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Masterclass

Preventing and managing stress fractures in athletes

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Abstract

Stress fractures are common overuse injuries of bone resulting from the repeated application of submaximal load. Factors that reduce bone strength or increase the load applied to bone can place an athlete at risk of developing a stress fracture. These factors include low bone density, menstrual disturbances, inadequate dietary intake and eating disorders, training errors, inadequate muscle function and biomechanical features. Identification of the at-risk athlete can allow prevention strategies to be implemented. Diagnosis of a stress fracture is generally made clinically but investigations such as bone scan, CT or MRI can be performed to confirm the diagnosis, grade the stage of the bone response and localize the site. Most stress fractures will heal with modified rest and permit return to sport around 8 weeks. However, there is a group of stress fractures that requires additional treatment and special consideration. Treatment of the typical stress fracture requires pain management, modification (or cessation) of the aggravating activity, muscle strengthening and maintenance of aerobic fitness, identification and subsequent modification of risk factors and gradual resumption of bone loading activities. The use of braces has been shown to reduce the time to return to full activity in some lower limb stress fractures. Similarly the use of electrical stimulation and ultrasound may be helpful. Recovery should be monitored clinically.

Keywords: Stress fracture; Prevention; Risk factors; Treatment

1. Introduction

Physically active individuals such as athletes (Bennell et al., 1996b; Johnson, Weiss, & Wheeler, 1994) and military recruits (Kaufman, Brodine, & Shaffer, 2000; Pope et al., 1999) are prone to stress fractures. These overuse injuries frequently occur in lower limb bones particularly the tibia (Matheson et al., 1987b) and result from bone's inability to withstand repetitive load.

Physical activity loads bone through ground reaction forces and muscle contraction. Under normal circumstances, bone microdamage that develops with loading stimulates a reparative remodelling process. In this process, bone resorption occurs first followed by bone formation. Accelerated remodelling due to loading may also make the bone more vulnerable to damage because there is a lag time between resorption and formation. Thus, if loading continues and microdamage accumulates and cannot be repaired by remodeling then symptoms of excessive bone strain may result (Burr et al., 1990; Schaffler, Radin, & Burr, 1989, 1990). Because bone strain occurs along a continuum, stress fractures represent one end of this continuum (Fig. 1). Bone's ability to resist damage depends on a number of factors including its structural and material properties and the activity of muscles in attenuating loads. Thus factors which increase the load on bone or impair bone strength may place an athlete at risk of developing a stress fracture (Fig. 2).

2. Prevention

In order to prevent a stress fracture, identifying risk factors then implementing prevention strategies to modify the risk factors or reduce the amount of load applied to bone is necessary (Table 1). There are a limited number of randomized controlled clinical trials (RCTs) evaluating the effectiveness of risk factor modification in reducing stress fracture occurrence. To date all trials have been performed

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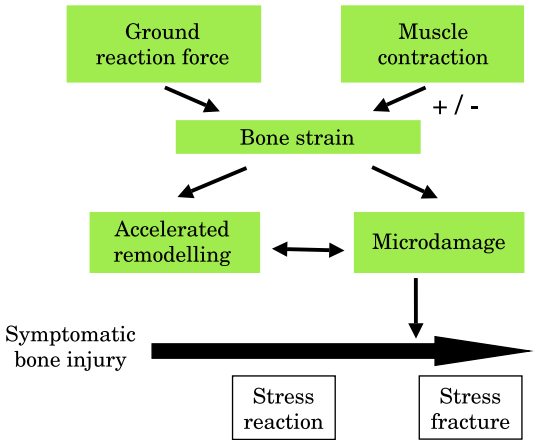

Fig. 1. Pathogenesis of a stress fracture. Loading via ground reaction force and muscle contraction results in bone strain. This leads to both accelerated remodelling and to microdamage. Remodelling also makes the bone more vulnerable and so increased microdamage can occur at bone sites undergoing remodelling. If the microdamage cannot be repaired by remodelling, then a symptomatic bone injury can occur.

