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Stress Fracture Management: Current Classification and New Healing Modalities

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Stress or fatigue fractures are readily appreciated in sports medicine. Although the general principles of rest, activity modification, and analgesia are accepted by the injured athlete, return to play is a key focus, particularly for competitive athletes. Any intervention will be tried in the hope that it will aid recovery and accelerate return to play. The factors determining prognosis will be discussed on the basis of upon the site of the fracture and the imaging characteristics. This article will also present the general management principles and the factors that guide return to play decisions. There are many new stylistic modalities that are being adopted in an attempt to stimulate bone healing. Current evidence for the biological therapies of hyperbaric oxygen therapy, bisphosphonates, growth factors, bone morphogenic protein, and recombinant parathyroid hormone will be considered, together with the physical modalities of ultrasound and electromagnetic field stimulation.
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Stress or fatigue fractures are now readily appreciated in the sports medicine and sports surgery setting. Stress fractures occur in bone of normal quality with repeated loading at a magnitude well below the ultimate strength of the bone. Insufficiency fractures occur in bone with reduced mechanical properties, usually associated with low bone density. In athletes who have a normal diet and train and bear load normally, the bone quality is generally normal. Low bone density may occur in women with poor diet and menstrual disturbance related to exercise. This may render the sportswoman more susceptible to insufficiency fracture.

Wolff in the 1890s recognized that bone responds and strengthens in response to stresses placed upon it.[1] The remodeling and strengthening of bone takes time and in the presence of sustained repeated loads, microdamage may occur. At a microscopic level, the applied stresses lead to both increased osteoclastic and osteoblastic activity. Given that in the bone remodeling cycle, there is a lag time between bone resorption and bone formation, continued loading of a weakened area may lead to the development of microdamage.[2] From this damaged area, a crack may propagate as a fracture and ultimately as bone failure with the application of a normally sustainable load.[3]

As development of stress fractures occurs along a continuum athletes may seek medical attention at any stage along this process. The bone stress may become symptomatic causing the athlete to modify their activity, or the bone may ultimately fail resulting in a fracture that prevents activity.

Bone stress responses will settle with time, activity modification, and reversing the cause of the increased stress. The general principles of rest and activity modification are easily tolerated in the general population; however, in the sporting community and, in particular, professional sports, prompt return to training and play are at the forefront of players, medical staff, and coaches' minds. Any intervention will be tried in the hope that it will aid recovery and accelerate return to play. There are many new modalities that are being adopted to potentially stimulate bone healing. Surgical intervention may be considered either early in the management or as a last resort for certain fractures. This article presents the general management principles and the factors that guide return to play. The science behind and the evidence for the use of fashionable new modalities will also be considered.

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General Management Principles

The general principles behind the management of fractures include rest, immobilization and analgesia. The sports medicine community is also well aware of the important role of physiotherapy and rehabilitation to maintain aerobic fitness during the recovery period, apply graduated bone loading to assist with healing and to correct potential predisposing factors to prevent stress fracture recurrence (Table 1).

The repair of stress fractures occurs by a process different from the repair of acute fractures. Acute fracture repair occurs by the formation of cartilage callus and by proceeding through the stages of endochondral ossification to consolidation and mineralization. Only after initial stabilization does the bone remodeling begin to reconstruct the lamellar bone structure. The healing of stress fractures works directly and only through bone remodeling, which resorbs the damaged region and then replaces it with new bone. Resportion is at a maximum at 4 weeks postinjury, at which point intracortical formation is at its highest.[5]

Table 1 Risk Factor Assessment Either as Primary Prevention or as Part of Secondary Prevention in Someone Presenting With a Stress Fracture (Reprinted with permission.[4])

Risk Factor	Variables
Training	• Type • Volume • Intensity • Surface • Changes in training
Footwear	• Type • Age of shoe • Use of insoles
Lower limb alignment	• Foot type • Tibial torsion • Knee varus/valgus • Femoral anteversion • Leg length
Muscle function	• Muscle strength and endurance particularly of the calf muscles
Muscle length and joint range	• Flexibility of calf, hamstrings, hip flexors • Range of ankle dorsiflexion, hip internal/external rotation
Menstrual status	• Current and past menstrual patterns • Use of the oral contraceptive pill • Sex hormonal levels if irregular
Bone density–dual energy x-ray absorptiometry (DXA)	• If amenorrheic or multiple stress fracture history
Dietary intake	• Calcium • Energy • Other nutrients influencing absorption of calcium or bone health, for example, protein, fiber • Presence of eating disorder

Most stress fractures with a relatively brief history of symptoms will heal with rest and activity modification alone, and permit return to sport within the 4-8 weeks.[6] Immobilization is rarely used for most stress fractures, given the well-known detrimental effects of immobilization on other structures, such as muscle, ligaments, and joints and the fact that most will heal without it. However, there are some stress fracture sites in which immobilization may be the preferred treatment method. These include the tarsal navicular, the sesamoids, the patella, and the posteromedial tibia. The use of a pneumatic air brace may assist with stress fracture healing in the leg and reduce the time taken to return to sport. Swenson et al[7] propose that the pneumatic leg brace shifts a portion of the weightbearing load from the tibia to the soft tissue, which results in less impact loading. They also suggest that the brace facilitates healing at the fracture site by compressing the soft tissue, thereby increasing the intravascular hydrostatic pressure and resulting in a shifting of the fluid and electrolytes from the capillary space to the interstitial space. This theoretically enhances the piezoelectric effect and osteoblastic bone formation.[8]

