Clinical Article

Management of sports overuse injuries of the lower limb: an evidence-based review of the literature

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Abstract

This article reviews common lower limb sports overuse injuries relevant to the orthopaedic surgeon. The following conditions are covered:

- Snapping hip syndrome
- Iliotibial band syndrome
- Patellar tendinopathy
- Achilles tendinopathy
- Medial tibial stress syndrome
- Tibial stress fractures
- Chronic compartment syndrome

These conditions can be managed conservatively inmost cases. Adequate rest followed by a graded rehabilitation is extremely important. Intrinsic and extrinsic contributing factors must be sought for and corrected. It is only in the uncommon case of failed conservative treatment that surgical intervention is necessary. For each of the above conditions, the indications for surgery, surgical principles, various surgical procedures and results and complications thereof are analysed and discussed. A meta-analysis of surgical treatment studies with similar methodologies was performed. The majority of studies found were of Level IV evidence with small patient numbers. The recommended outcome measures to assess results of surgical intervention are relief of pain and return to pre-injury level of sporting activity. Surgical treatment often does not result in a cure but an improvement of symptoms. Prospective randomised control trials with adequate patient numbers comparing different surgical treatments are needed.

Introduction

Sports overuse injuries are common in a modern society of increased sports participation and professionalism. Impressively 50% of sports injuries are due to overuse injuries.[1]

This article reviews the most common lower limb sports overuse injuries relevant to the orthopaedic surgeon, specifically: snapping hip syndrome, iliotibial band syndrome, patellar tendinopathy, Achilles tendinopathy, medial tibial stress syndrome, tibial stress fractures and chronic compartment syndrome.[2]

Most of the time, these injuries can be successfully treated conservatively by a multidisciplinary team consisting of a combination of the general practitioner, physiotherapist, biokineticist and sports physician. Surgery is only indicated in uncommon cases of failed conservative treatment. Apart from these infrequent referrals for possible surgical intervention, the orthopaedic surgeon is commonly consulted by general practitioners for opinion on lower limb pathology, which includes sports overuse injuries. In lieu of the above, a broad understanding of the conditions is necessary to manage these patients effectively.

In general, lower limb sports overuse injuries occur when either repetitive friction between two tissue surfaces or tissue overload causes micro trauma. With continuous activity, there is inadequate time for recovery and tissue damage results.[2] Predisposing factors to sports overuse injuries are divided into intrinsic and extrinsic factors. Intrinsic factors are very often an athlete's biomechanical abnormalities. The most common extrinsic factors are changes in method, intensity or duration of training.[2] In the majority of cases a definitive diagnosis can be made on clinical grounds alone. Conservative treatments should be both supervised and adequate. Relative rest is important and must be followed by a graded rehabilitation programme. The intrinsic and extrinsic contributory factors must be sought for and corrected. There are a multitude of adjuvant treatments that both stimulate healing and improve tissue quality of strength, flexibility and endurance.

The athlete referred for surgery with a lower limb sports overuse injury has often undergone a prolonged and failed conservative treatment programme. At this stage, the athlete is not only frustrated but may also face a potentially career ending injury. Nevertheless, the orthopaedic surgeon must take time to revisit the history, physical examination, special investigations and treatment to confirm the correct diagnosis and ascertain whether conservative treatment has been adequate. It is imperative to know the surgical indications, principles, various techniques, as well as the results and complications. This article reviews the literature on these points for each condition. Data presented is based on articles obtained by a Medline literature search of studies on the surgical treatment of these lower limb sports overuse injuries over the last 10–20 years. A meta-analysis of studies with similar methodologies was performed and when possible, comparisons of the various surgical techniques were made.

Snapping hip syndrome

Snapping hip syndrome (coxa saltans) is a group of conditions characterised clinically by a painful, audible snap occurring during hip flexion and extension.[3] It affects mostly the young adult runner**,** dancer and rower.

There are two types of snapping hip:

1. **External snapping** is caused by increased tension of the iliotibial band (ITB) and/or its gluteus maximus muscle insertion as they slide anteriorly and posteriorly over the greater trochanter resulting in inflamed, thickened and fibrosed bands. There can be an associated greater trochanteric bursitis.[4-7]
2. **Internal snapping** is caused by iliopsoas tendon shifting from lateral to medial with snapping against the pubic bone.[8,9]

Pain-free snapping should be considered a normal occurrence.[9,10] The patient will give no history of trauma. The pain is described as a dull ache but becomes severe with certain movements. With external snapping hip, the snap is felt over the greater trochanter (patients frequently describe a sense that the hip is dislocating, termed pseudosubluxation) while with internal snapping it is over the groin area. The patient is often able to reproduce the snapping during examination.[6]

Clinically it can be difficult to differentiate internal snapping hip from intra-articular hip pathology causing sounds. Dynamic ultrasound studies, magnetic resonance arthrography and hip arthroscopy can be helpful.[11,12]

Treatment

The mainstay of treatment of snapping hip is conservative.[3,4,13] Rest and reassurance is often all that is needed. Adjuvant treatment modalities include non-steroidal anti-inflammatory drugs, steroid infiltration and physiotherapy. Importantly the patient must avoid activities that cause snapping.[6,14]

Surgical intervention is indicated for uncommon, refractory cases, where 3–6 months of conservative treatment has failed.[3,4,7,13,14]

Surgical treatment of external snapping hip

The important surgical principle is to decrease the tension of the proximal one-third of the ITB complex as it slides over the greater trochanter.[6]

This can be achieved by:

1. **Lengthening of the ITB by Z-plasty**. Careful planning of the Z-plasty is needed to obtain maximum length. An Ober's test should be performed intra-operatively to ascertain if lengthening is adequate.[4,15,16]
2. **Release of the ITB and/or gluteus maximus** most often by multiple incisions. Intra-operatively one must check if the release is adequate by taking the hip through a provocative range of movement – specifically flexion, adduction and internal/external rotation. Also make sure the tight fibrous bands of fascia lata posteriorly and gluteus maximus anteriorly are released.[5,7]
3. **Excision of posterior half of the ITB**. This is most often an open procedure[17] but a technically demanding arthroscopic technique has been described.[18]

Furthermore, an inflamed trochanteric bursa must be excised.[5,7,17,18]

Postoperatively there are no restrictions in release[5,7] or excisional[17,18] procedures but partial weight bearing for 2 weeks is needed after Z–plasty lengthening[4,15,16] procedures.

A meta-analysis of the results of studies on the different surgical techniques showed that similar, good results were achieved with all techniques. A cure, of no snapping and being pain-free, was achieved in 88% (75–100%) of Z-plasties,[4,15,16] 85% (73–89%) of multiple releases[5,7] and 76% (71–91%) of posterior resections[17,18] (*Figure 1*).