in military recruits meaning that results cannot necessarily be transferred to the athletic population. The interventions tested include shock absorbing insoles ($n=4$; (Andrish, Bergfeld, & Walheim, 1974; Milgrom et al., 1985b; Schwellnus, Jordaan & Noakes, 1990; Smith, Walter & Bailey, 1985)), orthotic inserts ($n=2$ (Gardner et al., 1988; Mundermann, Stefanyshyn & Nigg, 2001)), basketball shoes ($n=1$; (Milgrom et al., 1992)), training modifications ($n=2$; (Giladi et al., 1985a; Scully & Besterman, 1982)), calcium supplements ($n=1$, (Schwellnus & Jordaan, 1992)) and prophylactic risedronate ($n=1$; (Milgrom et al., 2004)). Instead, most of the studies on prevention are cohort studies where the relationship of a risk factor to stress fracture is evaluated in either a prospective or retrospective manner.

2.1. Pharmacological agents

It was hypothesized that short-term suppression of bone turnover using bisphosphonates (drugs commonly used to treat osteoporosis) might prevent the initial loss of bone during the remodelling response to high bone strains and potentially prevent stress fractures. This was recently evaluated in a randomized, double-blind placebo controlled study using risedronate or placebo given as 30 mg daily for 10 weeks then once a week for the next 12 weeks in 324 military recruits (Milgrom et al., 2004). However, results showed no statistically significant difference in the total, tibial, femoral or metatarsal stress fracture incidence between groups. Thus, risedronate does not appear to be a viable option for lowering stress fracture risk in active healthy populations.

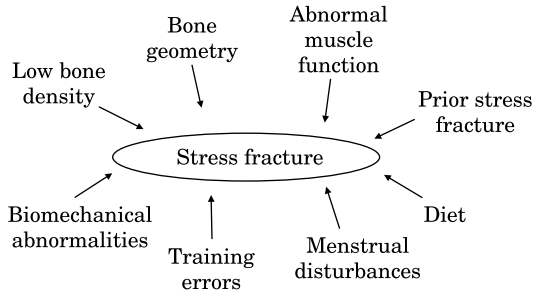

Fig. 2. Factors that can influence the risk of stress fracture.

Table 1
Risk factor assessment either as primary prevention or as part of secondary prevention in someone presenting with a stress fracture[a]

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 e.g. protein, fibre
	Presence of eating disorder

[a] Taken from Bennell K and Brukner P, 2002. How would you treat a stress fracture? In D MacAuley and T Best (Eds) Evidence-based sports Medicine. Blackwell Publishing.

2.2. Muscle strengthening

Skeletal muscle plays an important role in stress fracture development. At some regions, bone load is increased by muscular force while at other sites load is reduced as muscles absorb energy (Scott & Winter, 1990). In endurance sports, it is possible that even low levels of muscular fatigue can affect the total impact load to bone, particularly in the lower extremity. Following fatiguing exercise, bone strain, particularly strain rate, has been shown to increase (Fyhrie et al., 1998; Yoshikawa et al., 1994). Some studies have shown that reduced muscle strength (Hoffman et al., 1999) and smaller muscle size (Armstrong et al., in press; Bennell et al., 1996a; Milgrom,

1989) predispose to stress fractures in athletes and military recruits. Thus there is the potential that muscle strengthening programs particularly of the calf muscles may help reduce the risk of stress fracture. Interventions to assist in muscle recovery between training sessions may also be implemented such as massage, icing etc.

2.3. Training alterations

While training errors have been anecdotally linked to stress fracture development, there is little research to identify the contribution of each training component (type, volume, intensity, frequency and rate of change) especially in athletes. High impact activity places the greatest load on bone and two RCTs in the military (Giladi et al., 1985a; Scully & Besterman, 1982) suggest that reducing high impact activities decreases stress fracture incidence. In athletes, Brunet et al. (1990) surveyed 1505 runners and found that increasing mileage correlated with an increase in stress fractures in women but not men. In a study of ballet dancers, those who trained more than five hours daily had an estimated risk for stress fracture that was 16 times greater than those who trained less than five hours per day (Kadel, Teitz & Kronmal, 1992). Coaches should ensure that training regimens for athletes are individualised. What may be appropriate for most members of a team may be excessive for some. Athletes should be encouraged to keep an accurate training log book to help devise appropriate training levels and changes to training. It is important to allow adequate recovery time after hard sessions or hard weeks of training. This can be accommodated by developing micro- and macrocycles. Alternating hard and easy training sessions is a microcycle adjustment but graduating the volume of work or alternating harder and easier sessions can also be done weekly or monthly. During periods of increases in training, it is worth introducing these on a step wise basis. For example, introduce the increase then remain at this level for a few weeks until bone becomes adapted to the load.