Two randomized controlled trials, 1 in military recruits[9] and 1 in athletes,[7] showed a significant reduction in the time to recommence training after diagnosis of stress fracture, using a pneumatic leg brace (weighted mean difference, 42.6 days, 95% CI, 55.8-29.4 days). Gillespie and Grant[10] found the median time from the initiation of treatment to the completion of a standard functional progression program was 21 days in the brace group ($n = 8$) as compared with 77 days in the conventional group ($n = 10$). However, a recent study with similar numbers of military recruits with tibial stress fractures ($n = 20$ completing) failed to find an effect of the brace on time to pain-free hop and run.[11] Although results are conflicting, there is some evidence to suggest that a pneumatic brace accelerates return to activity[10] in fibular or tibial stress fractures. A similar brace and modified rest and rehabilitation has also been used in high risk anterior cortex tibial fractures in athletes who became symptom-free and returned to their previous level of activity at a mean of 12 months.[12]

Athletes regularly take over-the-counter analgesics to control symptoms. These include acetaminophen and/or paracetamol and non–steroidal anti-inflammatory drugs (NSAIDs). NSAIDs inhibit the cyclo-oxygenase pathway responsible for transforming arachidonic acid to prostaglandins, prostacyclins, and thromboxanes. These, in turn, have been shown to play a major role in bone formation, resorption, and repair.[13] NSAIDs may, therefore, theoretically delay fracture healing. A recently published review commented that there is no conclusive evidence to document any effect of NSAIDs on stress fracture healing in human beings, but the authors recommend limited use of it[14] and generally are not in favour of using it in the treatment of stress fractures.[15] The only exception to this may be the short-term use for analgesic purposes in the early stages, when they provide improved analgesia when used in addition to other analgesics.

Healing may be improved by the optimization of biomechanical alignment and gait kinematics. In the presence of abnormality, orthotic insoles are thought to offer benefits making minor adjustments to alignment and reducing abnormal loading stresses, thus aiding healing and preventing recurrence. There are, however, many misconceptions associated with insole use and footwear selection, for example, the generalized belief that a neutral running shoe provides pressure alleviation for cavus feet.[16] This has led to the development of prospective research in insoles use, based on the location of injury and elevated plantar pressure. Customized semi-rigid foot orthoses have been shown to have moderate to large beneficial effects in treating and preventing posterior tibial stress fractures.[17] A Cochrane review revealed that the use of shock absorbing inserts in footwear probably reduces the incidence of stress fractures in military personnel, but this has not been seen in studies in athletic cohorts. There was insufficient evidence to determine the best design of such inserts, but comfort and tolerability should be considered.[18]

Rearfoot type, alignment, and function are likely to influence the occurrence of stress fractures in the lower limb and foot, although results of studies have not necessarily been consistent. Athletes with lower limb stress fractures were 40% more likely to have high-arched feet than uninjured controls, and in this group tibial and femoral stress fractures were more prevalent.[19,20] Cavus or supination deformity places increasing stresses on the outer aspect of foot leading to fifth metatarsal base stress fractures,[3,21,22] whereas forefoot hyperpronation leads to increased risk of fibula fractures.[23] Greater rearfoot movement and reduced ankle dorsiflexion secondary to tightness of the gastro-soleus complex have been associated with risk of stress fractures.[24] A Morton's foot, with a short hypermobile first ray and long second ray, has been associated with second metatarsal fractures,[25] although other studies have suggested than this is not a direct relationship.[26]

Classification: Risk and Grade

Stress fractures have been classified into either high- or low-risk stress fractures[6] according to their recognized healing pattern, imaging characteristics, risk of fracture propagation, and catastrophic failure. Low-risk fractures are likely to heal with simple measures alone,[27] whereas high-risk injuries are likely to progress to delayed or established nonunion causing chronic problems.[28] In a series of 369 cases of stress fractures in athletes by Orava and Hulkko,[29] 10% of cases progressed into delayed or nonunion. Of these, fractures of the sesamoid, the middle third of the tibia, and the fifth metatarsal base were the most common.

Established nonunion will be easily detectable on plane radiographs with the classical appearance of cysts, sclerosis, and intramedullary bone formation, commonly seen in the fifth metatarsal base.[30] The acute displacement of high-risk fractures is dramatic and may be catastrophic, depending on the location, for example, anterior tibial stress fractures and tensile femoral neck fractures, but because of pre-existing symptoms this hopefully can be prevented. Certain stress fractures are well known to be particularly slow to unite and may cause significant problems if they become completely or acutely displaced. These include fractures of the femoral neck (tension side), the tibia anterior cortex, the patella, the medial malleolus, the talus, the tarsal navicular, the fifth metatarsal, and the sesamoids.

Owing to the problems associated with high-risk fractures, internal fixation may be considered as a first-line treatment. Low-risk stress fractures will heal with conservative measures and patients may bear weight according to their comfort level, for example, calcaneum and metatarsal shafts[31,32] (Fig. 1, Table 2).

