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## SHIN SPLINTS — A LITERATURE REVIEW

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### ABSTRACT

"Shin splints" is not a specific diagnosis. It is merely a descriptive term that describes chronic exertional shin pain in an athlete. The evidence seems clear that shin splint pain has many different causes and this reflects the variation in the anatomy. It would be preferable to describe shin splint pain by location and aetiology, for example, lower medial tibial pain due to periostitis or upper lateral tibial pain due to elevated compartment pressure. This would aid communication between physicians and also direct therapy more accurately.

Key words: Shin splints, Medial tibial syndrome, Compartment syndrome, Medial tibial stress syndrome.

### INTRODUCTION

The fitness boom is undoubtedly a child of the 60's. To its credit, it has grown up to become a mature, young adult, and has certainly gone beyond what may be called fadism. As many as 10 million Americans run regularly, many more than 8 kilometres a day (Medical News, 1978). It hardly needs to be added that there has been an associated increase in athletic injuries. Indeed, it would appear that most athletes are more limited by their "musculoskeletal max" than their "VO<sub>2</sub> max".

This dissertation concerns itself with a critical examination of the literature concerning one particular sports injury — shin splints.

### DEFINITION

What exactly are shin splints? In 1968 the American Medical Association (AMA) published the "Standard Definition of Athletic Injuries" after canvassing the opinion of hundreds of physicians, trainers and physical educators. They defined shin splints as "pain and discomfort in the leg from repetitive running on hard surfaces or forcible extensive use of flexors . . . The diagnosis should be limited to musculotendinous inflammation, excluding a stress fracture or ischaemic disorder". Yet this definition is widely criticised. Detmar (1980) states: "The limitation (of the AMA definition) is not sensible since in a busy clinical practice, there are numerous cases in which one cannot localise the tenderness exclusively as musculotendinous". Several authors consider that shin splint pain comes from a bony reaction to stress (Devas, 1958; Mubarak et al, 1982), whilst others have considered it may come from elevated pressure within muscle compartments (Puranen, 1974; Subotnik, 1976). Both these views contradict the AMA definition.

The term is, in effect, a waste-basket term (Sperryn, 1984) that has been used to describe any pain between the tibial tuberosity and the ankle (Jackson, 1978). In an effort to make

a more specific description of chronic shin pain in athletes, various authors have used other terms. These include:

1. Medial tibial syndrome (Puranen, 1974):
2. Medial tibial stress syndrome (Dr. D. Drez, personal communication to Mubarak, 1982):
3. Shin soreness (Devas, 1958):
4. Anterior compartment syndrome (various authors):
5. Posteromedial compartment syndrome (various authors).

### LOCATION

There is no "usual" location of shin splints. Jackson (1978) comments: "There is no agreement in the literature . . . whether the pain associated with shin splints is more common in the proximal, medial or distal third of the tibia, or in the posterior, anterior, medial or lateral aspect of the leg".

Many studies have concentrated on chronic medial sided shin pain (Devas, 1958; Nutig, 1981; Puranen, 1974; Puranen and Alavaikko, 1981), others on chronic anteriolateral shin pain (Reneman, 1975) and some on both (Orava and Puranen, 1979; Wallensten, 1983) (see Fig. 1).

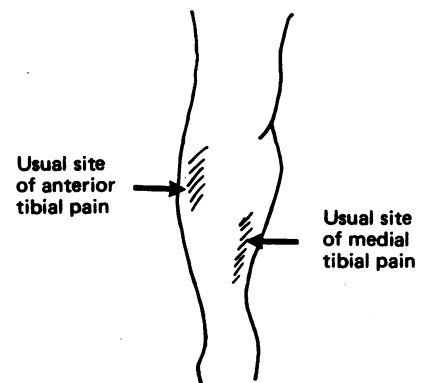


Fig. 1

In practical terms, the term shin splints is used to describe an exertional pain occurring in athletes' shins. It obviously does not refer to certain well-defined pathological entities, such as tumour, sepsis, deep venous thrombosis, etc., but gives no indication of the location or aetiology of the pain.

## SYMPTOMS

The predominant symptom is pain felt on or around the tibia. It is felt on exertion, initially towards the end of a run; but if more severe, can occur earlier and earlier during exercise (Mubarak et al, 1982).