2.4. Footwear and insoles

A Cochrane review identified six RCTs or quasi-experimental trials that evaluated the effect of insoles or other footwear modifications on prevention of stress fractures in the military (Andrish, Bergfeld & Walheim, 1974; Gardner et al., 1988; Milgrom et al., 1992; Milgrom et al., 1985b; Schwellnus, Jordaan & Noakes, 1990; Smith, Walter & Bailey, 1985). The authors of the review concluded that 'the use of insoles inside boots in military recruits during their initial training appears to reduce the number of stress fractures and/or stress reactions of bone by over 50%' (Gillespie & Grant, 2000). Another study published since then also found that various types of orthotic inserts were associated with less foot stress fractures (Mundermann, Stefanyshyn & Nigg, 2001). Whether the results can be generalised to the sporting population who often wear different footwear and perform different training is not clear. This is emphasized by a laboratory experiment study that found semirigid orthoses lowered tibial bone strain when worn in boots while walking but increased strains during running (Ekenman et al., 2002). Another important contributing factor to stress fracture development may be inadequate training shoes. These shoes may be inappropriate for the particular foot type of the individual, may have general inadequate support/shock absorption or may be worn out. However, there is no evidence to show differences in stress fracture risk with different sports shoes.

2.5. Alterations in biomechanical features

Intrinsic biomechanical abnormalities are also thought to be contributing factors to the development of overuse injuries in general and stress fractures in particular. The structure of the foot will partly determine how much force is absorbed by the bones in the foot and how much force is transferred to proximal bones such as the tibia during ground contact. The high arched (pes cavus) foot is more rigid and less able to absorb shock resulting in more force passing to the tibia and femur. The low arched (pes planus) foot is more flexible allowing stress to be absorbed by the musculoskeletal structures of the foot. It is also often associated with prolonged pronation or hyperpronation which can induce a great amount of torsion on the tibia and may exacerbate muscle fatigue as the muscles have to work harder to control the excessive motion, especially at toe-off. Theoretically, either foot type could predispose to a stress fracture. Results have been conflicting and may depend upon the site of the stress fracture (Brosh & Arcan, 1994; Giladi et al., 1985b; Korpelainen et al., 2001; Montgomery et al., 1989; Simkin et al., 1989).

There is evidence from cohort studies to show that a leg length discrepancy increases the likelihood of stress fractures in both military (Friberg, 1982) and athletic (Bennell et al., 1996a; Brunet et al., 1990; Korpelainen et al., 2001) populations but the injury does not seem to occur on either the shorter or longer leg preferentially. Other alignment features include the presence of genu varum, valgum or recurvatum, Q angle, and tibial torsion. Of these, only an increased Q angle has been found in association with stress fractures (Cowan et al., 1996) although this is not a universal finding (Montgomery et al., 1989; Winfield et al., 1997). A thorough biomechanical assessment is an essential part of prevention of stress fractures. Until the contribution of biomechanical abnormalities to stress fracture risk is clarified through scientific research, correction of such abnormalities should be attempted, if possible.

