade stress fracture. How far the athlete's injury has progressed on this continuum of early microcrack formation to complete fracture has bearing on treatment, prognosis, and return-to-play considerations.

DIAGNOSIS

Most athletes who have stress fractures present with an insidious onset of progressive pain in a focal area [17]. Initially, pain is present only with activity and may be associated with localized muscular soreness. As the fracture continues to develop, the pain usually begins to affect performance, and with continued training, pain persists into activities of daily living. In more severe cases, pain becomes continual.

Physical examination usually demonstrates tenderness within a localized area. There may also be painful loss of joint motion or muscular tightness surrounding the injury. With more severe cases, there may be swelling or palpable irregularities at the fracture site.

Many imaging modalities are helpful in the diagnosis of stress fractures. Plain radiographs are useful when positive, but the findings may be subtle and may not accurately depict the severity of the injury. This is especially true if the radiograph is taken within the first few weeks of the injury. Bone scans have traditionally been used in the diagnosis of stress fractures because of their high sensitivity [18]. The sensitivity has been shown to be 84% to 100% within 3 days of symptoms [19]. Bone scans, however, are not as specific as other modalities. Bone scans are also time-consuming for the patient. Because the uptake can persist for months after clinical healing, bone scans are not as useful in follow-up care.

The two other imaging techniques that have become more useful in the diagnosis and management of stress fractures are CT and MRI. CT is less expensive and offers eloquent bony detail including fracture location, orientation, and extent and signs of healing. It is particularly helpful in areas such as the tarsal navicular to determine whether the fracture is complete or incomplete [20]. MRI has become the method of choice for many physicians because of its availability, speed, and the amount of information that is provided [21–23]. Its sensitivity is similar to that found with bone scan, but MRI is capable of showing more precise location and extent of fracture [24]. For tibial stress injuries, one study demonstrated the specificity and the positive predictive values to approach 100% [22]. T2-weighted images not only show the fracture site but also accurately confirm the degree of marrow edema [25].

CLASSIFICATION

The classification of stress fractures into high risk or low risk has been suggested by multiple authors [4,5,26]. This distinction allows the medical staff to quickly determine whether they can be aggressive or conservative with their decision to return an athlete to participation. Low-risk stress fractures include the femoral shaft, medial tibia, ribs, ulna shaft, and first through fourth metatarsals—all of which have a favorable natural history. These sites tend to be on the compressive side of the bone and respond well to activity modification. Low-risk stress fractures are less likely to recur, become a nonunion, or have a significant complication should it progress to complete fracture. Management of these injuries is discussed later and is guided primarily by the individuals' symptoms.

In comparison to low-risk stress fractures, high-risk stress fractures do not have an overall favorable natural history. With delay in diagnosis or with less aggressive treatment, high-risk stress fractures tend to progress to nonunion or complete fracture, require operative management, or recur in the same location [27]. High-risk stress fracture locations include the femoral neck, the patella, the anterior tibial diaphysis, the medial malleolus, the talus, the tarsal navicular, the proximal fifth metatarsal, and the first metatarsal phalangeal sesamoids. It is clear that location determines whether a stress fracture is low risk or high risk.

In addition to knowing the classification of a stress fracture as high versus low risk as determined by its anatomic site, the extent of the fatigue failure or the grade of the stress fracture is also needed to completely describe the injury. As mentioned earlier, stress injuries to bone create a continuum, from mild micro-failure to cortical disruption to complete fracture. There have been two previously published grading scales for the stress reaction/fracture continuum. The scale published by Arendt and Griffiths [21] has been used for the femur, tibia, fibula, navicular, calcaneus, and forefoot, whereas the scale published by Fredericson and colleagues [28] was developed using data for the tibia alone. Both scales consider grade 4 to be a complete stress fracture and grades 1 to 3 to be increasing levels of periosteal changes and marrow edema. One large study by Arendt and Griffiths [21] demonstrated that grade 3 and 4 injuries took longer to heal than grade 1 and 2 injuries. This study demonstrated that the grade of injury has prognostic implications regarding the time of healing. The management of bony stress injuries should be based on the location and grade of the injury. These two details give the amount of microdamage that has accumulated and whether it is a high- or low-risk injury. The following discussion focuses on treatment and return-to-play strategies for stress fractures depending on their anatomic location and associated classification as high or low risk.