Extremely severe cases may have pain on walking or even at rest (Puranen, 1974). Medial sided tibial pain seems usually to be described as a soreness or a dull aching pain (Slocum, 1967) whereas lateral sided pain may be more aching and cramping in quality but this is extremely variable (Martens et al, 1984; Matsen et al, 1980). Pain is usually well localised but may radiate down the foot or be associated with dysaesthesias (Rorabeck et al, 1983). In these cases, the pain is probably due to neural compression by elevated compartment pressure but this is increasingly being regarded as a cause of shin splint pain (Puranen, 1974; Nutig, 1981).

## SIGNS

In general, there are few signs associated with the condition. Most of the patients have tenderness at the site of pain. Slight oedema is sometimes noted (Devas, 1958) as is thickening of the subcutaneous border of the tibia. Highly developed musculature may be seen (Mavor, 1956) and the muscle compartments may feel tense, or have muscle herniae (Martens et al, 1984). Neurological symptoms are sometimes reported and occasionally there may be weakness and pain on passive stretch of the muscles running through the compartment (Matsen et al, 1980). Vascular disturbances are rare, even in cases when the shin splint is due to elevated compartment pressures (Mavor, 1956; Puranen et al, 1981). Tibial stress fractures usually exhibit well localised tenderness and pain, often with palpable callus (Devas, 1958). These are most commonly found at the lower and upper margins of the tibia (Sperry, 1984). "Springing" the tibia (straining it against a fulcrum) may reproduce the pain in a stress fracture (Devas, 1958).

## INCIDENCE

Shin splints account for about 10-15% of all running injuries (Gudas, 1980; James et al, 1978) and up to 60% of all lesions causing pain in athletes' legs (Orava and Puranen, 1979). Andrich et al (1974) in the only prospective study relating to shin splints reported an incidence of 4.07% after over 2,000 recruits underwent basic physical education training.

## ANATOMY

The pathophysiology of shin splints is more easily understood after examining the relevant cross-sectional anatomy.

There are 4 muscle compartments in the leg (Grant, 1972):

- Anterior: this compartment contains the tibialis anterior muscle, the extensor hallucis longus, the extensor digitorum longus and the peroneus tertius. The tibialis anterior dorsiflexes the ankle and inverts the foot. The extensor hallucis longus extends the great toe, the extensor digitorum longus extends the other toes and assists in eversion as does the peroneus tertius.
- Deep posterior: this contains the flexor digitorum longus, the tibialis posterior and the flexor hallucis longus. The tibialis posterior plantar flexes and inverts the foot. The others are predominantly toe flexors.
- Superficial posterior: this is the gastrocnemius and soleus group; predominantly plantar flexors of the ankle.
- Lateral: this compartment contains the peroneus brevis and longus, mainly foot evertors (see Fig. 2).

## PATHOLOGICAL AND AETIOLOGICAL FACTORS

### 1. Overstress to Bone

Shin splint pain may be caused by a bony reaction to the stress of overuse. An explanation of the pathology of the bony stress

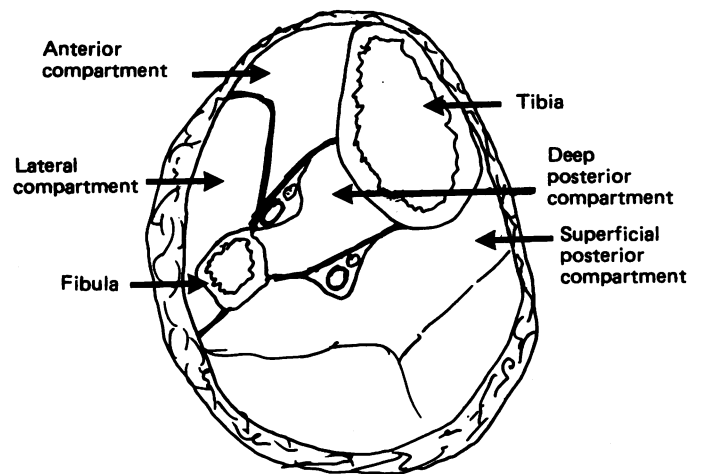


Fig. 2: The cross-sectional anatomy of the leg, with the fascial outlines marked.

reaction explains how this may occur (Devas, 1975).