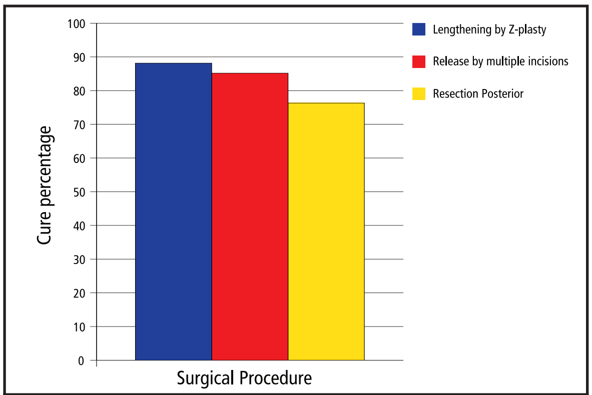

Figure 1. Meta-analysis of results of surgical treatment of external snapping hip

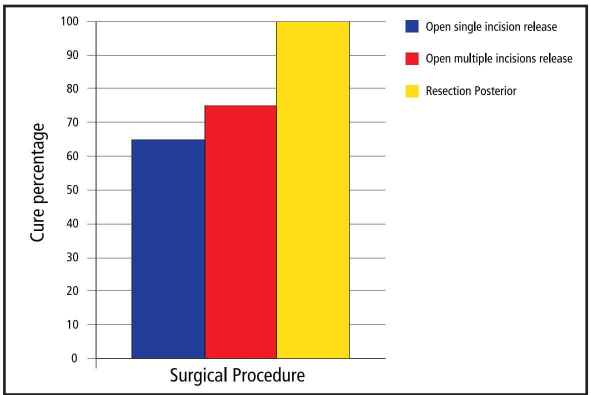

Figure 2. Meta-analysis of results of surgical treatment of internal snapping hip

There were minimal complications, although some patients reported mild subjective weakness and a limp with lengthening procedures.[7]

The most common cause of failed surgery was residual bands or recurrent adhesions.[5,16] In these patients snapping may have recurred but pain was often significantly less.[5,7,15,16] In those with significant pain, a second procedure (a repeat of the initial surgical technique) was found to be curative.[5,16]

Surgical treatment of internal snapping hip

The important surgical principle is to decrease the tension on the iliopsoas tendon.[6]

This can be done by:

1. **Fractional lengthening** of the iliopsoas tendon via either a single incision of the tendinous portion at the musculotendinous junction[13,19,20] or multiple partial incisions.[3,14] It is most often an open procedure and here care must be taken to avoid injuring the femoral and lateral cutaneous nerves.[3,13,14,19,20]
2. **Full release** of the iliopsoas tendon at its insertion on the lesser trochanter. This is most often performed arthroscopically.[10,11,21] Postoperatively patients mobilise as pain allows.[3,10,11,21] Return to sports activity is usually at 3 months.[11,13]

Open fractional lengthening procedures[3,13,14,19,20] gave a cure, of having no snapping and being pain-free, in 74% (56–89%) of cases. Multiple incision release procedures[3,14]produced better results than single incision releases:[13,19,20] 75% (70–78%) versus 65% (56–89%) cure rates (*Figure 2*). Complications reported were sensory nerve injury in 9–50 % of cases and hip flexor weakness occurring in 4–45% of patients.[3,13,14,19,20] If snapping did recur, there was often a significant reduction in pain.[3,13,14,19,20] Recurrence of snapping can be due to inadequate tendon lengthening and excessive scarring with adhesions.[21]

Arthroscopic treatment of internal snapping hip is most often a full release procedure[10,11,21] but a fractional lengthening procedure[10] has also been studied. It must be noted that there is a 75% (57–83%) incidence of associated intra-articular hip pathology in internal snapping hip patients, most often anterosuperior labral tears and femoral acetabular impingement.[10,11,21] The few small case series[11,21] (n = 6 and 7 respectively) on arthroscopic release have shown promising results with 100% cure rates (*Figure 2*). The intra-articular hip pathology can be addressed at the same time and sensory nerve injury complications are avoided. However, a concern is that full release procedures resulted in hip flexor weakness in 54% (0–100%) of patients.[11,21]

With an open release of the iliopsoas tendon it might be beneficial to assess for treatable intra-articular hip pathology either by a pre-operative MRI arthrogram[11] or alternatively a hip arthroscopy at the time of surgery.[6,10,11,21] If arthroscopic release of the iliopsoas tendon is the preferred treatment, it should be considered to perform fractional lengthening rather than full release procedures in order to avoid the complication of hip flexion weakness.

The majority of studies on external snapping hip (n = 5–44)[4,5,7,15-18] and internal snapping hip (n = 6–92)[3,10,11,13,14,19-21] to date have been case series with small sample sizes. Randomised controlled trials comparing the various surgical techniques are required. A significant problem is the lack of study patient numbers as conservative treatment is mostly successful.

Iliotibial band syndrome

Iliotibial band syndrome (ITBS) is a common cause of lateral knee pain in athletes participating in sports with repetitive knee flexion. It is particularly prevalent in long-distance runners, cyclists and football players.[22]

It results from excessive lateral to medial compression of fatty, connective tissues between the posterior iliotibial band (ITB) and the lateral femoral epicondyle at an impingement zone of 30° flexion.[23,24] The resultant inflamed tissues may form a pathological adventitial bursa. An alternative theory is that this 'bursa' is actually an out-pouching of synovium from the lateral synovial recess in the suprapatellar pouch.[22,25,26]

Extrinsic predisposing factors include increase in training intensity, downhill running and running in same direction on a track. Common intrinsic factors are weak hip abductors, varus deformities of the leg and heel as well as forefoot supination.[22,27]

The diagnosis is made on clinical grounds. The athlete complains of lateral knee pain coming on after a specific duration of exercise or distance run, usually not before 3 km. The pain is then progressive. An Ober's test is often positive. Noble's compression test (pressure applied to the lateral femoral epicondyle while passively extending the knee causes familiar pain at 30°) is usually confirmatory. Importantly, the hip abductors should be assessed for weakness.[28,29]

Treatment

Conservative treatment is mostly successful.[28,30,31] A rest period of 4–6 weeks, with graded return to sporting activity, is imperative. Physiotherapy is often beneficial. If recurrent, the entire lower limb kinetic chain must be analysed and abnormalities addressed. Excessive supination is corrected with lateral heel wedges.[22,28,29,31]

Surgery is warranted in the uncommon situation (1–6% of patients) in which symptoms do not improve after 6–12 months of conservative treatment.[22,23,29]