MANAGEMENT OF AND RETURN-TO-PLAY STRATEGIES FOR LOW-RISK FRACTURES

Return-to-play decisions continue to challenge sports medicine practitioners. In the modern era of evidence-based decision making, practitioners continue to be faced with scenarios in which the best available evidence is expert opinion. Many factors need to be discussed with the athlete, and none of these is more

important than the risks of continued participation, particularly in the setting of an ongoing injury. Low-risk does not equate to no risk for any athlete with a stress fracture. A treatment plan should always be tailored to the individual's athletic and personal goals, with an honest discussion of the risks and benefits of continued participation versus relative or absolute rest. In the treatment of low-risk stress fractures, a major consideration is often where the athlete is in his or her competitive season.

Athletes at the end of a competitive season or in their off season often desire to be healed from their stress fracture before resumption of preseason training or competition. For these athletes, treatment includes relative rest and activity modification to a pain-free level. The acceptable level of activity differs for each athlete and may include discontinuation of only the aggravating activity alone, discontinuing all training activities, or placing the patient on non-weight-bearing status (Table 1). To maintain fitness, athletes should be allowed to cross-train if it is pain-free. Frequent cross-training activities include cycling, swimming, and aqua-running. Low-risk stress fractures usually heal when the athlete is limited to pain-free activity for 4 to 8 weeks [26]. This period of healing is an ideal time to assess the modifiable risk factors that may decrease the chance for recurrent injury. Gradual increase in activity (activities of daily living) should begin after the athlete is pain-free and the site is nontender [26].

Table 1
Low-risk stress fracture treatment guide

Symptoms	Goal	Treatment suggestions
Any level of pain	Heal injury	Titrate activity to a pain-free level for 4–8 w depending on the grade of injury Braces/crutches Modify risk factors
Pain with no functional limitations	Continue participation	Titrate activity to a stable or decreasing level of pain Closely follow Modify risk factors
Pain with functional limitation	Continue participation	Decrease activity level to point at which pain level is decreasing and until a functional level of pain has been achieved, then titrate activity to stable or continued decrease level of pain Modify risk factors
Limiting pain intensifies despite functional activity modification (ie, unable to continue to perform at any reasonable functional level despite activity modification)	Heal injury	Complete rest Immobilization Surgery Modify risk factors

Although there are no supporting data, a common recommendation is that no more than a 10% increase in activity should be added per week of training.

In contrast, athletes in the midst of their competitive season with low-risk stress fractures often desire to finish the season and treat for a cure later. A treatment plan for these individuals should be based on their ability to function at the time of diagnosis. Those athletes who present with pain without functional limitation should be able to continue participation, using pain to guide their activities (see [Table 1](#)). They may continue to train and compete at a level that does not cause the pain to intensify. Hence, relative rest and activity modification are titrated to the level of discomfort. When athletes who have low-risk stress fractures have pain that limits their function, they should be treated with activity modification for training and activities of daily living until the pain decreases to a functional level. The goal is to decrease the repetitive stress at the fracture site enough to allow the body to restore the dynamic balance between damage and repair. This strategy may include decreasing volume and intensity of activity, equipment changes, technique changes, or cross-training. One benefit to such a strategy is that the athlete typically does not suffer a substantial loss of conditioning or competition while allowing their body to repair the bone injury. If their pain intensifies and activity modification alone is inadequate for recovery, then treatment should be intensified to include complete rest, immobilization, or surgical intervention.

Athletes diagnosed with a stress reaction or low-grade injury at a low-risk site should follow treatment guidelines similar to those of a higher-grade injury. The major difference is the expected time of treatment and the degree of relative rest required. It has been shown that grade 1 and 2 stress reactions can heal with no changes in activity level [29]. In Arendt and Griffith's [21] study, return to full activity for early stress injuries (grade 1, 3.3 weeks; grade 2, 5.5 weeks) was significantly faster than for more severe injuries (grade 3, 11.4 weeks; grade 4, 14.3 weeks). This finding stresses the importance of temporarily modifying activity to the level at which symptoms and injuries do not progress. This study also demonstrated the value of grading the severity of the fatigue failure. A low-grade stress fracture at a low-risk site has a better prognosis for time to recovery than a higher-grade injury at the same low-risk site.

In addition to modification of activities, there are treatment modalities that have been shown to increase the healing rate and decrease the time to return to play. For example, in the treatment of tibial stress fractures, the use of pneumatic leg braces has been effective in reducing healing time [30]. It is also important to evaluate the nutritional history, menstrual history, training schedule, and equipment use of any athlete with a bone stress injury before returning the athlete to play.