Bone reacts to stress by remodelling itself in an attempt to become stronger. This involves, in essence, removal of the part of the bone matrix that was not strong enough (the apatite crystal) and replacing it with bone matrix strong enough to cope with the increased demands on it. The bone may successfully adapt to the increased stress. This would explain the tibial cortical hypertrophy sometimes seen in distance runners, especially the young athlete (Jackson, 1978). Alternatively, if the athlete continues to stress a weakened bone, it may fracture — hence stress fracture. Between these extremes, athletes may show a variety of features including periostitis, cortical demineralisation, cortical hypertrophy or mixed patterns (Devas, 1958; Jackson, 1978). There is thus a whole spectrum of bony reactions to stress and athletes may combine features of each.

It is thought that muscle pull rather than impact may cause the bony stress reaction (Devas, 1958). Devas believed that strong calf muscles may stress the tibia, much the same way as a taut bow string would stress a bow. But the exact mechanism is unclear, as tibial stress fractures commonly occur in various tibial sites depending on the type of activity.\*

### 2. Compartment Syndromes

Several authors have demonstrated that elevated muscle compartment pressure in athletes may cause either medial sided shin pain (Puranen et al, 1981; Wallensten, 1983) or lateral sided shin pain (Rorabeck et al, 1983) but this is highly contentious (D'Ambrosia et al, 1977; Mubarak et al, 1982).

The pathology of the compartment syndrome is that with exercise a muscle retains fluid and increases bulk. As the muscles are bound by their fascial compartments, the increased pressure begins to block off intramuscular blood vessels and causes ischaemia of exertion. Increased pressure may also compress nerves to cause referred distal symptoms but rarely will cause diminished distal pulses in a healthy blood vessel (Paton, 1968; Puranen, 1974).

\*Devas found in his series of athletes that most tibial stress fractures occurred in the distal third of the tibia. On reviewing world literature, he found most recruits had stress fracture in the upper third of the tibia, whereas ballerinas tended to have stress fracture in the middle. Orava and Puranen (1979) also noted that stress fracture occurred most often in the distal and proximal tibia.

In the mid-1970's, various techniques were developed to measure the tissue pressures inside muscle compartments. These included the needle manometer technique (Whitesides and Harvey, 1975), the infusion technique (Matson et al, 1976), the wick catheter (Scholander et al, 1968; Mubarak et al, 1976), and the slit catheter (Rorabeck et al, 1980). All these techniques involve placing a catheter inside a muscle compartment and measuring pressure with a transducer.

Puranen had previously (1974) successfully treated athletes with medial tibial pain by fasciotomy. In 1981 he repeated his earlier work using pressure studies (Puranen et al, 1981). In a well designed study, he took 22 patients (17 athletes) with medial tibial pain; and 10 patients (2 athletes) with antero-lateral pain. All had exertional pain, and conservative treatment and investigations were negative. For controls, he used 5 healthy subjects and the asymptomatic leg. Essentially, he found that:

1. The symptomatic leg had similar resting pressures to the control leg,
2. During exercise, the symptomatic leg had higher pressures than the control (73 mmHg vs. 23 mmHg for medial side, 100 vs. 28 for lateral side);
3. The usual "pain threshold" pressure was 50 mmHg.

All underwent fasciotomy of the relevant compartment. Post-operatively, 5 patients remained symptomatic, so they were restudied and found to have pressures greater than 50 mmHg. The first fasciotomy was therefore considered inadequate, and after re-operation all became asymptomatic. One criticism of this study is that there is no indication of length of follow-up, nevertheless, Puranen could justifiably conclude: "Pain on exertion on the inner border of the leg is a common problem in runners. We believe it results from ischaemia to the medial compartment".

Others have disputed these results (Mubarak et al, 1982). Mubarak studied 12 athletes with typical medial tibial pain, and concluded that the increase in pressure he had seen with exercise was insufficient to cause a compartment syndrome. He concluded that the syndrome was due to periostitis.

His methodology is however, open to criticism. He had no direct controls, and based his conclusions on a comparison between a symptomatic anterior compartment and a symptomatic posterior compartment. His "control" values came from asymptomatic anterior compartments, yet intuitively it seems reasonable to suppose that anterior and posterior compartments may have different pressures and in fact, this has been experimentally demonstrated (Puranen et al, 1981; Wallensten, 1983). D'Ambrosia had previously (1977) also found that shin splint patients did not have an increase in tissue pressure, but as he measured his subjects at rest, and only measured 3 posterior compartments, his results have limited value.