The diagnosis and prediction of recovery are aided by radiological imaging (Table 3). When plain radiography, ultrasound, and bone scintigraphy were compared for the diag-

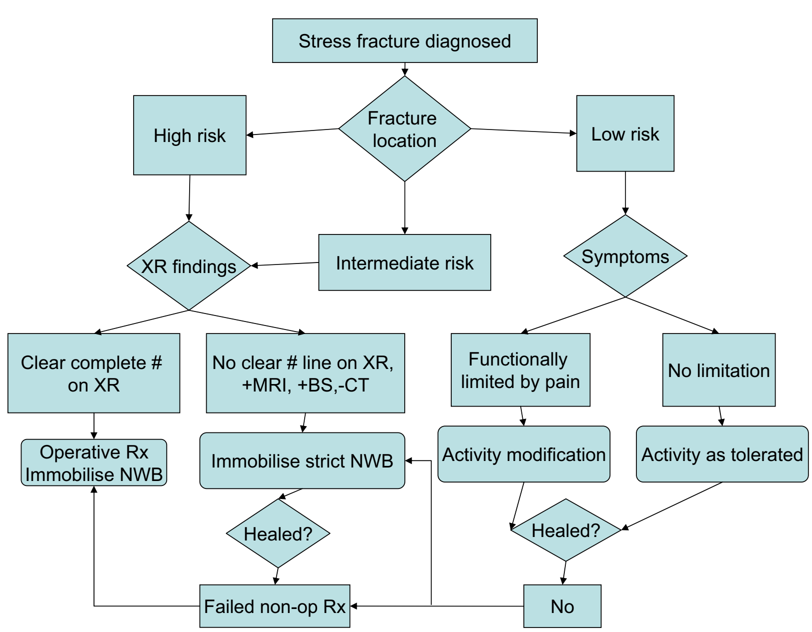

Figure 1 Algorithm for the management of lower extremity stress fractures. (Reprinted with permission.[33])

Table 2 Treatment of a Stress Fracture (Reprinted with permission.[4])

Treatment	Strategies
Decide on overall management approach	• Consider site of stress fracture • If problematic, may require special treatment • Decide on stage of continuum of bone strain • Use of appropriate diagnostic procedures
Relieve pain and any swelling	• Gait aids if necessary • Ice • Electrotherapy modalities
Accelerate repair and remodeling	• Potential therapies not yet proven • Low-intensity pulsed ultrasound • Electrical stimulation • Avoid use of NSAIDs
Modified rest	• Maintain fitness • Deep water running • Low impact activities (eg, cycling, stepper) • Muscle strengthening • Major muscle groups
Modification of risk factors	• Training • Footwear and insoles • Biomechanical abnormalities • Muscle flexibility and joint range • Menstrual status • Dietary intake
Facilitate return to sport	• Use of a pneumatic air brace for leg fractures • Progressive loading regimen • Monitor symptoms

nosis of stress fractures, scintigraphy was the most sensitive and plane radiographs the most specific.[35] There have been reports of a small number of cases where patients presented with symptoms of tibial stress injury evident after scintigraphic findings in the presence of a normal radiograph and which had gone on to develop after tracer uptake.[36] Computed tomography provides exquisitely fine osseous detail but should be reserved only for specific indications because it also involves ionizing radiation.[37] The advent of magnetic resonance imaging (MRI) has allowed treatment periods to be more confidently predicted.[34] MRI allows increased discrimination between medullary and cortical involvement. This permits subclassification of stress fractures into high- and low-grade injuries, permitting further prognostication. Low-grade lesions just involving the medullary cavity require relatively short periods of rest, to a maximum of 6 weeks, whereas cortical high-grade lesions require longer periods.

Risk of Complete Fracture

Adopting the principle of rest and graduated return to loading on the basis of symptomatology rather than radiographic appearance is a sound management principle.[38] To date, there have been no studies on progression of a stress response to complete fracture. The x-ray presence of the dreaded black line on an anterior tibial cortex clearly identifies a stress riser and indicates higher likelihood of complete fracture. Work on the likelihood of fracture on the basis of the radiographic appearance of lytic bone lesions has suggested the following important factors: pain, location, appearance of bone (lytic or blastic), and extent of lesion (in thirds of the bone width).[39] On the basis of this score, an anterior tibial stress response with a dreaded black line and moderate pain will have a high likelihood of fracture and should proceed to operative stabilization. Posteromedial stress fractures on the compression side of the tibia may score similarly except the black line, that is, stress riser, and so may settle with conservative management.

Time to Union, Healing, Maintenance of Aerobic Fitness, and Return-to-Play Decisions

The elapsed time for healing and return to play is certainly a concern for both recreational and professional athletes. This may manifest itself in both loss of income and potential loss of position or selection in a competitive team. The relatively common fifth metatarsal base fracture may arguably be most likely to be treated by internal fixation based purely to shorten the time taken to return to play. For acute fractures of the fifth metatarsal base, return to sport appears to be sooner after operative fixation (8 weeks) than after cast treatment (15 weeks).[40-42]

The distinction between acute fracture and an acute fracture of a subclinical stress response may not be so important.[43] However, stress fractures may be preceded by a 2-3

Table 3 A Radiological Imaging Grading System for Stress Fractures (Reprinted with permission.[34])

Grade	Radiograph	Bone Scan	MRI	Treatment
1	Normal	Mild uptake confined to one cortex	Positive STIR image	Rest for 3 wks
2	Normal	Moderate activity, larger lesion confined to unicortical area	Positive STIR and T2-weighted images	Rest for 3-6 wks
3	Discrete line (±), periosteal reaction (±)	Increased activity (50% width bone)	No definite cortical break; positive T1- and T2-weighted images	Rest for 12-16 wks
4	Fracture or periosteal reaction	More intense bicortical uptake	Fracture line; positive T1- and T2-weighted images	Rest for >16 wks

STIR, short-tau inversion recovery.