Surgical treatment

The important surgical principles are:

1. **Decrease impingement of the tighter posterior fibres of the ITB on the lateral femoral epicondyle**.[22,28]
2. **The pathological tissue underlying the ITB needs to be excised**.[25,27]

In a meta-analysis of studies of the various surgical techniques, the following procedures gave similar excellent (no pain with activity) to good (significantly less pain with activity) results in 95% or more of cases operated:

- incision of the posterior fibres of the ITB[27,32,33]
- excision of either a triangle[29] or ellipse[33] of the posterior fibres of ITB
- Z-plasty lengthening of the distal ITB[31]
- arthroscopic resection of the lateral synovium.[25]

Less success was achieved in procedures done under local anaesthetic,[27] standalone open bursectomies[23] and percutaneous releases[33] with excellent to good results obtained in 85%, 82% and 25% of cases respectively. Routine knee arthroscopy does not add to the treatment of ITBS, unless there is associated intra-articular pathology.[23,31,33]

Procedures generally have low morbidity and quick recovery, with weight-bearing postoperatively as pain allows and usually return to sport at 4–6 weeks.[22,25]

Tendinopathies of the lower limb

Patella and Achilles tendinopathies are common tendinopathies of the lower limb in the athlete.

Tendinopathy refers to overuse injuries of tendons presenting with pain, swelling and functional limitation, as well as the histopathological entities of paratendinitis and tendinosis. **Tendinosis** describes lipoid degeneration of the tendon with associated in-growth of disorganised vascular and neural elements (neoneurovascularisation).[34] The latter is thought to be the primary pain generator in tendinopathies.[34-37] **Paratendinitis** is a precursor to tendinosis[38] and refers to the pathology or imaging proven presence of inflammation and adhesions in the paratenon.[34,39]

Repetitive eccentric and concentric tendon overload is hypothesised to be the cause of tendinopathies. This theory is questioned however, especially as eccentric overload exercises are a well-described treatment modality.[35,36,39]

The diagnosis of a tendinopathy is most often made clinically.[34,40]

Special investigation with conventional ultrasound shows a hypo-echoic area representing the degenerative tendon; however, this finding is present in 20% of asymptomatic individuals. Colour Doppler ultrasound is more specific and can be used to prognosticate and assess treatment response. It depicts regions of increased blood flow corresponding to the neoneurovascularised tissue. These findings disappear with successful treatment.[37]

The aims of treatment are to deload the tendon, reactivate healing and then strengthen the tendon. Ablation of the neoneurovascularised tissue eliminates pain and stimulates healing.[35-37]

Relative rest is paramount.[34,41] Adjuvant treatment modalities of proven benefit include eccentric exercises, injection of a sclerosing agent, dry-needling with subsequent autologous blood injection, cryotherapy, deep friction massage, ultrasound and extracorporeal shock wave therapy.[36,37] The beneficial effect of peritendinous steroid injections is still controversial.[36,39]

Surgery is indicated if the condition fails to improve after 6 months of conservative treatment.[37,38,42-44]

The surgical principle is to excise degenerative tissue and reactivate healing.[36,39,41]

Excising the degenerative tissue alone is not enough and multiple longitudinal tenotomies (scarification) should be performed in addition to stimulate a more vigorous healing response.[36,45-47] With paratendinitis, the paratenon should not be excised but rather only the adhesions released and the degenerative areas trimmed away.[37,38] This avoids complications of seroma formation and preserves the gliding function of the paratenon.[48]

Postoperatively, the patient mobilises as pain allows, followed by a graded physiotherapy rehabilitation programme.[37,49]

Prolonged recovery (6–12 months) from tendinopathy is normal, whether following conservative or surgical treatment. This is due to the often advanced tendon degeneration that is present as well as the slow metabolic rate of tendon tissue.[41,42,50,51]

Patella tendinopathy

With patella tendinopathy (Jumper's knee) there is tendinosis of the deep posterior portion of the patella tendon at its proximal attachment to the distal pole of patella.[42]

It is common in young athletes involved in repetitive jumping sports as well as long-distance runners. A contributing causative factor could be mechanical impingement of the tendon by an elongated distal pole of the patella, perhaps representing a traction osteophyte.[43,52] Other intrinsic factors include inflexibility of hamstrings and quadriceps muscles, reduced ankle dorsiflexion and higher body mass index (BMI).[42,53]

The patient complains of anterior knee pain, which is worse during sporting activities. On examination there is pin-point, marked tenderness of the proximal patella tendon. In uncertain cases, MRI can be useful in excluding the wider differential of anterior knee pain.[42,43]

Treatment

Additional conservative treatments related to patella tendinopathy management include varying the training programme, playing on softer surfaces and improving abnormal landing.[43,53]

Surgical treatment

Specifics of the surgical treatment to the patella tendon are:

1. **Excision of the tendinosis tissue**;[40,49,51,54,55] arthroscopic techniques are well described.[49]
2. Reactivation of the healing process via **drilling of the inferior pole of patella**[40,55] and **scarification**.[40,56]
3. **Resection of the impinging inferior pole of the patella**.[52]

Following this is a discussion comparing the results of different treatments of patella tendinopathy.

Excision of the tendinosis tissue alone[49,51,54] obtained excellent results (return to pre-injury level of sports activity without pain) in 37% (25–42%) and good results (return to pre-injury level of sporting activity with mild or moderate pain) in 13% (4–25%) of patients. It must be noted that similar results were achieved in a conservatively treated control group[51] (*Figure 3*). Arthroscopic excision procedures[49] gave comparable results to open excision procedures.[49,51,54]

Excision of tendinosis tissue combined with multiple longitudinal tenotomies (scarification) and distal patella pole drilling[40,55] produced significantly better outcomes than excision of tendinosis tissue alone,[49,51,54] with excellent results in 67% (56–70%) and good results in a further 19% (15–44%) of patients (*Figure 3*).

It must be noted that **arthroscopic resections of the distal pole of the patella with no debridement of the patella tendon**[52] gave similar outcomes to excision, scarification and patella pole drilling procedures,[40,55] with excellent to good results in 90% of patients (*Figure 3*). This supports the proposed pathogenesis of distal patella pole impingement.

Prospective randomised controlled trials, comparing the different surgical procedures as well as combinations of them, are needed to establish best practice.