It is generally accepted though, that elevated anterior compartment pressures can cause anterior shin splint pain (Rorabeck et al, 1983). Mavor (1956) describes one of the earliest cases of exertional anterior tibial pain successfully treated by fasciotomy.

Freedman (1953) gives a fascinating history of the symptoms of a medical officer on the last journey of Captain Scott, Dr. Edward Wilson, who undoubtedly had an exertional anterior compartment syndrome. Paton (1968) describes the case of a man who developed anterior shin pain after an anterior compartment fascial defect was closed. Obviously, the defect was the only valve through which pressure could escape. Martens (1984) got good results after operating on a mixture of patients with elevated anterior and posterior pressures.

Wallensten (1983) had interesting results from a well designed study. He operated on 8 patients, all with anterior

pain. Post-operatively, all had a decrease in pressure and all were cured. Of 9 patients he operated on with medial tibial pain, only 5 were cured, but there was no significant decrease in tissue pressures! Wallensten could not understand how these 5 patients could have been cured when there was no decrease in pressures, but he postulated that:

1. The periosteum may have been denervated at operation or;
2. By cutting the fascia, he had stopped fascial pull on the periosteum. (Action of the posterior muscles may cause associated fascial pull.)

This may explain the dichotomy of opinion that exists about the value of fasciotomy in the treatment of shin splints, but as the numbers in Wallensten's study were small, further study is needed.

It may be concluded that the body of evidence supports the idea that increased compartment pressure may cause shin splint pain, but not all shin splint pain is due to increased compartment pressure.

### 3. Biomechanical Factors

Biomechanical factors are widely considered to play a role in the origin of shin splints (Clement, 1974; Sheehan, 1977; Subotnik, 1976). This is not surprising when one considers that a long distance runner's feet hit the ground more than 5,000 times per hour so that even a small biomechanical abnormality is magnified (Medical News, 1978).

To understand how biomechanics plays a role in the development to shin splints, it is necessary to explain briefly the biomechanics of running and in particular, the role of the foot.

Running is a series of smoothly co-ordinated strides (Slocum and James, 1968) that are divided into a support phase (weight-bearing) and a swing phase (non-weight-bearing). During the support phase, the foot occupies 3 distinct positions:

	% of ground contact time
1 Initial foot contact (contact period)	27
2 Mid foot contact (mid stance)	40
3 Push off	33
	100%

When the foot hits the ground, it is in the supinated position which is stiff and rigid. On ground strike, the foot must do two things. First, it must adapt to the surface it has landed on; secondly, it must dissipate some of the impact of the shock. Thus, it must change from a high arched stiff lever (supination) to a low arched, flexible bag of bones (pronation). The pronated position thus gives maximal flexibility and shock absorption. The pronated position is maintained throughout contact and mid-stance; but at push off, the foot must once again become a stiff lever which it does by moving into supination.

How the foot actually achieves these changes is extremely complex, but one can summarise by saying that the pronated position is one of eversion, abduction and dorsiflexion, and supination is a combination of inversion, adduction and plantar flexion (Langer, 1984). Hence, these are called triplane movements. The muscles of the leg are intimately involved in these movements and thus it is easy to see that any abnormality of foot motion could cause pathology in the shins where those muscles attach.

When a runner's foot hits the ground, it is inverted and dorsiflexed. The forefoot must be slowly let to the ground, otherwise it would slap (Subotnik, 1976). Consequently, the muscles of the anterior compartment contract eccentrically, to let the forefoot slowly to the ground. The heel then moves

from inversion to eversion. As this must be a controlled movement, the invertors (primarily the anterior and posterior tibial muscles) eccentrically contract to control the rate and degree of eversion. If a runner runs on hard surfaces, especially if he over-strides, then he will need more forceful contraction from his anterior group to make a controlled descent of his forefoot (Subotnik, 1976). It follows that these muscles may hypertrophy, to meet the increased demands and hence cause an exertional compartment syndrome. Alternatively, they could strain at their site of origin onto bone, causing a tendonitis or a periostitis.