weeks history of discomfort over the lateral aspect of the foot and radiographs may show degrees of periosteal reaction and intramedullary sclerosis.[28,30] Some physicians may initially opt for conservative management, but athletes who have displaced fractures and delayed or nonunion fractures usually proceed to treatment with internal fixation.[44]

The determination of fracture healing may be difficult. Ideally an athlete's fracture should have fully united and consolidated before they return to full contact play. Assessments on clinical grounds, with symptoms on loading and manual stressing of the fracture site, are simple methods. The formation of callus and obliteration of fracture line on plain radiographs are established determinants of healing. It has been suggested that visual inspection does not accurately define union in internally fixed tibial fractures[45,46] and surgeons may turn to other physical measurements as a guide. The determination of fracture stiffness is a quantitative measurement of motion with a known applied load and is an advanced form of manual stressing. However, this method is only useful in fractures treated with external fixation using a bending load applied between the fixator pins. Fractures were considered to have healed when they had a fracture stiffness of 15 Nm/degree.[47] Although intramedullary fixation is the first fixation method considered for the internal fixation of tibial stress fractures, the development of improved external fixation techniques may increase the use of fracture stiffness to determine healing in the future.

CT scans rather than plain radiographs may be used to determine callus formation and union, but we have found that they may also be unreliable (Fig. 2).

After union has been established, athletes can progress to the resumption of activities. Return to sport should be goalled rather than based on time or radiographic findings. The lack of pain with activities of daily living and absence of bone tenderness should be identified before increased loading of exercise is considered.[48,49] During this healing period, aerobic fitness may be maintained with loading by deep water running,[50,51] pool running,[52] and cross training. Running and/or loading should be started on a treadmill, as this more compliant surface results in lower tibial bone strain than running over ground (less compliant surface).[53] Graduated return to sport occurs after an athlete has been pain free 10-14 days. Once the distance run has been tolerated pace may then be increased. A reasonable recommendation is a 10% increased activity per week of training.[54]

Figure 2 Computed tomography scan showing the stress fracture of the second metatarsal base in an international footballer, showing partial healing. The patient continued to report symptoms despite subsequent radiological union.

New Modalities

With advancing technology, many new modalities are being developed and the sporting community are ever eager to adopt these to speed healing and return to play. These modalities may be subclassified into biological and physical therapies on the basis of the technique used. The use of physical therapies to aid fracture healing has recently been summarized by Nelson et al[55] the World Anti Doping Agency code[56] must be adhered to with respect to biological bone healing techniques.

This section focuses on these modalities, particularly relating to stress fractures. The published research focuses on modalities used in combination with general fracture management principles, but multiple modalities may be used to gain the maximal benefits of biological and physical stimulation methods, for example, the use of recombinant human parathyroid hormone (PTH-134) and low intensity pulsed ultrasound have contrasting additive effects during fracture healing.[57]

Oxygen Therapy

Supplementary oxygen therapy has been encouraged to promote healing and aid recovery in a variety of clinical situations. The administration of hyperbaric oxygen therapy (HBOT) consists of administering 100% oxygen at pressures greater than one atmosphere absolute in a pressure vessel.

In vitro studies have shown that osteoblasts exposed to HBOT have enhanced differentiation toward the osteogenic phenotype, promoting bone formation at a cellular level.[58] Clinical studies have shown a less significant effect. A recent Cochrane review into the benefit of hyperbaric oxygen for promoting fracture healing and treating fracture nonunion failed to locate any clinical evidence that supported or refuted the effectiveness of HBOT for the management of delayed union or established nonunion of bony fractures.[59] Thus, the role of HBOT in stress fracture healing is questionable at this stage.

Bisphosphonates

Bisphosphonates suppress bone reabsorption by osteoclasts and thus prevent bone loss during the initial remodeling in response to high bone strains. This may allow increased osteoblastic healing and speed recovery. Intravenous pamidronate has been used successfully on 5 collegiate athletes who had symptomatic tibial stress fractures. Four of the 5 athletes were able to continue training without symptoms within 72 hours, suggesting a therapeutic effect.[60] Given the costs and potential adverse effects of bisphosphonates, it may currently be considered prudent to restrict the use of bisphosphonates for the treatment of stress fractures in athletes.[61] Any prophylactic effects of bisphosphonates have also yet to be proven.[62] When intravenous pamidronate was prophylactically administered to military recruits no improvement in the incidence of stress fractures was seen.[63]

Growth Factors

The use of growth factors and preparations rich in growth factors (PRGF) has become increasingly popular.[64,65] Platelet rich plasma secretes a complex mixture of biological mediators essential for natural repair, including transforming growth factor β, platelet-derived growth factor, vascular endothelial growth factors, epithelial growth factor, hepatocyte growth factor, and insulin-like growth factor. After the platelets concentrate is activated, the growth factors are progressively released into the surrounding tissue contributing to the accelerated healing, tissue repair, and vascularization.[65] The localized injection of concentrations has shown increased healing in muscle, tendon, and ligamentous injuries,[65-67] however, there has as yet been little reported in the healing of stress fractures. Autologous preparations rich in growth factors can enhance the healing of hypertrophic nonunions, where PRGF was added at the time of internal fixation for unstable and injection for stable nonunions.[68] There has been little reported on the effect of PRGF on the healing of stress fractures but PRGF are likely to speed union in high-risk fractures when identified before complete fracture has occurred and at the time of operative fixation of fractures.