Achilles tendinopathy

Achilles tendinopathy can present as a **paratendinitis, tendinosis or both**.[34]

It is common in runners, soccer and racquet ball players. Contributing intrinsic factors include the older athlete, female sex, high BMI, tall height, pes cavus, forefoot varus and lateral instability of the ankle. Important extrinsic factors are training errors and poor footwear.[2,36,44]

The patient presents with pain in the mid-Achilles region. Initially pain is experienced after exercising but with disease progression it can be present throughout sporting activity. On examination the tendon may be swollen and tender as well as thickened and nodular.[36,44] Insertional tendinopathy is a separate pathological entity that does not form part of this review.

Treatment

Orthotics can be used to correct foot and ankle malalignment.[44] It has been shown that conservative treatment is unsuccessful in 24–29% of patients with Achilles tendinopathy.[57,58]

Surgical treatment

The need for surgery increases with patient age, duration of symptoms and occurrence of tendinopathic changes.[59] It is important not to delay surgery as longer-standing Achilles tendinopathies treated surgically have poorer outcomes.[44,47]

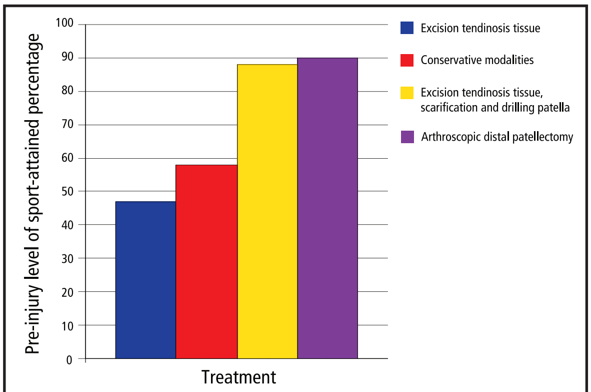

Figure 3. Meta-analysis of results of treatment of resistant patella tendinopathy

Tallon *et al* in 2001 performed a systematic review (26 studies) of the outcome of surgical treatment of Achilles tendinopathy. The authors recommended surgical treatment was to debride the paratenon, excise the tendinosis tissue and scarify the tendon.[60]

If tendinosis alone is present, a percutaneous scarification procedure is an alternative to open procedures giving 77% excellent (pain-free) to good (full return to pre-injury sport activity with occasional mild discomfort) results.[46]

The most common complication of surgery is wound breakdown and infection, especially occurring in open, more extensive procedures.[48]

Chronic leg pain

Chronic leg pain in the athlete is common. The differential diagnosis, in order of prevalence, includes medial tibial stress syndrome, stress fracture of the tibia and chronic compartment syndrome of the leg.[61] A definitive diagnosis of the cause of the leg pain can most often be made on clinical grounds alone.[61,62] However, in difficult cases, a bone scan or more specific MRI[63] can be helpful. If chronic compartment syndrome is suspected, it is best to rule out other differential diagnoses before performing compartmental pressure measurements, as the latter are invasive.[64]

Medial tibial stress syndrome

Medial tibial stress syndrome (MTSS) or 'shin splints' is a condition in which the running and jumping athlete experiences diffuse pain along the distal, posteromedial border of the tibia.[62]

It typically occurs when intensity or duration of activity is increased. An important intrinsic factor is excessive foot pronation.[62,65]

A definitive diagnosis of the cause of the leg pain can be made on clinical grounds alone, but a bone scan or MRI can be helpful

Two pathogeneses have been proposed:

1. **Chronic soleus traction injury** relates to the soleus muscle being the anatomical structure that arises at the site of injury.[63,65]
2. **Chronic bone stress injury** where chronic, repetitive, weight-bearing activities induce tibial bending, leading to a stress injury.[63,66]

Pain and tenderness is located along a **third of the length of the posteromedial tibia**, centred at the junction of distal and middle thirds of the leg. The pain is present at the beginning of the workout, often disappearing while exercising, only to return during the cool-down period. **Bone scanning shows diffuse uptake in the affected region of the tibia**.[2,62,65]

Treatment

Relative rest for 2 weeks, followed by a graded rehabilitation for 4–6 weeks, is most important.[2,65] Foot orthoses can be used in patients who excessively pronate.[65] Recurrence is most often the result of the athlete returning to pre-injury pace too quickly.[2,62]

Surgical treatment is rarely needed. It is indicated in the athlete who has failed one year's conservative treatment or in whom the condition is recurrent (two or more times).[2,62,65]

Surgical treatment

The proposed surgical principle is to **alleviate the pull of the soleus muscle at its fascial insertion**.[65]

Release of the soleus insertion is achieved by performing a superficial posterior compartment fasciotomy.[65,67] Importantly, it must include the soleus bridge (the distal thicker and stronger fascia just proximal to the flexor retinaculum) as this is the primary fascial insertion of the medial soleus.[62] In addition, the periosteum must be removed or cauterised. This denervates the bone and stimulates healing.[65] Some authors believe that the origin of flexor digitorum longus is also involved in causing pain in MTSS and perform, in addition, a deep posterior compartment release.[62,65]

Table I: Results of surgical treatment of MTSS

		No. of limbs operated	Excellent %	Good %	Fair %	Poor %
Yates (2003)[62]	Superficial and deep compartment release	n=88	35	34	22	9
Holen (1995)[67]	Superficial compartment release only	n=61	33	39	21	7

Result	Reduction in pain
Excellent	81-100 %
Good	61-80 %
Fair	41-60 %
Poor	< 40 %

Postoperatively patients must follow a graded rehabilitation programme similar to that used in non-operative treatment.[67]

The results of releasing the superficial posterior compartment alone[67] and releasing both the superficial and deep posterior compartments[62] were found to be similar (*Table I*).

It must be noted that surgery is not curative. Excellent results were achieved in only 33–35% of patients operated.[62,67] Mean reduction in pain was found to be 70% at long-term (> 6 months) follow-up.[62]

Reviewing the proposed pathogeneses, if MTSS is caused by chronic bone stress, performing a fasciotomy makes little sense. Possible benefits of the surgical procedure could be stimulation of healing, denervation of the bone, an enforced rest period postoperatively and the graded rehabilitation programme.

It is important that patients be informed pre-operatively regarding the results of surgery and cautioned not to expect complete relief of pain and uninhibited return to pre-injury sporting level.