2.6. Muscle flexibility and joint range of motion

Numerous variables have been assessed in relation to stress fractures including range of rearfoot

inversion/eversion, ankle dorsiflexion/plantarflexion, knee flexion/extension and hip rotation/extension together with length of calf, hamstring, quadriceps, hip adductors and hip flexor muscles (Bennell et al., 1996a; Ekenman et al., 1996; Giladi et al., 1987; Hughes, 1985; Milgrom et al., 1994; Montgomery et al., 1989; Winfield et al., 1997). Of these, only increased range of hip external rotation (Giladi et al., 1991, 1987; Milgrom et al., 1994) and decreased range of ankle dorsiflexion (Hughes, 1985) have been associated with stress fracture development and even these findings have been inconsistent. Until better evidence is available to the contrary, it is worth prescribing stretches if muscle flexibility and joint range are found to be restricted in the athlete who presents with a stress fracture.

2.7. Menstrual alterations and bone density

The prevalence of menstrual disturbances such as amenorrhea (<3 menstrual cycles in a year) is more common in sportswomen than in the general population, particularly those competing at elite levels and involved in sports where leanness is emphasized. Cross-sectional and cohort studies show that current and past menstrual disturbances increase the risk of stress fracture (Barrow & Saha, 1988; Bennell et al., 1995, 1996a; Carbon et al., 1990; Frusztajer et al., 1990; Grimston et al., 1991; Kadel et al., 1992; Lindberg et al., 1984; Lloyd et al., 1986; Marcus et al., 1985; Myburgh et al., 1990; Nelson et al., 1987; Tomten, 1996; Warren et al., 1986; Winfield et al., 1997) and lead to premature bone loss particularly at trabecular sites (Gremion et al., 2001; Hetland et al., 1993; Jonnavithula et al., 1993; Myburgh et al., 1993; Robinson et al., 1995). Lower bone density in women may be associated with a greater risk of stress fractures although results from studies are mixed (Bennell et al., 1995, 1996a; Carbon et al., 1990; Cline, Jansen & Melby, 1998; Frusztajer et al., 1990; Girrbach et al., 2001; Grimston et al., 1991; Lauder et al., 2000; Myburgh et al., 1990). It may be that low bone density plays a greater role in predisposing to stress fractures at cancellous bone sites rather than cortical sites (Marx et al., 2001).

Low energy availability due to dietary restriction and/or excessive exercise is now thought to be the primary cause of bone loss in athletic amenorrhea rather than a lack of estrogen (Kaufman et al., 2002; Zanker & Swaine, 1998). Low energy availability uncouples bone turnover and suppresses bone formation (Ihle & Loucks, 2004). This implies that treatment of the amenorrheic athlete should focus on reducing the energy imbalance either via reducing the amount and intensity of her activity and/or increasing her daily energy intake.

The role of bone density measurement by dual energy xray absorptiometry in these athletes is still unclear. Bone density measurements compare the patient's bone density to the "average", but it is known that bone density is increased in those involved in weight bearing exercise (Wallace & Cumming, 2000) and there are no normative databases for athletes. Bone density measurement may be useful in the amenorrheic athlete both to provide a baseline measurement prior to treatment and as a potential additional factor in convincing the athlete to commence treatment.

2.8. Dietary intake

Macro- and micro-nutrients that are important for skeletal health are often inadequate in sportspeople particularly women (Ronsen, Sundgot-Borgen & Maehlum, 1999; Ziegler, Nelson & Jonnalagadda, 1999). Furthermore, athletes report a greater frequency of disordered eating patterns than the general population (Picard, 1999). As discussed, low caloric intake has been hypothesized as one of the mechanisms for menstrual disturbances in sportswomen (Zanker & Swaine, 1998). Disordered eating, amenorrhea and osteopenia often occur simultaneously in athletic females, a syndrome that has been referred to as the 'female athlete triad' (Otis et al., 1997). Therapists need to be aware of athletes who present with one of these features as the others may also be present.

There is currently little evidence to support low calcium intake as a risk factor for stress fractures in athletic (Bennell et al., 1996a; Carbon et al., 1990; Frusztajer et al., 1990; Grimston et al., 1991; Kadel et al., 1992; Warren et al., 2002, 1991) or military (Cline et al., 1998) populations. In the only controlled trial, calcium supplementation of 500 mg daily had no significant effect on stress fracture incidence in male military recruits (Schwellnus & Jordaan, 1992). Conversely, abnormal and restrictive eating behaviours and low energy intake do seem to increase the likelihood of stress fracture in women (Armstrong et al., in press; Bennell et al., 1995; Bennell et al., 1996a; Frusztajer et al., 1990; Nattiv, Puffer & Green, 1997; Warren et al., 2002). Thus, attention should be paid to identifying those athletes with unhealthy eating habits and implementing strategies to improve these habits.