Bone Morphogenic Protein

Bone morphogenic proteins (BMPs) are a family of bioactive factors responsible for the bone inductive activity of bone matrix, and members of this family of proteins have been developed as osteoinductive compounds for a variety of applications. They primarily function by causing the differentiation of mesenchymal cells into bone forming and cartilage forming cells that may result in endochondral and direct ossification. They are believed to be important mediators of fracture repair. Most of these factors are placed intraoperatively, at the fracture site to promote healing. Recent work has been performed on the percutaneous injection of osteoinductive factor.[69] In a rat femoral fracture model, a single local percutaneous injection of recombinant human bone morphogenetic protein-2 has been demonstrated to accelerate fracture healing.[70] The development of percutaneous injections may offer a minimally invasive method of administration to gain the therapeutic effects of BMP.

Recombinant Parathyroid Hormone

Parathyroid hormone (PTH) is an 84 amino acid polypeptide and an essential component of mineral homeostasis. It increases serum calcium levels by enhancing gastrointestinal calcium absorption, increased renal calcium and phosphate absorption, and liberates calcium from the skeleton in response to systemic needs. Although primarily promoting osteoclast activity with regular administration, intermittent exposure to PTH stimulates osteoblasts and results in increased bone formation. Animal studies of the daily administration of PTH[1-32,35-37] have shown increased bone mineral content, density, and strength as well as a sustained anabolic effect throughout the remodeling phase of fracture healing.[71] In the United States, PTH has been approved as an anabolic treatment for osteoporosis. Current research clearly demonstrates that systemic PTH treatment can enhance both endochondral and intramembranous bone repair. PTH affects both chondrogenic and osteogenic events during bone repair.[72] Again there has been little reported on stress fractures, but this will be an interesting area for future developments.

Ultrasound

The application of ultrasound therapy is increasingly used to improve the healing of fractures. The emitted acoustic energy is of high frequency, above the limit of human hearing. The sound waves produced are thought to place the bone and surrounding tissue under microstresses and strains, promoting healing. Although this treatment is certainly effective, the mode of action is actually uncertain.[73] It has been suggested that ultrasound stimulates the mechanical properties of the healing fracture callus by stimulating earlier synthesis of extracellular matrix proteins in cartilage, possibly by altering chondrocyte maturation and endochndral bone formation.[74]

Most reported studies utilize the Sonic Accelerated Fracture Healing System (Exogen, Piscataway, NJ). Treatment sessions consist of 20- minute sessions daily, with an ultrasound composed of burst width 200 μsecond containing 1.5 MHz sine waves, with a repetition rate of 1 kHz and a spatial average temporal intensity of 30 mW/cm^2. The duration of treatment varies between study groups, and patient compliance is determined by alarms attached to the system.

The daily application of noninvasive pulsed ultrasound has been shown to increase acute tibial[75] and distal radial fracture healing.[76] The public has been aware of the use of ultrasound for stress fracture healing, as its use was associated with an improvement in symptoms in an Olympic level gymnast. The athlete was able to progress to full work out activities at 4.5 weeks after a mid tibial stress injury, and at week 6 participated in the United States gold medal winning team.[77]

When a series of 8 patients with tibial stress fractures, 7 of whom were posteromedial, received 20 minutes of low-intensity pulsed ultrasound (frequency 1.5 MHz, radiating area 3.88 cm^2, pulse width 200 μs, and temporal average power

117 mW) 5 times a week for 4 weeks, patients reported reduced pain and improved functional performance without the use of a brace. The 1 patient who did not improve had an anterior stress fracture and went on to intramedullary nailing.

A meta-analysis of the effect of low intensity pulsed ultrasound on the healing of all types of fractures found conflicting results, with evidence that they were moderate to low in quality.[78] For nonoperatively treated stress fractures, moderate quality evidence suggests that there is no effect of low intensity pulsed ultrasonography on return to function. Naval personnel with tibial stress fractures who received ultrasound returned to active duty after a mean of 55.8 days as compared with 56.2 days for those who received sham therapy.[79]

Interestingly, animal studies have confirmed that low intensity pulsed ultrasound and NSAIDs have opposing effects during fracture repair, and when used in combination the beneficial effects of ultrasound are not impaired by the detrimental effects of NSAIDs.[80]

Further research is required before firm conclusions can be made, regarding the efficacy of ultrasound for stress fractures.

Magnetic Field Application

Electrical stimulation can be applied to a fracture either by direct current stimulation requiring operative placement of an electrode against the fracture site, capacity coupled electrical field (CCEF) devices (Fig. 3), or pulsed electromagnetic field (PEMF) stimulation of bone. Capacity coupled devices have been shown to produce more DNA production from the bone cells than does the PEMF. PEMF relies on release of intracellular stores of calcium, whereas CCEF uses the calcium ions within the extracellular fluid.

Electric fields are well known to promote bone healing in vitro[81] on the basis of cellular stimulation. Optimal capacitively coupled signal (60 kHz, 20 mV/cm at a 50% duty cycle for 24 hours) was shown to specifically, selectively, and simultaneously upregulate the expression of several osteoinductive BMPs, and other BMPs and antagonists were only moderately affected.[82]

Figure 3 Capacitively coupled devices are easily transportable and applied close to the stress fracture.