Stress fracture of the tibia

The tibia is the most common bone to get a stress fracture in athletes.[68]

The fracture is the result of excessive direct and indirect bone stresses. An increase in the intensity of training is the most frequent extrinsic factor. Intrinsic factors contributing are: female sex, hormonal imbalance, poor nutrition, metabolic bone disorders and a narrow tibial width.[2,61,69,70]

There are two types of tibial stress fractures:

1. **Compression type (85%)** occurs in the **proximal and distal third** of the tibia, on the compression side, i.e. **posteromedially**. This is a stress fracture of **runners**. They **heal well**.
2. **Tension type (15%)** occurs in the **central portion** of the tibia, on the tension side, i.e. **anteriorly**. This is a stress fracture of the **jumping athlete**. They **heal poorly** due the fracture being under tension as well as the hypovascularity of the anterior tibia. This makes these fractures prone to delayed unions and non-unions as well as increasing risk of progression to a complete fracture.[69,71,72]

The patient complains of the insidious onset of leg pain which is aggravated by training and relieved with rest. On examination the **tenderness is well localised to the bone**. X-ray changes of a periosteal reaction, cortical scalloping or a transverse fracture line are visible only after 2–3 weeks. In tension type fractures there is a characteristic V-shaped defect in the anterior cortex of the tibia, referred to as the 'dreaded black sign'. With attempted remodelling, the cortex thickens resulting in obliteration of the medullary cavity. **Bone scan shows localised, fusiform uptake**.[69,73,74]

Predictors of non-union are an unusually narrow medullary canal ratio of less than 0.3 and poor uptake on a three-phase bone scan.[69,73]

Treatment

The vast majority of tibial stress fractures, both tension and compression type, heal with conservative treatment.[2,61,75]

This consists of restricting activities until pain-free (on average 4–6 weeks) followed by a graded return to sports activity.[70,74] Treatment adjuvants include electrical bone stimulators, pulsed low-intensity ultrasound and physiotherapy.[71,74] The wearing of a pneumatic brace may allow return to activity earlier.[69,70] The athlete with multiple stress fractures or with delayed fracture healing needs a nutritional, hormonal and medical work-up.[70]

For tension type fractures, some authors recommend initial treatment of below-knee casting for a 6–8 week period, prolonged to 3 months if necessary.[71,76]

Surgery is only ever necessary in the tension-type tibial stress fracture. It is indicated when 3–6 months of conservative treatment has failed.[72-74]

Surgical treatment

The surgical principles are **to improve stability at the fracture site and biologically stimulate fracture healing**.[77]

Various surgical options have been studied:

- drilling and bone grafting[72,74]
- intramedullary nailing[73,74,78]
- anterior tension plating.[71]

Studies on these surgical treatments[71-74,78] are of small sample size (n = 3–11) and their results (presented below) must be interpreted with caution. All the above mentioned surgical techniques[71-74,78] resulted in fracture union, but their indications, times to union and return to sport as well as complications differed.

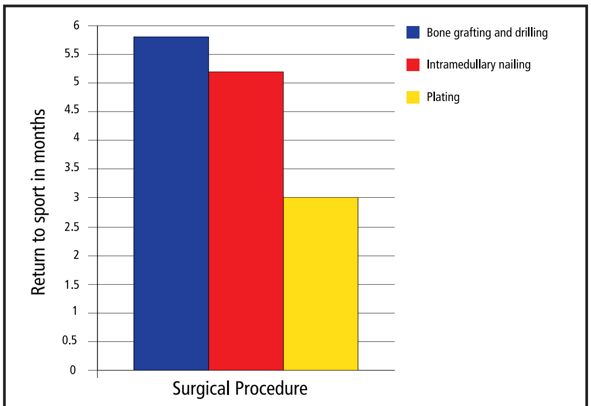

Figure 4. Meta-analysis of time to return to sport after surgical treatment of delayed union stress fractures of the tibia

Open reduction and internal fixation (tibial nailing and plating), rather than drilling and bone grafting, should be performed in complete, displaced or multiple fractures.[74]

Tibial plating procedures[71] gave the best results, with return to sport at a mean time of 3 months compared to tibial nailing[73,74,78] at 5.3 months (4–10) and drilling and bone grafting[72,74] at 5.8 months (5.5–6) (*Figure 4*).

Both tibial nailing and plating procedures stabilise the fracture allowing the patient to fully weight bear immediately postoperatively. This is compared to drilling and bone-grafting procedures where patients must toe-touch weight-bear for 4–6 weeks.[74] Tibial plating neutralises deforming forces more effectively than tibial nailing with resultant earlier union.[71,73,74,78] The disadvantage of plating is its subcutaneous placement on the anterolateral tibia which can cause problems with wound healing and prominent hardware.[71] Tibial nailing can be complicated by anterior knee pain.[73] The advantage of a drilling and bone-grafting procedure is that it avoids the complications associated with tibial nailing and plating.

Larger, prospective randomised control trials comparing the different surgical treatments are recommended.

Chronic compartment syndrome

Chronic compartment syndrome (CCS) of the leg is an exertion-induced compartment syndrome that affects young athletes, especially runners and football players.[79]

Normal, resting leg compartmental pressures are less than 15 mmHg and with exercise these pressures increase transiently.[80] In CCS, either baseline compartment pressures are increased or there is a delay in return of pressures to normal post-exercise. This is most often due to thickened and unyielding surrounding fascias and/or leg muscle hypertrophy. Symptoms result from the muscle ischaemia or the pressure effects alone.[79-81]

The most common compartments affected are the anterior compartment (40–70%) and the deep posterior compartment (15–30%), with the superficial posterior (12%) and the lateral compartment making up the remainder. Multiple compartments are often involved.[79,80]

The patient complains of a dull ache in the muscle mass of the leg during training. There may be associated paraesthesias. The symptoms begin after a specific distance or duration of exercise and resolve within minutes to hours after stopping the sporting activity. **Examination of the leg is normal if the patient is presently asymptomatic**. Both legs are affected in 70–80% of athletes and if unilateral, there is likely a history of previous leg injury.[80]

The diagnosis is confirmed by demonstrating elevated, post-exercise, resting, intra-compartmental pressures. These are commonly measured with a portable, simple needle measurement device and all four of the leg's compartments should be measured.[64] The diagnostic criteria of Pedowitz[82] are most often used and include any one of the following:

- pre-exercise pressure greater than 15 mmHg
- 1 minute post-exercise pressure of greater than 30 mmHg
- 5 minute post-exercise pressure in excess of 20 mmHg.

Treatment

There is no place for conservative treatment of CCS in the patient who wishes to return to same level sporting activities.[61,64,80,83]

The treatment principle is to surgically decompress the affected compartments.[64,80]

Most surgical techniques are variants of the classic double incision fasciotomy technique described by Mubarak and Owen.[84]

Treatment options are:

1. **Fasciotomy** utilising:
   - an open technique[85]
   - minimally invasive subcutaneous technique[79,80,86]
   - endoscopic technique[61] (for anterolateral compartments only)
2. **Fasciectomy** in which a strip of fascia is excised[80]

Only the affected compartments need release.[64,85] The skin is closed primarily in all procedures. The limb must be elevated for 24–48 hours postoperatively whereafter weight-bearing is allowed as tolerated. A graded rehabilitation programme should be followed with return to sporting activity at 8–12 weeks.[83]

Best results were achieved with open procedures with excellent to good results (no limitation of duration and extent of exercise as well as minimal to no discomfort during or after exercise) in 90% of open fasciotomies,[85] and 92% of open fasciectomies[79,80] were less effective with excellent to good results in 82% (75–85%) of cases (*Figure 5*).

