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Medial Tibial Stress Syndrome
A Critical Review

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Contents

Abstract

Medial tibial stress syndrome (MTSS) is one of the most common leg injuries in athletes and soldiers. The incidence of MTSS is reported as being between 4% and 35% in military personnel and athletes. The name given to this condition refers to pain on the posteromedial tibial border during exercise, with pain on palpation of the tibia over a length of at least 5 cm. Histological studies fail to provide evidence that MTSS is caused by periostitis as a result of traction. It is caused by bony resorption that outpaces bone

formation of the tibial cortex. Evidence for this overloaded adaptation of the cortex is found in several studies describing MTSS findings on bone scan, magnetic resonance imaging (MRI), high-resolution computed tomography (CT) scan and dual energy x-ray absorptiometry.

The diagnosis is made based on physical examination, although only one study has been conducted on this subject. Additional imaging such as bone, CT and MRI scans has been well studied but is of limited value. The prevalence of abnormal findings in asymptomatic subjects means that results should be interpreted with caution.

Excessive pronation of the foot while standing and female sex were found to be intrinsic risk factors in multiple prospective studies. Other intrinsic risk factors found in single prospective studies are higher body mass index, greater internal and external ranges of hip motion, and calf girth. Previous history of MTSS was shown to be an extrinsic risk factor.

The treatment of MTSS has been examined in three randomized controlled studies. In these studies rest is equal to any intervention. The use of neoprene or semi-rigid orthotics may help prevent MTSS, as evidenced by two large prospective studies.

Medial tibial stress syndrome (MTSS) is one of the most common causes of exercise-induced leg pain.[1] Incidences varying from 4% to 35% are reported, with both extremes being derived from military studies.[2-4] This condition is most frequent among military personnel, runners and athletes involved in jumping, such as basketball players and rhythmic gymnasts.[5,6]

There is much controversy about the definition and terminology of this condition. Different authors have used different names, such as 'shin soreness',[7] 'tibial stress syndrome',[8] 'medial tibial syndrome',[9] 'medial tibial stress syndrome',[10] 'shin splints syndrome'[11] and 'shin splints'.[12] In this review we chose to use 'medial tibial stress syndrome' because, in our opinion, this best reflects the aetiology of the syndrome.

MTSS is characterized by exercise-related pain on the posteromedial side of the mid- to distal tibia. In 1966 the American Medical Association defined the condition (then termed shin splints) as: ''pain or discomfort in the leg from repetitive running on hard surfaces or forcible excessive use of the foot flexors; diagnosis should be limited to musculotendinous inflammations, excluding fracture or ischaemic disorder.''[13] This definition is the only available official definition given in the literature, but in our opinion is outdated and was never well accepted among clinicians. It does not describe signs on physical examination. Frequently when in the (older) literature the term 'shin splints' is used, 'medial tibial stress syndrome' is meant.

More recently, an updated and better definition was proposed by Yates and White.[4] They described MTSS as ''pain along the posteromedial border of the tibia that occurs during exercise, excluding pain from ischaemic origin or signs of stress fracture.'' Additionally, they stated that on palpation with physical examination, a diffuse painful area over a length of at least 5 cm should be present. However, since no official definition exists, many authors use their own definition of MTSS. This makes comparison between studies difficult.

Before diagnosing MTSS, the diagnosis of tibial stress fracture and exertional compartment syndrome should be excluded (see section 4).

Detmer[14] in 1986 developed a classification system to subdivide MTSS into three types: (i) type I – tibial microfracture, bone stress reaction or cortical fracture; (ii) type II – periostalgia from chronic avulsion of the periosteum at the periosteal-fascial junction; and (iii) type III – chronic compartment syndrome. In the recent literature, stress fracture and compartment syndrome are qualified as separate entities.

The objective of this review is to provide a critical analysis of the existing literature on MTSS. Aetiology, biomechanics, histology, patient

evaluation, diagnostic imaging, risk factors, therapy and prevention are discussed.