In a clinical setting, the use of electricity and electricity-generated magnetic fields has been shown to aid the healing of troublesome fractures.[83] A randomized controlled trial was unable to detect an effective of capacitively coupled electric field stimulation on the healing of 44 posteromedial tibial stress fractures, but greater device use and less weight bearing loading enhanced bone healing. The CCEF devices used are small portable stimulators that apply a sinusoidal wave of 3-6 V at 60 kHz and 5-10 mA through 2 adhesive water-based electrodes for 15 hours per day to maximize usage. Twenty-five stress fractures were treated with CCEF stimulation for a mean time of 15 hours per day for 7.4 weeks.[84] In this study, 88% of fractures healed, 8% improved and 4% did not heal. Although this study showed improved, healing there was no control group or further analysis by fracture type.[85]

Early studies of the treatment of fractures with pulsed electromagnetic fields on the healing of fifth metatarsal fractures suggested improved healing times of 3 months compared with a mean healing time of 4 months for all fractures of the fifth MT base.[86]

Clearly the scientific evidence of the use of electromagnetic fields for the healing of stress fractures is still in an early stage and more work will have to be undertaken before firm conclusions can be made.

Conclusions

Stress injuries and stress fractures are clearly troublesome for the sporting population, particularly the professional athlete. Injuries should be fully imaged to assess and determine the risk and grade of the stress response, as this can direct further management. The relative infrequency of stress fractures means that the determination of the efficacy of new treatment modalities is difficult. Athletes are willing to utilize any method to promote healing and earlier return to play. Although this review reports little efficacy for the use of new modalities, the physical methods used are unlikely to delay healing and may be preferable to biological techniques in which the full implications of their use may not be fully understood. Many of these new techniques will be difficult to obtain and are expensive. Thus, during the healing phase, the athlete should focus on conventional methods of relative rest, analgesia, and rehabilitation. Although surgical stabilization involves iatrogenic trauma to the area, the pain related to surgery may well force the athlete to rest, thus promoting healing and recovery.

References

1. Wolff J: Das Gesetz der Transformation der knochen. Berlin, Verlag August Hirschwald, 1892
2. Knapp TP, Garrett WE Jr: Stress fractures: General concepts. Clin Sports Med 16:339-356, 1997
3. Kaeding CC, Spindler KP, Amendola A: Management of troublesome stress fractures. Instr Course Lect 53:455-469, 2004