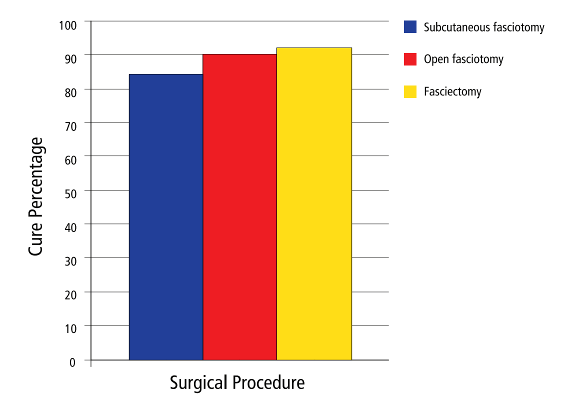

Figure 5. Meta-analysis of the results of the surgical treatment of CCS

The majority of sports overuse injuries of the lower limb can be treated successfully with conservative treatment

Furthermore, open procedures are proposed to be safer than subcutaneous procedures since they provide better exposure with improved haemostasis, lower risk of neurovascular injuries and a more complete release.[80] The disadvantage of an open procedure is a large scar.[83] The advantage of a fasciectomy procedure over a fasciotomy procedure is that the former theoretically decreases the risk of scarring and therefore recurrence of CCS.[80] Some authors recommend open fasciectomy as a primary treatment while others reserve it for the patient with recurrent CCS.[64]

Conclusion

The majority of sports overuse injuries of the lower limb can be treated successfully with conservative treatment. It is only in the uncommon situation where a supervised course of conservative treatment (usually 6 months) fails that surgery is warranted.

Most studies to date on the surgical treatment of sports overuse injuries of the lower limb have been small, retrospective cohort studies or case series of Level IV evidence. Comparison of these studies is complicated by varied study methodologies. Larger, prospective, randomised control trials, comparing the different surgical treatments, are needed to further elucidate best practice. Study methodologies, including selection criteria for surgery, results analysis and outcomes measures, must be standardised. Recommended postoperative outcome measures are relief of pain and return to pre-injury level of sporting activity.[60]

Surgery can offer the failed conservatively treated athlete a fair chance of returning to pre-injury level of sports activity. However, the athlete must be well informed of the results of surgical treatment in that cure is not guaranteed and many will still suffer from residual symptoms. Furthermore the athlete must be diligent in following a supervised, graded postoperative rehabilitation programme.

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References

1. Herring SA, Nilson KL. Introduction to overuse injuries. *Clin Sports Med* 1987 Apr;**6**(2):225-39.
2. Wilder RP, Sethi S. Overuse injuries: tendinopathies, stress fractures, compartment syndrome, and shin splints. *Clin Sports Med* 2004 vi; Jan;**23**(1):55-81.
3. Hoskins JS, Burd TA, Allen WC. Surgical correction of internal coxa saltans: a 20-year consecutive study. *Am J Sports Med* 2004 Jun;**32**(4):998-1001.
4. Provencher MT, Hofmeister EP, Muldoon MP. The surgical treatment of external coxa saltans (the snapping hip) by Z-plasty of the iliotibial band. *Am J Sports Med* 2004 Mar;**32**(2):470-6.
5. White RA, Hughes MS, Burd T, Hamann J, Allen WC. A new operative approach in the correction of external coxa saltans: the snapping hip. *Am J Sports Med* 2004 Sep;**32**(6):1504-8.
6. Byrd JWT. Snapping hip. *Oper Tech Sports Med* 2005;**13**(1):46-54.
7. Yoon TR, Park KS, Diwanji SR, Seo CY, Seon JK. Clinical results of multiple fibrous band release for the external snapping hip. *J Orthop Sci* 2009 Jul;**14**(4):405-9.
8. Deslandes M, Guillin R, Cardinal E, Hobden R, Bureau NJ. The snapping iliopsoas tendon: new mechanisms using dynamic sonography. *AJR Am J Roentgenol* 2008 Mar;**190**(3):576-81.
9. Winston P, Awan R, Cassidy JD, Bleakney RK. Clinical examination and ultrasound of self-reported snapping hip syndrome in elite ballet dancers. *Am J Sports Med* 2007 Jan;**35**(1):118-26.
10. Ilizaliturri VM,Jr, Chaidez C, Villegas P, Briseno A, Camacho-Galindo J. Prospective randomized study of 2 different techniques for endoscopic iliopsoas tendon release in the treatment of internal snapping hip syndrome. *Arthroscopy* 2009 Feb;**25**(2):159-63.
11. Flanum ME, Keene JS, Blankenbaker DG, Desmet AA. Arthroscopic treatment of the painful 'internal' snapping hip: results of a new endoscopic technique and imaging protocol. *Am J Sports Med* 2007 May;**35**(5):770-9.
12. Yamamoto Y, Hamada Y, Ide T, Usui I. Arthroscopic surgery to treat intra-articular type snapping hip. *Arthroscopy* 2005 Sep;**21**(9):1120-5.
13. Gruen GS, Scioscia TN, Lowenstein JE. The surgical treatment of internal snapping hip. *Am J Sports Med* 2002 Jul-Aug;**30**(4):607-13.
14. Jacobson T, Allen WC. Surgical correction of the snapping iliopsoas tendon. *Am J Sports Med* 1990 Sep-Oct;**18**(5):470-4.
15. Lamb KS, Sirvastiva VM, Moulton A. Z-plasty lengthening of the iliotibial band for the snapping hip. *J Bone Joint Surg Br* 2003;**85-B**(Suppl 2):103.
16. Brignall CG, Stainsby GD. The snapping hip. Treatment by Z-plasty. *J Bone Joint Surg Br* 1991 Mar;**73**(2):253-4.
17. Larsen E, Johansen J. Snapping hip. *Acta Orthop Scand* 1986 Apr;**57**(2):168-70.
18. Ilizaliturri VM,Jr, Martinez-Escalante FA, Chaidez PA, Camacho-Galindo J. Endoscopic iliotibial band release for external snapping hip syndrome. *Arthroscopy* 2006 May;**22**(5):505-10.
19. Dobbs MB, Gordon JE, Luhmann SJ, Szymanski DA, Schoenecker PL. Surgical correction of the snapping iliopsoas tendon in adolescents. *J Bone Joint Surg Am* 2002 Mar;**84-A**(3):420-4.
20. Taylor GR, Clarke NM. Surgical release of the 'snapping iliopsoas tendon'. *J Bone Joint Surg Br* 1995 Nov;**77**(6):881-3.
21. Ilizaliturri VM,Jr, Villalobos FE, Jr, Chaidez PA, Valero FS, Aguilera JM. Internal snapping hip syndrome: treatment by endoscopic release of the iliopsoas tendon. *Arthroscopy* 2005 Nov;**21**(11):1375-80.