MANAGEMENT OF AND RETURN-TO-PLAY STRATEGIES FOR HIGH-RISK FRACTURES

Return-to-play considerations for athletes who have high-risk stress fractures are more difficult than for low-risk stress fractures ([Table 2](#)). In general, return

should be recommended only after proper treatment and complete healing of the injury. As previously mentioned, high-risk stress fractures have more frequent complications such as delayed union, nonunion, and refracture. High-risk stress fractures also have a significantly poorer prognosis should they progress to complete fracture. The treatment for high-risk stress fractures should be based on the immediate goal of preventing any progression of the fracture and avoiding delayed healing, nonunion, or refracture. Depending on the exact site and grade of the high-risk stress fracture, this treatment often requires absolute rest or internal fixation. Avoiding delay in diagnosis of a high-risk stress fracture is key to minimizing the risk of potential complications. If radiographs do not demonstrate a stress fracture but the history and examination are concerning for a stress fracture in a high-risk area, then further imaging should be pursued to exclude a stress fracture before returning the athlete to play [5]. Because of the significant complications associated with injury progression, it is suggested that individuals who have evidence of a high-risk stress fracture do not continue to participate in their activity. Depending on the site of the high-risk stress fracture, prolonged immobilization with non-weight-bearing restrictions or operative management are often the treatments of choice.

The low-grade injury at a high-risk location should be managed with the goal of healing based on the individual's risk for reinjury and desired speed of recovery. Most early stress reactions at high-risk sites (grades 1 and 2) heal with nonoperative management [5]. A period of rest to eliminate the individual's symptoms and a return to training with activity modification is suggested for early stress reactions at high-risk sites.

The key difference between a low-grade stress fracture at a high-risk versus a low-risk location is that an individual who has a low-grade fracture at a low-risk site can be allowed to continue to compete but an individual who has a low-grade fracture at a high-risk site needs to heal before full return to activity. This difference is due to the marked increased risk of significant complication with fracture progression at the high-risk site. This risk is unacceptable in the vast majority of cases. For example, a runner may be allowed to continue to run with stable and tolerable pain with a second metatarsal stress fracture but not a superior femoral neck stress fracture. The risk associated with progression of the second metatarsal fracture is minor compared with the risk of progression of the femoral neck stress fracture. Regardless of grade and location, the risk of continued participation should be discussed with each athlete.

The management of each fracture should be individualized. For example, a recreational runner with a low-grade stress reaction of the fifth metatarsal can be treated with an ankle-foot orthosis boot and cross-training until symptoms resolve and healing is noted radiographically. Cross-training can maintain fitness at high levels [31,32].

Highly competitive athletes who place a greater amount of stress at these high-risk locations are at increased risk for fracture progression and reinjury and, in the opinion of the authors, should be managed aggressively. For example, a competitive runner with a grade 3 stress reaction of the fifth meta-

Table 2
Management of and return-to-play strategies for high-risk stress fractures

Anatomic site	Complications	Suggested treatment	Level of data
Femoral neck	Displacement Nonunion Avascular necrosis	Tension: Strict NWB or bed rest Surgical fixation RTP when healed Compression: NWB until pain-free with radiographic evidence of healing, then slow activity progression RTP after no pain on examination or with any activities Surgical fixation (optional)	Level C (expert opinion) Level D (case series)
Anterior tibia	Nonunion Delayed union Fracture progression	Nonoperative: NWB until pain-free with ADL; pneumatic leg splints RTP with slow progression after nontender and pain-free with ADL (9 mo) Operative: Intramedullary nailing RTP is usually faster (2–4 mo)	Level A (RCT) Level B (nonrandomized) Levels C and D
Medial malleolus	Fracture progression Nonunion	Nonoperative: (No fracture line) 4–6 wk pneumatic casting Avoid impact; rehabilitation RTP when nontender, no pain with ADL Operative: (Fracture line, nonunion, or progression) ORIF with bone graft	Levels C and D
Tarsal navicular	Nonunion Delayed union Displacement	Nonoperative: NWB cast 6–8 wk, then WB cast 6–8 wk RTP is gradual after pain-free with ADL Orthotics and rehabilitation suggested Operative: (Complete, nonunion) RTP only when healed	Levels C and D