4. Brukner P, Bennell K: How should you treat a stress fracture? in MacAuley D, Best T (eds): Evidence-based Sports Medicine (ed 1). London, UK, Wiley/BMJ books, 2002, pp 491-517
5. Mori S, Burr DB: Increasing intracortical remodelling following fatigue damage. Bone 14:103-109, 1993
6. Brukner P, Bradshaw C, Bennell K: Managing common stress fractures: Let risk level guide treatment. Physician Sports Med 26:39-47, 1998
7. Swenson EJ Jr, DeHaven KE, Sebastinelli WJ, et al: The effect of a pneumatic leg brace on return to play in athletes with tibial stress fractures. Am J Sports Med 25:322-328, 1997
8. Whitelaw G, Wetzler M, Levy A, et al: A pneumatic leg brace for the treatment of tibial stress fractures. Clin Orthop 270:301-305, 1991
9. Slayter M: Lower limb training injuries in an army recruit population. University of Newcastle, Newcastle, 1995
10. Gillespie WJ, Grant I: Interventions for preventing and treating stress fractures and stress reactions of bone of the lower limbs in young adults. Cochrane Database Syst Rev 2:CD000450, 2005
11. Allen CS, Flynn TW, Kardouni JR, et al: The use of pneumatic leg brace in soldiers with tibial stress fractures-a randomised controlled trial. Mil Med 169:880-884, 2004
12. Batt ME, Kemp S, Kerslake R: Delayed union stress fractures of the anterior tibia: Conservative management. Br J Sports Med 35:74-77, 2001
13. Kawaguchi H, Pilbeam CC, Harrsion JR, et al: The role of prostaglandins in the regulation of bone metabolism. Clin Orthop 13:36-46, 1995
14. Wheeler P, Batt ME: Do non-steroidal anti-inflammatory drugs adversely affect stress fracture healing? A short review. Br J Sports Med 39:65-69, 2005
15. Mehallo CJ, Drezner JA, Bytomski JR: Practical management: Non steroidal anti-inflammatory drug (NSAID) use in athletic injuries. Clin J Sport Med 16:170-174, 2006
16. Wegener C, Burns J, Penkala S: The effect of neutral-cushioned running shoes on plantar pressure loading and comfort in athletes with cavus feet. Am J Sports Med 36:2139-2146, 2008
17. Hume P, Hopkins W, Rome K, et al: Effectiveness of foot orthoses for treatment and prevention of lower limb injuries: A review. Sports Med 38:759-779, 2008
18. Rome K, Handoll HH, Ashford R: Interventions for preventing and treating stress fractures and stress reactions of bone of the lower limbs in young adults. Cochrane Database Syst Rev 2:CD000450, 2005
19. Korpelainen R, Orava S, Karpakka J, et al: Risk factors for recurrent stress fracture in athletes. Am J Sports Med 29:304-310, 2001
20. Simkin A, Leichter I, Giladi M, et al: Combined effect of foot arch structure and an orthotic device on stress fractures. Foot Ankle Int 10:25-29, 1989
21. Matheson GO, Clement DB, Mckenzie DC, et al: Stress fractures in athletes: A study of 320 cases. Am J Sports Med 15:46-58, 1987
22. Sommer HM, Vallentyne SW: Effect foot posture on the incidence of medial tibial stress syndrome. Med Sci Sports Exerc 27:800-804, 1995
23. Maitra RS, Johnson DL: Stress fractures. Clinical history and physical examination. Clin Sports Med 16:259-274, 1997
24. Manioli A, Graham B: The subtle cavus foot: “the under pronator”: A review. Foot Ankle Int 26:256-263, 2005
25. Khan K, Brown J, Way S, et al: Overuse injuries in classical ballet. Sports Med 19:341-357, 1995
26. Drez D Jr, Young JC, Johnston RD, et al: Metatarsal stress fractures. Am J Sports Med 8:123-125, 1980
27. Boden BP, Osbahr DC, Jimenez C: Low risk stress fractures. Am J Sports Med 29:100-111, 2001
28. Boden BP, Osbahr DC: High risk stress fractures: Evaluation and treatment. J Am Acad Orthop Surg 8:344-353, 2000
29. Orava S, Hulkko A: Delayed unions and non-unions of stress fractures in athletes. Am J Sports Med 16:378-382, 1988
30. Torg JS, Balduini FC, Zelko RR, et al: Fractures of the base of the fifth metatarsal distal to the tuberosity. Classification and guidelines for non surgical and surgical management. J Bone Joint Surg Am 66:209-214, 1984
31. Brukner PD, Bennell KL: Stress fractures in runners. J Back Musculoskelet Rehab 5:341-351, 1995
32. Micheli LJ, Sohn RS, Soloman R: Stress fractures of the second metatarsal involving Lisfranc’s joint in a ballet dancer: A new overuse injury of the foot. J Bone Joint Surg Am 67:1372-1375, 1985
33. Kaeding CC, Yu JR, Wright R, et al: Management and return to play of stress fractures. Clin J Sport Med 15:442-447, 2005
34. 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 16:291-306, 1997
35. Giladi M, Nili E, Ziv Y, et al: Comparison between radiography, bone scan and ultrasound in the diagnosis of stress fractures. Mil Med 149: 459-461, 1984
36. Milgrom C, Chisin R, Giladi M, et al: Negative bone scans in impending tibial stress fractures. Am J Sports Med 12:488-491, 1984
37. Moran DA, Evans RK, Hadad E: Imaging of lower extremity stress fracture injuries. Sports Med 38:345-356, 2008
38. Raasch WG, Hergan DJ: Treatment of stress fractures: The fundamentals. Clin Sports Med 25:29-36, 2006
39. Mirels H: Metastatic disease in long bones. A proposed scoring system for diagnosing impending pathologic fractures. Clin Orthop Relat Res 249:256-264, 1989
40. Mologne TS, Lundeen JM, Clapper MF, et al: Early screw fixation versus casting in the treatment of acute Jones fractures. Am J Sports Med 33:970-975, 2005
41. DeLee JC, Evans JP, Julian J: Stress fracture of the fifth metatarsal. Am J Sports Med 11:110-114, 1983
42. Zelko RR, Torg JS, Rachun A: Proximal diaphyseal fractures of the fifth metatarsal-treatment of the fractures and their complications in athletes. Am J Sports Med 7:95-101, 1979
43. Landorf KB: Clarifying proximal diaphyseal fifth metatarsal fractures. The acute fracture versus the stress fracture. J Am Podiatr Med Assoc 89:398-404, 1999
44. Chuckpaiwong B, Queen RM, Easley ME, et al: Distinguishing Jones and proximal diaphyseal fractures of the fifth metatarsal. Clin Orthop Relat Res 466:1966-1970, 2008
45. Davis BJ, Roberts PJ, Moorcroft CI, et al: Reliability of radiographs in defining union of internally fixed fractures. Injury 35:557-561, 2004
46. McClelland D, Thomas PB, Bancroft G, et al: Fracture healing assessment comparing stiffness measurements using radiographs. Clin Orthp Relat Res 457:214-219, 2007
47. Richardson JB, Cunningham JL, Goodship AE, et al: Measuring stiffness can define the healing of tibial fractures. J Bone Joint Surg Br 76:389-394, 1994
48. Perron AD, Brady WJ, Keats TA: Principles of stress fracture management. The why’s and how’s of an increasingly common injury. Postgrad Med 110:115-118, 123-124, 2001
49. Perron AD, Brady WJ, Keats TA: Management of common stress fractures. When to apply conservative therapy, when to take an aggressive approach. Postgrad Med 111:95-96, 99-100, 105-106, 2002
50. Frangolias DD, Taunton JE, Rhodes EC, et al: Maintenance of aerobic capacity during recovery from right foot Jones fracture: A case report. Clin J Sports Med 7:57-58, 1997
51. Burns AS, Lauder TD: Deep water running: An effective non weight bearing exercise for the maintenance of land based running performance. Mil Med 166:253-258, 2001
52. Gehring MM, Keller BA, Brehm BA: Water running with and without a flotation vest in competition and recreation runners. Med Sci Sports Exerc 29:1374-1378, 1997
53. Milgrom C, Finestone A, Segev S, et al: Are overground or treadmill runners more likely to sustain tibial stress fracture? Br J Sports Med 37:160-163, 2003
54. Diehl JJ, Best TM, Kaeding CC: Classification and return to play considerations for stress fractures. Clin Sports Med 25:17-28, 2006
55. Nelson FR, Brighton CT, Ryaby J, et al: Use of physical forces in bone healing. J Am Acad Orthop Surg 11:344-354, 2003
56. The World Anti Doping Agency: The World Anti-Doping Code: The 2009 Prohibited List International Standard. WADA, Montreal, 2008
57. Warden SJ, Komatsu DE, Rydberg J, et al: Recombinant human parathyroid hormone (PTH-134) and low intensity pulsed ultrasound have