3. Diagnosis

Stress fractures are usually diagnosed clinically. The patient complains of pain associated with physical activity which worsens as the activity continues. If the pain is reduced after a warm up, this is more likely to be a soft tissue injury. There may be pain on palpation of the bone if the site is superficial. Clinical suspicion may be confirmed by performing an X-ray. X-ray appearance of a stress fracture is often quite subtle with the most frequent finding a periosteal reaction. However, X-rays may fail to show a stress fracture until after it has been present for some time and some fractures are notoriously difficult to detect using plain X-ray. Thus, further investigation is often indicated in the patient in whom stress fracture is suspected but X-ray is normal.

A more sensitive examination is the radioisotopic bone scan (scintigraphy), which can demonstrate the location of

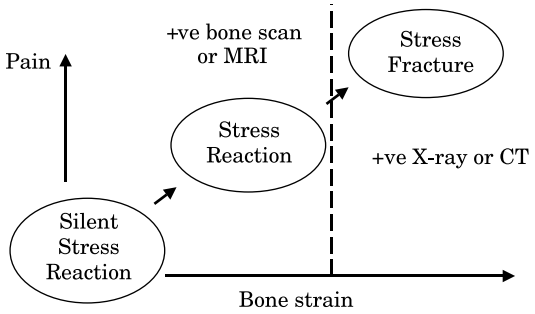

Fig. 3. Relationship between the bone strain continuum and diagnostic findings on bone scan, MRI or CT.

an overuse bony lesion. A localized area of increased uptake or 'hot spot' indicates a stress fracture. These are highly sensitive so that a negative bone scan generally excludes a stress fracture (Matheson et al., 1987a). However, there have been reports of stress fractures diagnosed on MRI when the bone scans have been negative (Keene & Lash, 1992). Since the bone scan is a non-specific investigation other bony abnormalities such as tumours and osteomyelitis may cause similar pictures.

In most cases of stress fracture, a radioisotopic bone scan is sufficient to confirm the diagnosis and no further investigations are required. However, in a few sites that are known to present problems with treatment, such as the tarsal navicular, further information regarding the site and extent of the fracture is required. In these cases, a computed tomographic (CT) scan or MRI may be performed to show the exact site and extent of the fracture.

MRI is being increasingly advocated as the investigation of choice for stress fractures. While MRI does not image fractures as clearly as do CT scans, it is of comparable sensitivity to radioisotopic bone scans in assessing bony damage. The typical MRI appearance of a stress fracture shows periosteal and marrow edema plus or minus the actual fracture line (Aoki et al., 2004). It also does not expose the patient to radiation although is generally more costly than other imaging modalities (Fig. 3). A variety of edema-sensitive MRI sequences is widely available and these generally use some form of fat suppression to enhance contrast. The most widely used edema sequences are short tau inversion recovery (STIR), newer and faster STIR sequences, and fat-suppressed proton density and T2-weighted fast spin echo sequences.

4. Treatment

A number of factors including the site of the fracture, the length of the symptoms and the severity of the lesion (stage in the spectrum of bone strain) will influence the time from diagnosis of a stress fracture to full return to sport or physical activity. Most stress fractures with a relatively brief history of symptoms will heal without complication or delay and permit return to sport within the 4–8 week range. However there is a group of stress fractures that require additional treatment and special consideration including the femoral neck, anterior tibial shaft, sesamoids, navicular, proximal 5th metatarsal and pars interarticularis. While it is beyond the scope of this chapter to cover the treatment of these in detail, readers are referred to other reviews in this area (Brukner, Bennell & Matheson, 1999; Brukner & Bennell, 1997; Egol & Frankel, 2001).