Talus	Nonunion Delayed union	Nonoperative: NWB cast 6–8 wk RTP is gradual after pain-free with ADL Orthotics and rehabilitation suggested Operative: Reserved for nonunion Nonoperative: (Nondisplaced) Long-leg NWB cast 4–6 wk Rehabilitation following RTP is gradual after pain-free with ADL Operative: Horizontal—ORIF Vertical—lateral fragment excision RTP when healed	Level C
Patella	Displacement Fracture completion	Nonoperative: NWB 6–8 wk RTP is gradual after pain-free with ADL Operative: Excision if fail nonoperative Nonoperative: (No fracture line) NWB cast 4–6 wk followed by WB cast until healed RTP after nontender and pain-free Operative: (Fracture line, nonunion, or individual at high risk for refracture) Intramedullary screw fixation RTP 6–8 wk, early ROM/rehabilitation	Level C
Seasmooids	Nonunion Delayed union Refracture	Nonoperative: NWB 6–8 wk RTP is gradual after pain-free with ADL Operative: Excision if fail nonoperative Nonoperative: (No fracture line) NWB cast 4–6 wk followed by WB cast until healed RTP after nontender and pain-free Operative: (Fracture line, nonunion, or individual at high risk for refracture) Intramedullary screw fixation RTP 6–8 wk, early ROM/rehabilitation	Level C
Fifth metatarsal	Nonunion Delayed union Refracture	Nonoperative: NWB 6–8 wk RTP is gradual after pain-free with ADL Operative: Excision if fail nonoperative Nonoperative: (No fracture line) NWB cast 4–6 wk followed by WB cast until healed RTP after nontender and pain-free Operative: (Fracture line, nonunion, or individual at high risk for refracture) Intramedullary screw fixation RTP 6–8 wk, early ROM/rehabilitation	Levels C and D

Abbreviations: ADL, activities of daily living; NWB, non-weight bearing; ORIF, open reduction with internal fixation; RCT, randomized controlled trial; ROM, range of motion; RTP, return to play; WB, weight bearing.

tarsal should consider operative management because of the reported high incidence of recurrence at the same site after healing with nonoperative methods. This aggressive treatment may prevent the athlete from having a repeat injury in a subsequent season. Similarly, running athletes with anterior cortex stress fractures of the tibia may return to activity faster with surgical management compared with conservative treatment.

In summary, surgical intervention may be considered for high-risk stress fractures for several reasons. Surgical intervention may speed healing of the fracture and allow earlier return to play (the tibial shaft dreaded black line is often nailed to achieve this goal). Another indication for surgical intervention may be to prevent refracture (eg, the internal fixation of a proximal fifth metatarsal stress fracture). A third indication for surgery is to prevent a catastrophic fracture progression (eg, the internal fixation of a femoral neck stress fracture).

UPPER EXTREMITY

Upper-extremity stress fractures account for less than 10% of all stress fractures and are commonly found in throwing athletes and rowers. There has been increased attention focused on upper-extremity stress fractures in recent years. Upper-extremity stress fracture sites are primarily considered low risk and heal with activity modification alone. The one exception may be an olecranon stress fracture in a competitive thrower. This stress fracture heals well before a fracture line forms with conservative management [33]; however, if a true stress fracture line is found in a throwing athlete's olecranon, then the individual may be better served with internal fixation (level C and D) [34].

SUMMARY

Stress fractures are common injuries, particularly in endurance athletes. Stress fracture management should take into consideration the injury site (low versus high risk), the grade (extent of microdamage accumulation), and the individual's competitive situation. Low-risk stress fractures usually respond well to nonoperative management, and treatment is largely guided by the patient's symptoms. High-risk stress fractures should be treated more aggressively with absolute rest or surgical fixation, with the goal of fracture healing and minimizing risk of complete fracture or refracture. The overall goal of treatment is to allow activity and avoid deconditioning when appropriate, while minimizing the risk of significant complication to the athlete. Overtreatment of low-risk stress fractures can result in unnecessary deconditioning and loss of playing time. Undertreatment of a high-risk stress fracture may place the athlete at risk of a significant complication, putting the athlete's career at risk. Understanding the classification and grade of stress fractures and their implications on return-to-play decisions is key to optimal care of the athlete.

References

- [1] Matherson GO, Clement DB, McKenzie DC, et al. Stress fractures in athletics: a study of 320 cases. *Am J Sports Med* 1987;15:46–58.