contrasting additive effects during fracture healing. Bone 44:485-494, 2009
58. Wu D, Malda J, Crawford R, et al. Effects of hyperbaric oxygen on proliferation and differentiation of osteoblasts from human alveolar bone. Connect Tissue Res 48:206-213, 2007
59. Bennet MH, Stanford R, Turner R: Hyperbaric oxygen therapy for promoting fracture healing and treating fracture non union. Cochrane Database Syst Rev 1:CD004712, 2005
60. Stewart GW, Brunet ME, Manning MR, et al: Treatment of stress fractures in athletes with intravenous pamidronate. Clin J Sports Med 15: 92-94, 2005
61. Shima Y, Engebretsen L, Iwasa J, et al: Use of bisphosphonates for the treatment of stress fractures in athletes. Knee Surg Sports Traumatol Arthrosc 17:542-550, 2009
62. Ekenman I: Do not use bisphosphonates without scientific evidence, neither in the treatment nor prophylactic, in the treatment of stress fractures. Knee Surg Sports Traumatol Arthrosc 17:433-434, 2009
63. Milgrom C, Finestone A, Novack V, et al: The effect of prophylactic treatment with risedronate on stress fracture incidence among infantry recruits. Bone 35:418-424, 2005
64. Molloy T, Wang Y, Murrell GAC: The roles of growth factors in tendon and ligament healing. Sports Med 33:381-394, 2003
65. Anitua E, Sanchez M, Nurden AT, et al: New insights into and novel applications for platelet rich fibrin therapies. Trends Biotechnol 24: 227-234, 2006
66. Hammond JW, Hinton RY, Curl LA, et al: Use of autologous platelet-rich plasma to treat muscle strain injuries. Am J Sports Med (in press)
67. Sanchez M, Anitua E, Azofra A, et al: Comparison of surgically repaired Achilles tendon tears using platelet rich fibrin matrices. Am J Sports Med 35:245-251, 2007
68. Sanchez M, Anitua E, Cugat R, et al: Non unions treated with autologous preparation rich in growth factors. J Orthop Trauma 23:52-59, 2009
69. Bostrom MP: Expression of bone morphogenic proteins in fracture healing. Clin Orthop 355:S116-S123, 1998
70. Einhorn TA, Majeska RJ, Mohaideen A, et al: A single percutaneous injection of recombinant human bone morphogenic protein-2 accelerates fracture repair. J Bone Joint Surg Am 85:1425-1435, 2003
71. Alkhiary YM, Gerstenfeld LC, Krall E, et al: Enhancement of experimental fracture healing by systemic administration of recombinant human parathyroid hormone PTH(1-34). J Bone Joint Surg Am 87:731-741, 2005
72. Barnes GL, Kakar S, Vora S, et al: Stimulation of fracture healing with systemic intermittent parathyroid hormone treatment. J Bone Joint Surg Am 90S:120-127, 2008
73. Bolin D, Kemper A, Brolinson PG: Current concepts in the evaluation and management of stress fractures. Curr Sports Med Rep 4:295-300, 2005
74. Yang KH, Parvizi J, Wang SJ, et al: Exposure to low intensity ultrasound increase aggrecan gene expression in a rat femur fracture model. J Orthop Res 14:802-809, 1996
75. Heckman JD, Ryaby JP, McCabe J, et al: Acceleration of tibial fracture healing by non-invasive low intensity pulsed ultrasound. J Bone Joint Surg 76:26-34, 1994
76. Kristiansen TK, Ryaby JP, McCabe J, et al: Accelerated healing of distal radius fractures with the use of specific low intensity ultrasound. A multicenter, prospective, randomized double blind, placebo controlled study. J Bone Joint Surg 79:961-973, 1997
77. Jensen JE: Stress fracture in a world class athlete: A case study. Med Sci Sports Exerc 30:783-787, 1998
78. Busse JW, Kaur J, Mollon B, et al: Low intensity pulsed ultrasonography for fractures: Systemic review of randomised controlled trials. BMJ 338:b351, 2009
79. Rue JP, Armstring DW III, Frassica FJ, et al: The effect of pulsed ultrasound in the treatment of tibial stress fractures. Orthopedics 27:1192-1195, 2004
80. Li J, Waugh LJ, Hui SL, et al: Low intensity pulsed ultrasound and non steroidal anti-inflammatory drugs have opposing effects during stress fracture repair. J Orthop Res 25:1559-1567, 2007
81. Hartig M, Joos U, Wiesmann HP: Capacity coupled electric fields accelerate proliferation of osteoblast like primary cells and increase bone extracellular matrix formation in vitro. Eur Biophys J 29:499-506, 2000
82. Wang Z, Clark C, Brighton CT: Up-regulation of bone morphogenetic proteins in cultured murine bone cells with use of specific electric fields. J Bone Joint Surg Am 88:1053-1065, 2006
83. Simonis RB, Parnell EJ, Ray PS, et al: Electrical treatment of tibial non-union: A prospective randomised double blind trial. Injury 34: 357-362, 2003
84. Beck BR, Matheson GO, Bergman G, et al: Do capacity coupled electric fields accelerate tibial stress fracture healing. Am J Sports Med 36:545-553, 2008
85. Benazzo F, Mosconi M, Beccarisi G, et al: Use of capacity coupled electric fields in stress fractures in athletes. Clin Orthop Relat Res 310: 145-149, 1995
86. Holmes GB, Jr: Treatment of delayed unions and non unions of the proximal fifth metatarsal with pulsed electromagnetic fields. Foot Ankle Int 15:552-556, 1994