Few treatments for stress fractures have been evaluated in RCTs and these include pneumatic air braces ($n=3$; (Allen et al., 2004; Slayter, 1995; Swenson et al., 1997)), low energy laser ($n=1$; (NissenAstvad & Madsen, 1994)) and low intensity pulsed ultrasound ($n=1$; (Rue et al., 2004)). While there are many subtleties involved in the treatment of stress fractures, the primary treatment is modified activity. During the phase of modified activity, a number of important issues are attended to including modification of risk factors, maintenance of muscular strength and fitness, pain management, investigation of bone health, and prescription of orthotic devices. Following this, there needs to be a period of reintroduction of physical activity to full return to sport (Table 2).

4.1. Pain management

Pain is seldom severe but can be a problem even with normal walking. Practitioners often suggest non-steroidal

Table 2
Treatment of a stress fracture[a]

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 remodelling	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 (e.g. 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

[a] Taken from Bennell K and Brukner P, 2002. How would you treat a stress fracture? In D MacAuley and T Best (Eds) Evidence-based sports Medicine. Blackwell Publishing.

anti-inflammatories (NSAIDs) to assist with initial pain reduction and healing of stress fractures. Because they inhibit cyclooxygenases, NSAIDs will help control inflammatory processes that may accompany injury or overload. However, prostaglandins are essential for normal bone turnover and fracture healing and thus could slow or prevent repair of the stress fracture. This area has been well reviewed by Wheeler and Batt (2005). Since there is no conclusive evidence to date, it would be prudent to limit the use of NSAIDs in patients with stress fractures and use mild analgesics instead for pain relief. In some cases where activities of daily living (ADL) are painful it may be necessary for the patient with a stress fracture to be non-weightbearing or partial weightbearing on crutches for a period of up to 7–10 days. In the majority of cases this is not necessary and merely avoiding the aggravating activity will be sufficient.

4.2. Electrotherapy modalities

Various methods of electrical stimulation have all been shown to have a positive effect on healing of non-union of traumatic fractures (Brighton et al., 1995; Esterhai et al., 1981; Parnell & Simons, 1991; Scott & King, 1994; Swenson et al., 1997). There have been no studies of the efficacy of this treatment on healing of stress fractures and only one non-blinded, uncontrolled study of its effect on time to return to sport in stress fractures in athletes. Benazzo et al. (1995) reported on the use of capacitive coupling in stress fractures predominantly of the navicular and 5th metatarsal which are prone to delayed or non-union. They claimed that 22 of 25 stress fractures were healed and two more showed improvement. Further controlled studies are required to determine the efficacy of this treatment in the management of both the acute stress fracture and in cases of non-uniting stress fractures.

A growing body of evidence provides support for the application of low intensity pulsed ultrasound (US) during fracture repair (Cook et al., 1997; Frankel, 1998; Frankel, Koval & Kummer, 1999; Fujioka et al., 2000; Heckman et al., 1994; Kristiansen et al., 1997; Mayr, Frankel & Ruter, 2000a; Mayr et al., 2000b) but there is only one clinical trial in those with stress fractures. This recent randomized, double-blind placebo controlled trial failed to find an effect of US on healing time in 46 tibial stress fractures treated for 20 min daily with a commercially available US system (Rue et al., 2004). This lack of effect may be due to the relatively small sample size. At this stage, US would not be recommended for routine treatment of stress fractures but might be an option for stress fractures at sites prone to delayed or non-union or in elite athletes. Research is needed to further assess the effects of pulsed US on stress fracture healing including different dosages and different sites.

There has only been one study investigating the application of low power laser treatment in the healing of stress fractures/shin splints (Nissen et al., 1994) with no effect noted.

4.3. Muscle strengthening

While there are no studies that have evaluated the role of muscle strengthening in the treatment of stress fractures, it is logical to include a specific strengthening program because of the important role of muscles in shock absorption and to help counteract the effects of detraining. A specific program of muscular strengthening exercises in muscle groups surrounding the joints above and below the fracture line should be given. Attention should be paid to developing muscle endurance. Muscle strengthening programs are usually prescribed for a period of 6–12 weeks and can begin immediately after diagnosis of the stress fracture. However, it is important that the exercises do not cause pain at the stress fracture site.