- [2] Kadel NJ, Teitz CC, Kronmal RA. Stress fractures in ballet dancers. *Am J Sports Med* 1992; 20:445–9.
- [3] Bennell KL, Malcolm SA, Thomas SA, et al. The incidence and distribution of stress fracture in the competitive track and field athletes. *Am J Sports Med* 1996;24:211–7.
- [4] Boden BP, Osbahr DC, Jimenez C. Low-risk stress fractures. *Am J Sports Med* 2001;29: 100–11.
- [5] Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. *Am Acad Orthop Surg* 2000;8(6):344–53.
- [6] Korpelainen R, Orava S, Karpakka J, et al. Risk factors for recurrent stress fractures in athletes. *Am J Sports Med* 2001;29:304–10.
- [7] Cowan DN, Jones BH, et al. Lower limb morphology and risk of overuse injury among male infantry trainees. *Med Sci Sports Exerc* 1996;28:945–52.
- [8] Hoffman JR, Chapnik L, Shamis A, et al. The effect of leg strength on the incidence of lower extremity overuse injuries during military training. *Mil Med* 1999;164:153–6.
- [9] Bennell KL, Matherson G, Meeuwisse W, et al. Risk factors for stress fractures. *Sports Med* 1999;28:91–122.
- [10] Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am J Sports Med* 1988;16:209–16.
- [11] Almeda SA, Williams KM, Shaffer RA, et al. Epidemiological patterns of musculoskeletal injuries and physical training. *Med Sci Sports Exerc* 1999;30:1176–82.
- [12] Gardner LI, Dziados JE, Jones BH, et al. Prevention of lower extremity stress fractures: a controlled trial of shock absorbent insole. *Am J Public Health* 1988;78:1563–7.
- [13] Giladi M, Milgrom C, Kashtan H, et al. Recurrent stress fractures in military recruits. One-year follow-up of 66 recruits. *J Bone Joint Surg Br* 1986;68:429–41.
- [14] Beck BR. Tibial stress injuries: an aetiological review for the purposes of guiding management. *Sports Med* 1998;26:265–79.
- [15] Bennell KL, Malcolm SA, Wark JD, et al. Models for the pathogenesis of stress fractures in athletes. *Br J Sports Med* 1996;30:200–4.
- [16] Johnell O, Rausing A, Wendeberg B, et al. Morphological bone changes in shin splints. *Clin Orthop* 1982;167:180–4.
- [17] Jones BH, Thacker SB, Gilchrist J, et al. Prevention of lower extremity stress fractures in athletes and soldiers: a systematic review. *Epidemiol Rev* 2002;24:228–47.
- [18] Wilcox JR, Moniot AL, Green JP. Bone scanning in the evaluation of exercise-related stress injuries. *Radiology* 1977;117:699–703.
- [19] Couture CJ, Karlson KA. Tibial stress injuries. *Physician Sports Med* 2002;30:29–37.
- [20] Torg JS, Pavlov H, Freiburger RH. Stress fractures of the tarsal navicular. A retrospective review of twenty-one cases. *J Bone Joint Surg Am* 1982;4:700–12.
- [21] Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. *Clin Sports Med* 1997;16: 291–306.
- [22] Gaeta M, Minutoli F, Seribano E, et al. CT and MR imaging findings in athletes with early tibial stress injuries. *Radiology* 2005;235:553–61.
- [23] Lee JK, Yao L. Stress fractures: MR imaging. *Radiology* 1988;169:217–20.
- [24] Stafford SA, Rosenthal DI, Gebhardt MC, et al. MRI in stress fracture. *AJR Am J Roentgenol* 1986;147:553–6.
- [25] Amendola A. MRI of foot and ankle: the orthopedic surgeon's perspective in ractical MR imaging of the foot and ankle. Boca Raton (FL): CRC Press LLC; 2000.
- [26] Brukner P, Bradshaw C, Bennell K. Managing common stress fractures: let risk level guide treatment. *Physician Sports Med* 1998;26(8):39–47.
- [27] Orava S, Hulkko A. Delayed unions and nonunions of stress fractures in athletes. *Am J Sports Med* 1988;16:378–82.
- [28] Fredericson M, Bergman AG, Hoffman KL, et al. Tibial stress reactions in runners; correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. *Am J Sports Med* 1995;23:472–81.

- [29] Chisin R, Milgrom C, Giladi M, et al. Clinical significance of nonfocal scintigraphic findings in suspected tibial stress fractures. *Clin Orthop* 1987;220:200–5.
- [30] Swenson EI, DeHaven KE, Sebastianelli WJ, et al. The effect of a pneumatic leg brace on return to play in athletes with tibial stress fractures. *Am J Sports Med* 1997;25(3):322–8.
- [31] Eyestone ED, Fellingham G, George J, et al. Effect of water running and cycling on maximum oxygen consumption and 2-mile run performance. *Am J Sports Med* 1993;21:41–4.
- [32] Frangolias DD, Taunton JE, Rhodes EC, et al. Maintenance of aerobic capacity during recovery from right Jones' fracture. *Clin J Sports Med* 1997;7:54–8.
- [33] Schickendantz MS, Ho CP, Koh J. Stress injuries of the proximal ulna in professional baseball players. *Am J Sports Med* 2002;30:737–41.
- [34] El Attrache NS, Ahmad CS. Valgus extension overload syndrome and olecranon stress fractures. *Sports Med Arthroscopy Rev* 2003;11:25–9.