22. Barber FA, Sutker MJ. The iliotibial band syndrome: Diagnosis and surgical management. *Tech knee surg* 2008;**7**(2):102-106.
23. Hariri S, Savidge ET, Reinold MM, Zachazewski J, Gill TJ. Treatment of recalcitrant iliotibial band friction syndrome with open iliotibial band bursectomy: indications, technique, and clinical outcomes. *Am J Sports Med* 2009 Jul;**37**(7):1417-24.
24. Fairclough J, Hayashi K, Toumi H, Lyons K, Bydder G, Phillips N, *et al*. The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. *J Anat* 2006;**208**(3):309-316.
25. Michels F, Jambou S, Allard M, Bousquet V, Colombet P, de Lavigne C. An arthroscopic technique to treat the iliotibial band syndrome. *Knee Surg Sports Traumatol Arthrosc* 2009;**17**(3):233-6.
26. Nemeth WC, Sanders BL. The lateral synovial recess of the knee: anatomy and role in chronic Iliotibial band friction syndrome. *Arthroscopy* 1996;**12**(5):574-80.
27. Drogset JO, Rossvoll I, Grontvedt T. Surgical treatment of iliotibial band friction syndrome. A retrospective study of 45 patients. *Scand J Med Sci Sports* 1999;**9**(5):296-8.
28. Fredericson M, Weir A. Practical management of iliotibial band friction syndrome in runners. *Clin J Sport Med* 2006;**16**(3):261-8.
29. Martens M, Libbrecht P, Burssens A. Surgical treatment of the iliotibial band friction syndrome. *Am J Sports Med* 1989;**17**(5):651-4.
30. Ellis R, Hing W, Reid D. Iliotibial band friction syndrome – a systematic review. *Manual Ther* 2007;**12**(3):200-8.
31. Barber FA, Boothby MH, Troop RL. Z-plasty lengthening for iliotibial band friction syndrome. *J Knee Surg* 2007;**20**(4):281-4.
32. Noble CA, Ferguson MC, Johnson S. The role of surgery in the management of iliotibial friction syndrome in long-distance runners. *J Bone Joint Surg Br* 2003;**85-B**(Suppl 2):148.
33. Holmes JC, Pruitt AL, Whalen NJ. Iliotibial band syndrome in cyclists. *Am J Sports Med* 1993;**21**(3):419-24.
34. Maffulli N, Sharma P, Luscombe KL. Achilles tendinopathy: aetiology and management. *J R Soc Med* 2004;**97**(10):472-6.
35. Webborn AD. Novel approaches to tendinopathy. *Disabil Rehabil* 2008;**30**(20-22):1572-7.
36. Rompe JD, Furia JP, Maffulli N. Mid-portion Achilles tendinopathy – current options for treatment. *Disabil Rehabil* 2008;**30**(20-22):1666-76.
37. Longo UG, Ramamurthy C, Denaro V, Maffulli N. Minimally invasive stripping for chronic Achilles tendinopathy. *Disabil Rehabil* 2008;**30**(20-22):1709-13.
38. Paavola M, Kannus P, Orava S, Pasanen M, Jarvinen M. Surgical treatment for chronic Achilles tendinopathy: a prospective seven month follow up study. *Br J Sports Med* 2002;**36**(3):178-82.
39. Tan SC, Chan O. Achilles and patellar tendinopathy: current understanding of pathophysiology and management. *Disabil Rehabil* 2008;**30**(20-22):1608-15.
40. Ferretti A, Conteduca F, Camerucci E, Morelli F. Patellar tendinosis: a follow-up study of surgical treatment. *J Bone Joint Surg Am* 2002 Dec;**84-A**(12):2179-85.
41. Erasmus PJ, Dillon E. Patella tendinopathy. *S Afr Orthop J* 2005;**4**(3):80-4.
42. Warden SJ, Brukner P. Patellar tendinopathy. *Clin Sports Med* 2003;**22**(4):743-59.
43. Peers KH, Lysens RJ. Patellar tendinopathy in athletes: current diagnostic and therapeutic recommendations. *Sports Med* 2005;**35**(1):71-87.
44. Maffulli N, Kader D. Tendinopathy of the Achilles tendon. *J Bone Joint Surg Br* 2002;**84-B**:1-8.
45. Friedrich T, Schmidt W, Jungmichel D, Horn LC, Josten C. Histopathology in rabbit Achilles tendon after operative tenolysis (longitudinal fiber incisions). *Scand J Med Sci Sports* 2001;**11**(1):4-8.
46. Maffulli N, Testa V, Capasso G, Bifulco G, Binfield PM. Results of percutaneous longitudinal tenotomy for Achilles tendinopathy in middle- and long-distance runners. *Am J Sports Med* 1997 Nov-Dec;**25**(6):835-40.
47. Maffulli N, Binfield PM, Moore D, King JB. Surgical decompression of chronic central core lesions of the Achilles tendon. *Am J Sports Med* 1999;**27**(6):747-52.
48. Paavola M, Orava S, Leppilahti J, Kannus P, Jarvinen M. Chronic Achilles tendon overuse injury: complications after surgical treatment. An analysis of 432 consecutive patients. *Am J Sports Med* 2000;**28**(1):77-82.
49. Coleman BD, Khan KM, Kiss ZS, Bartlett J, Young DA, Wark JD. Open and arthroscopic patellar tenotomy for chronic patellar tendinopathy. A retrospective outcome study. Victorian Institute of Sport Tendon Study Group. *Am J Sports Med* 2000 Mar-Apr;**28**(2):183-90.
50. Glaser T, Poddar S, Tweed B, Webb CW. Clinical inquiries. What's the best way to treat Achilles tendinopathy? *J Fam Pract* 2008;**57**(4):261-3.
51. Bahr R, Fossan B, Loken S, Engebretsen L. Surgical treatment compared with eccentric training for patellar tendinopathy (Jumper's Knee). A randomized, controlled trial. *J Bone Joint Surg Am* 2006;**88**(8):1689-98.
52. Lorbach O, Diamantopoulos A, Paessler HH. Arthroscopic resection of the lower patellar pole in patients with chronic patellar tendinosis. *Arthroscopy* 2008;**24**(2):167-73.
53. Hyman GS. Jumper's knee in volleyball athletes: advancements in diagnosis and treatment. *Curr Sports Med Rep* 2008;**7**(5):296-302.
54. Peers KH, Lysens RJ, Brys P, Bellemans J. Cross-sectional outcome analysis of athletes with chronic patellar tendinopathy treated surgically and by extracorporeal shock wave therapy. *Clin J Sport Med* 2003;**13**(2):79-83.
55. Panni AS, Tartarone M, Maffulli N. Patellar tendinopathy in athletes. Outcome of nonoperative and operative management. *Am J Sports Med* 2000;**28**(3):392-7.
56. Panni AS, Biedert RM, Maffulli N, Tartarone M, Romanini E. Overuse injuries of the extensor mechanism in athletes. *Clin Sports Med* 2002 ix;**21**(3):483-98.
57. Kvist H, Kvist M. The operative treatment of chronic calcaneal paratenonitis. *J Bone Joint Surg Br* 1980 Aug;**62**(3):353-7.
58. Paavola M, Kannus P, Paakkala T, Pasanen M, Jarvinen M. Long-term prognosis of patients with achilles tendinopathy. An observational 8-year follow-up study. *Am J Sports Med* 2000 Sep-Oct;**28**(5):634-42.
59. Kvist M. Achilles tendon injuries in athletes. *Ann Chir Gynaecol* 1991;**80**(2):188-201.
60. Tallon C, Coleman BD, Khan KM, Maffulli N. Outcome of surgery for chronic Achilles tendinopathy. A critical review. *Am J Sports Med* 2001;**29**(3):315-20.
61. Pell RF,4th, Khanuja HS, Cooley GR. Leg pain in the running athlete. *J Am Acad Orthop Surg* 2004;**12**(6):396-404.