If the runner has a forefoot varus (see Fig. 3), then the muscle that lets the great toe hit the ground (the extensor hallucis longus) is also under strain and it may hypertrophy or tear. Evidence of this can be seen clinically from runners who complain of anterior tibial shin splints following emergence of blisters on the medial plantar aspect of the foot (Subotnik, 1976).

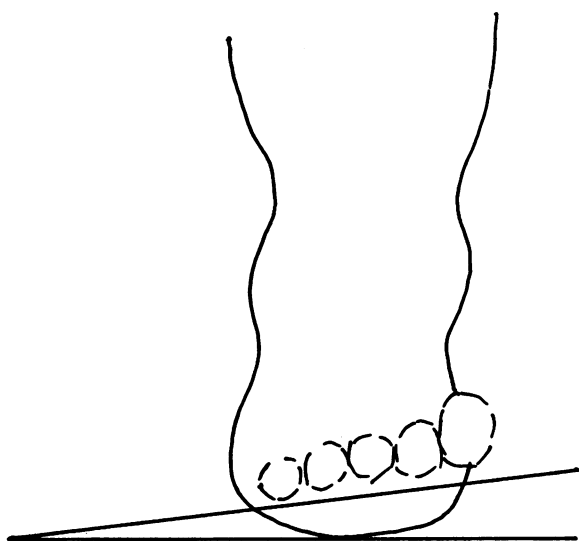


Fig. 3: The forefoot is inverted with respect to the rearfoot.

Overpronation will strain those muscles that control pronation, and these are primarily the anterior and posterior tibial muscles. Similarly, these may hypertrophy to cause elevated compartment pressure, or strain to cause periostitis, tendonitis or myositis. Thus, overpronation can be a factor in anterior or medial shin splint pain as has been frequently noted (Delacerda, 1980; Subotnik, 1976; Taunton et al, 1981).

Certain anatomical variations make overpronation more likely. These include:

1. Tibial varum because the foot hits the ground in an exaggerated, inverted position, therefore must "roll over" further to make adequate ground contact;
2. Forefoot varus because the rearfoot must "roll over" further (pronate) to compensate for the inverted forefoot;
3. Internal femoral torsion (Root et al, 1977) because the foot may pronate to increase abduction and therefore walk straighter.

Other causes of overpronation include; plantarflexed fifth metatarsal, limb length inequality, various neuromuscular conditions and tibial torsion (Root et al, 1977).

Overpronation is thus a well documented cause of shin splints. Several other authors have also found other biomechanical data to be statistically significant in relation to shin splints (Gehlsen and Seger, 1980; Lilletvedt et al, 1979).

To summarise, biomechanical studies indicate why certain athletes are predisposed to get shin splints and may explain the pathophysiology behind elevated compartment pressure or musculotendinous tears in the aetiology of shin splints.

#### 4. Muscle Fatigue in Shin Splints

It is believed that an overused, fatigued muscle can cause or exacerbate the condition of shin splints. Certainly it is widely held that shin splints are most frequently seen in the under-conditioned athlete trying to do too much too soon (Clement, 1974; Jackson, 1978).

Clement (1974) noted that earlier scientific work had suggested that muscles were able to act as "shock absorbers" and therefore absorb some of the stress of movement that would otherwise go direct to bone. He therefore suggested that the athlete that asked his body to do too much too soon would fatigue his muscles and may decrease the shock absorption function of the muscle. Consequently, all stress would be transmitted direct to bone; hence bone overload and periostitis. Taunton et al (1981) supported this; and pointed out that biomechanical abnormality would make muscle overstress occur earlier.

#### INVESTIGATIONS

Particularly in regard to evaluating the patient with shin splints, a good history and examination are paramount. In many cases, this alone will help distinguish between the various causes of shin splints. However, investigations may be helpful to distinguish the exact cause in many cases.

##### 1. Radiology

A lateral and anteroposterior view may show stress fracture (Devas, 1958). The earliest sign of stress fracture is slight loss of bone density (Devas, 1958) followed either by a fracture line or callus formation. The changes may take weeks or months to appear (Devas, 1969), and as they usually involve only one cortex, may be difficult to see (Detmar, 1980). X-ray studies may show other conditions masquerading as shin splints, such as tumour or sepsis.