4.4. Maintaining fitness and modification of risk factors

Maintenance of fitness during periods of forced inactivity due to injury is a major concern to coaches and athletes. It should be emphasised to the athlete that initially the rehabilitation program is designed to allow the damaged bone time to heal and gradually develop or regain full strength while maintaining the person's fitness in ways that avoid over-loading the bone. Non-loading activities that maintain fitness are those that use as many large muscle groups as possible without over-loading the bone. The most common methods of maintaining fitness are cycling, swimming, water running, rowing and stairmaster. These work-outs should as much as possible mimic the athlete's normal training program in both duration and intensity.

As with any overuse injury, it is not sufficient to merely treat the stress fracture itself. Stress fractures represent the result of incremental overload. Subtle adjustments to modifiable factors that contribute to the total load are an essential component of the management of an athlete with a stress fracture. A thorough history and clinical examination will assist in identifying the factors that may have contributed to the injury and those that can be modified to reduce the risk of injury recurring (Table 1). The fact that stress fractures have a high rate of recurrence is an indication that this part of the management program is often neglected (Bennell et al., 1996b; Milgrom et al., 1985a).

4.5. Bracing

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. (1997) 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 with walking, hopping and running. They also suggest that the brace facilitates healing at the fracture

site by acting to compress 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 enhances osteoblastic bone formation.

Two RCTs, one in military recruits (Slayter, 1995) and one in athletes (Swenson et al., 1997), showed a significant reduction in the time to re-commencing training after diagnosis of stress fracture with the use of a pneumatic leg brace (weighted mean difference −42.6 days, 95% CI −55.8 to −29.4 days; (Gillespie & Grant, 2000). Swenson et al. (1997) 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 traditional group ($n=10$). However, a more 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 (Allen et al., 2004). While results are conflicting, there is some evidence to suggest that a pneumatic brace accelerates return to activity (Gillespie & Grant, 2000) in fibular or tibial stress fractures.

4.6. Resumption of impact loading activities

Resumption of the impact loading activities can begin when normal, day-to-day ambulation is pain free. The rate of resumption of activity is individual and should be modified according to symptoms and physical findings. There are no studies that have compared different return to sport programs. However, since healing bone is weaker, a progressive increase in load is needed so that the bone will adapt with increases in strength. Some studies have evaluated the degree of tibial bone loading during various activities using strain gauges cemented on to the tibia of a small number of healthy volunteers (Kawamoto et al., 2002; Milgrom et al., 2000a, 2003, 2000). The results provide some insight into a possible progression of activities during rehabilitation of tibial stress fractures. A graduated sequence could include cycling, followed by walking, leg press and stairmaster, followed by treadmill running, then straight line outdoor running then sprinting and running on a curved track, then zig-zag running, up/down hills, jumping and lastly zig–zag hopping and rebounding.

For lower limb stress fractures where running is the aggravating activity, a program that involves initial brisk walking increased by 5–10 min per day up to a length of 45 min is recommended. Once 45 min of continuous brisk walking is achieved without pain, slow jogging can begin for a period of 5 min within the 45 min walk. Treadmill running provides less tibial strain than running outdoors. The amount of jogging can be increased by 5 min per session on a daily or every other day basis to a total of 45 min at slow jogging pace. The pace can then be increased, initially half pace then gradually increasing to full pace striding. Once full sprinting is achieved pain free, functional activities such as hopping, skipping, jumping, twisting and turning can be introduced gradually. It is important that this process is a graduated one and it is important to err on the side of caution rather than try to return too quickly.

Resumption of activity should not be accompanied by pain but it is not uncommon to have some discomfort at the site of the stress fracture. If bony pain occurs then activity should be ceased for 1–2 days. If pain free with normal ambulation, the activity is resumed at the volume and pace below the level at which the pain occurred. The patient should be clinically reassessed at two weekly intervals, to assess the progress of the training program and any symptoms related to the stress fracture. It is not necessary to monitor progress by radiography, scintigraphy, CT or MRI since radiological healing often lags behind clinical healing.

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