62. Yates B, Allen MJ, Barnes MR. Outcome of surgical treatment of medial tibial stress syndrome. *J Bone Joint Surg Am* 2003 Oct;**85-A**(10):1974-80.
63. Moen MH, Tol JL, Weir A, Steunebrink M, De Winter TC. Medial tibial stress syndrome: a critical review. *Sports Med* 2009;**39**(7):523-46.
64. Englund J. Chronic compartment syndrome: tips on recognizing and treating. *J Fam Pract* 2005 Nov;**54**(11):955-60.
65. Kortebein PM, Kaufman KR, Basford JR, Stuart MJ. Medial tibial stress syndrome. *Med Sci Sports Exerc* 2000;**32**(3 Suppl):S27-33.
66. Beck BR. Tibial stress injuries. An aetiological review for the purposes of guiding management. *Sports Med* 1998;**26**(4):265-79.
67. Holen KJ, Engebretsen L, Grontvedt T, Rossvoll I, Hammer S, Stoltz V. Surgical treatment of medial tibial stress syndrome (shin splint) by fasciotomy of the superficial posterior compartment of the leg. *Scand J Med Sci Sports* 1995;**5**(1):40-3.
68. Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. *Am J Sports Med* 1987;**15**(1):46-58.
69. Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. *J Am Acad OrthopSurg* 2000;**8**(6):344-53.
70. Young AJ, McAllister DR. Evaluation and treatment of tibial stress fractures. *Clin Sports Med* 2006 x;**25**(1):117-28.
71. Borens O, Sen MK, Huang RC, Richmond J, Kloen P, Jupiter JB, *et al*. Anterior tension band plating for anterior tibial stress fractures in high-performance female athletes: a report of 4 cases. *J Orthop Trauma* 2006;**20**(6):425-30.
72. Orava S, Karpakka J, Hulkko A, Vaananen K, Takala T, Kallinen M, *et al*. Diagnosis and treatment of stress fractures located at the mid-tibial shaft in athletes. *Int J Sports Med* 1991;**12**(4):419-22.
73. Chang PS, Harris RM. Intramedullary nailing for chronic tibial stress fractures. A review of five cases. *Am J Sports Med* 1996;**24**(5):688-92.
74. Miyamoto RG, Dhotar HS, Rose DJ, Egol K. Surgical treatment of refractory tibial stress fractures in elite dancers: a case series. *Am J Sports Med* 2009;**37**(6):1150-4.
75. Brukner P, Khan K. Clinical Sports Medicine. 2nd ed. New York: McGraw-Hill; 2000.
76. Delee J, Drez D, Miller M. Delee and Drez's Orthopaedic sports medicine: Principals and practice. 2nd ed. Philadelphia: Saunders; 2003.
77. Browner BD, Juipiter JB, Levine AM, Trafton PG, Krettek C. Skeletal Trauma: Basic Science, Management and Reconstruction. Philadelphia: Saunders; 2009.
78. Varner KE, Younas SA, Lintner DM, Marymont JV. Chronic anterior midtibial stress fractures in athletes treated with reamed intramedullary nailing. *Am J Sports Med* 2005;**33**(7):1071-6.
79. Detmer DE, Sharpe K, Sufit RL, Girdley FM. Chronic compartment syndrome: diagnosis, management, and outcomes. *Am J Sports Med* 1985;**13**(3):162-70.
80. Turnipseed WD. Diagnosis and management of chronic compartment syndrome. *Surgery* 2002 discussion 617-9; Oct;**132**(4):613-7.
81. Styf J, Korner L, Suurkula M. Intramuscular pressure and muscle blood flow during exercise in chronic compartment syndrome. *J Bone Joint Surg Br* 1987;**69**(2):301-5.
82. Pedowitz RA, Hargens AR, Mubarak SJ, Gershuni DH. Modified criteria for the objective diagnosis of chronic compartment syndrome of the leg. *Am J Sports Med* 1990;**18**(1):35-40.
83. Shah SN, Miller BS, Kuhn JE. Chronic exertional compartment syndrome. *Am J Orthop* 2004;**33**(7):335-41.
84. Mubarak SJ, Owen CA. Double-incision fasciotomy of the leg for decompression in compartment syndromes. *J Bone Joint Surg Am* 1977;**59**(2):184-7.
85. Schepsis AA, Gill SS, Foster TA. Fasciotomy for exertional anterior compartment syndrome: is lateral compartment release necessary? *Am J Sports Med* 1999;**27**(4):430-5.
86. Micheli LJ, Solomon R, Solomon J, Plasschaert VF, Mitchell R. Surgical treatment for chronic lower-leg compartment syndrome in young female athletes. *Am J Sports Med* 1999;**27**(2):197-201.