##### 2. Bone Scan

There has been much recent work published about bone scan findings in shin splints (Brill, 1983; Lieberman and Hemingway, 1980; Spencer et al, 1979). The results from bone scans may be extremely variable:

- (a) Stress fracture: This generally shows up as a localised "hot spot" on the scan;
- (b) Diffuse cortical hypertrophy: This is usually seen as a diffuse periosteal uptake. It has been called the "double stripe sign" by Lieberman and Hemingway, 1980, presumably due to a summation of the density of the radiopharmaceutical when seen in longitudinal section.

It is worthwhile emphasising that bony pain may precede positive bone scan findings. Milgrom et al (1984) report on 3 cases of stress fracture diagnosed on a second bone scan after the first scan was negative. These patients had a history of typical stress fracture pain for between 2 to 5 weeks prior to scan. All initial scans were negative and all returned to active military duties. Because of continuing pain, they were re-scanned after one month and all had stress fractures. However, it is of course possible that these patients only actually completed their fracture after resuming activity.

##### 3. Pressure Studies

This has been fully discussed previously. Typical features which may alert the physician of the need for pressure studies include neural symptoms, anterior tibial pain, pain persisting after exercise, a description of "cramping" type pain and the failure of symptoms to resolve after rest.

#### 4. Ultrasound

Use of ultrasound in the diagnosis of chronic tibial pain is purely experimental. Gershuni et al (1982) measured the cross-sectional width of the anterior compartment before and after exercise. He found that for 9 non-joggers, the width increased by 7.4%; and for 10 joggers it increased by 10.1%.

#### DIFFERENTIAL DIAGNOSIS OF SHIN PAIN

This includes (Sperryn, 1984):

1. Stress fractures: Usually a history of worsening pain which eventually spills over into rest time;
2. Vascular disease, especially in the older jogger. Diabetes may be an exacerbating factor;
3. Spinal stenosis: This would include tumour, disc herniation, arthritis and vertebral infection;
4. Fascial hernias;
5. Tenosynovitis;
6. Cellulitis;
7. Deep vein thrombosis;
8. Infective or varicose periostitis;
9. Tumour;
10. Popliteal artery entrapment syndromes;
11. Entrapment of superficial peroneal nerve;
12. "Shin Splints", which arises from multiple pathologies, and includes
  - (a) Musculotendinous tear;
  - (b) Musculotendinous inflammation;
  - (c) Musculofascial pull;
  - (d) Periostitis;
  - (e) Tibial cortical hypertrophy;
  - (f) Stress fracture;
  - (g) Elevated compartment pressure.

What causes one athlete to get shin splints and another to be spared is unclear. Anatomical variation leading to biomechanical abnormality is probably a major cause, as are training errors.

#### TREATMENT OF SHIN SPLINTS

The appropriate treatment depends on the aetiology of the shin splint:

##### 1. Prevention

Training errors are commonly thought to provoke shin splints. These may include wearing inappropriate shoes, running on hard surfaces and increasing mileage too rapidly. All these increase muscle fatigue and prevent the natural body reactions to stress from taking place. Stretching and strengthening of the leg muscles also helps to prevent muscular strain (Sperryn, 1984). Andrish et al (1974) looked at a group of over 2,000 army recruits undergoing physical education training. He tried various prophylactic regimens, including heel pads, heel cord stretching and a 2-week graduated running programme. He found none of these gave any prophylaxis for shin splints, but there was a highly significant negative correlation ( $p < 0.001$ ) for those recruits who were physically active before undergoing the physical education programme. This suggests that to develop adequate musculoskeletal strength, one needs a long-term build up rather than a short-term (2 weeks in this study).

##### 2. Rest

Rest is common to all treatment regimens. Rest allows time for healing and time for inflammation to settle down. It is essential though after rest gradually to re-introduce running and avoid any training errors that may have provoked the syndrome initially. It is advisable for the athlete to maintain cardiovascular fitness during rest, for example with swimming or cycling.

##### 3. Ice and Non-steroid Anti-inflammatory Drugs

These are also widely advised. They are thought to help by

decreasing inflammatory reaction and also by analgesia. Ice before and after running is also recommended to control pain (Sperryn, 1984).

##### 4. Muscle Stretching and Strengthening

Muscle strengthening is believed to help prevent the effects of fatigue on bone and muscle (Clement, 1974). This usually consists of progressive exercises to the dorsi and plantar flexors of the foot. Stretching may also increase muscle shock absorption.

##### 5. Correction of Biomechanical Defects

The device used will obviously depend on the biomechanical abnormality detected. The most common to be treated is over-pronation. These are usually treated with an orthotic device, which is an artificial prosthesis that supports the antipronation muscles. It is usually a wedge, built up on the medial side of the heel. Other biomechanical abnormalities such as leg length discrepancy or forefoot varus can be treated by an appropriate orthotic.

6. Injection of the medial tibia border with corticosteroids has been tried by some, but has very limited success.

#### SURGERY

In recalcitrant cases, or cases where increased compartment pressures are thought to be a factor, then operative therapy would be considered. In most cases, it would be worthwhile to try conservative measures first, and keep operation as a final option. Certain clinical features (e.g. neurological symptoms, persisting pain or failure of conservative treatment) may suggest increased compartment pressure as the diagnosis. It is worthwhile remembering too, that the reasons for success of operation are by no means clearly understood or guaranteed and the merits of each case need to be judged individually.

Much of the research is of limited value because of small subject numbers and also a tendency to focus on one cause of shin splints to the exclusion of all others. The ideal trial would include biomechanical assessment, measurement of tissue compartment pressures, radiological and radionuclide investigations and a follow-up period of one to two years! The obvious difficulties of setting up such a study make understandable any reluctance in doing so.

Meanwhile, an appreciation of the diversities of the condition may assist in management of a difficult syndrome.

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#### British Association of National Coaches — "Coach of the Year"

BANC are offering seven awards to coaches of various categories, team and individual events in any sport. Nomination forms and full particulars are available from: Gill Garside, Darwall Smith Associates Ltd., Smoke House Yard, 44-46 St. John Street, LONDON EC1M 4DT, or from the National Coaching Foundation, 4 Cottage Close, Becketts Park, LEEDS LS6 3QH (who have also developed video cassettes on safety and injury in sport).

Coaches can be nominated by Governing Bodies, Colleges, etc. and BANC members. The scheme is sponsored by the Best Foods Division of CBC (UK) Ltd. Closing date for nominations is 28th February, 1986.

#### Gravity-inversion Boots

In 1983 Vol. 17, No. 4, p. 154 we published a warning from Mr. R. Goldman of the Chicago Osteopathic Medical Center warning of possible hazards of treating lower back pain by head-down suspension. It was suspected that raised cerebral and intra-ocular pressures might be dangerous. The author has now written to say that these fears were without foundation, if used by healthy people, and they continue some activity while suspended, and do not hang for a long period immobile.

#### Eighth Windsor Colloquium

The Eighth Windsor Colloquium on Clinical Problems Peculiar to Sport will be held at the Postgraduate Centre, The King Edward VII Hospital, Windsor, on 14th and 15th November next.

The Colloquium will take its normal format and will include a Clinico-Pathological Conference at lunch time on

Friday. It will also include, on the Thursday, a Symposium on Alternative Therapies in Sports Medicine.

The draft Programme will be circulated in the Summer. In the meanwhile please regard this as a call for contributions particularly in the section "What's New". We look forward to short presentations on recent developments in Sports Medicine and I would be delighted to hear from anybody who wishes to contribute.

#### 23rd World Congress of the Federation Internationale de Medecine Sportive 22nd-26th September, 1986

This event, held every four years, will be taking place in Brisbane, Australia. An international coaching conference precedes it (20th-21st) and a conference on Health and Fitness follows (27th-28th). Topics to be included in the main congress are: Health through sporting activities; elite performers; the injured athlete; fitness assessment. There is an excellent social programme for non-participants, and other activities for all delegates.

The full programme is yet to be published, and those interested should write to:— The Organising Committee, 23rd FIMS World Congress, PO Box 439, FORTITUDE VALLEY, Queensland 4064, Australia.

We hope the UK will again send a reasonably large delegation, and that papers of merit be submitted for the scientific programme.

*(If Queensland looks after us as well as did Victoria in the 1974 World Congress in Melbourne, it should be an event well worth the long distance needed to travel from Europe and the Americas — Editor)*