1. Methods

1.1 Literature Search

The electronic databases MEDLINE (1966–2009), EMBASE (1980–2009), CINAHL (1982–2009), SPORTDiscus (1975–2009) and Cochrane Library were searched for articles. The search terms 'shin splints', 'medial tibial syndrome', 'medial tibial stress syndrome' and 'tibial stress syndrome' were used with no restrictions for language. The references from the articles were screened and in this way additional articles were obtained.

Using the search terms, 382 possible titles were screened. Of these, 334 were not relevant as they discussed sports injuries in general, stress fractures, compartment syndromes or other topics. The 48 relevant titles were screened for related titles in the references. In total, 110 references were found, of which 104 articles could be obtained.

Articles were judged using the Institute for Quality of Healthcare (CBO [Centraal Begeleidings Orgaan]) classification system[15] (table I) and methodological quality and level of evidence were assessed. Methodological quality status (A1, A2, B, C, D) and level of evidence status (1, 2, 3, 4) were assessed (see tables II and III). The assessment was done independently by two researchers (MM and MS). If methodological quality and level of evidence were scored differently, a third author (AW) made the final decision (on two occasions).

Randomized controlled studies on the prevention and treatment of MTSS were also assessed using the Delphi scoring list[39] (table IV and V). This is a list of criteria for quality assessment of randomized clinical trials when conducting systematic reviews. This list contains nine points and each was scored as being present or not. The maximal score for the Delphi list is nine points.

Table I. Assessment of methodological quality and level of evidence (reproduced from Institute for Quality and Healthcare, the Netherlands,[15] with permission)

Assessment of methodological quality of studies concerning intervention (treatment/prevention)
A1: Systematic review of at least two independently conducted studies of A2 level
A2: Randomized double-blind clinical comparing study of good quality and size
B: Randomized clinical study, with moderate quality and size, or other comparing research (case-control study, cohort study)
C: Case series
D: Expert opinion
Assessment of methodological quality of studies concerning imaging and aetiology
A1: Systematic review of at least two independently conducted studies of A2 level
Imaging
A2: Research comparing against a gold standard/reference test, with an adequate number of participants
B: Research comparing against a gold standard/reference test, with an inadequate number of participants
Aetiology
A2: Prospective research with adequate and non-selective follow-up, with control for confounding
B: Prospective research with not all criteria mentioned under A2, or retrospective research
Imaging and aetiology
C: Case series
D: Expert opinion
Level of evidence
1: One systematic review (A1) or at least two independently conducted studies of A2 level (strong evidence)
2: One study of A2 level, or at least two independently conducted studies of B level (moderate evidence)
3: One study of B or C level (limited evidence)
4: Expert opinion (no evidence)

2. Aetiology

2.1 Functional Anatomy

There is much controversy about the anatomical basis for MTSS. Post-mortem studies have been performed to examine the relationship between the location of the pain and the anatomical structures. In these studies the distal attachments of different leg muscles were compared with the site of symptoms in MTSS.

Michael and Holder[49] dissected 14 specimens and found fibres of the soleus muscle but not the posterior tibialis muscle on the posteromedial tibial border. Saxena et al.[50] dissected ten cadavers and found that the distal attachment of the tibialis posterior muscle was 7.5 cm proximal to the medial malleolus. He concluded from this that the tibialis posterior muscle caused MTSS.

Table II. Study characteristics and quality scores of studies involving imaging

Study (year)	Study design	Inclusion criteria	Imaging type	No. of subjects	Population/type of activity	Outcome	Methodological quality	Level of evidence
Holder and Michael[16] (1984)	Prospective cohort	Pain on palpation of middle and distal posteromedial tibial border	Bone scan	10	Athletes, 50% M, 50% F; 6 running, 2 hockey, 1 ballet, 1 basketball, 16–31 y	9 scans abnormal uptake, 1 normal	B	2
Chisin et al.[17] (1987)	Prospective cohort	Not clearly stated	Bone scan	171 scanned with suspicion of stress fracture	Male soldiers, 18–21 y	171 bone scans: 53% sharply defined abnormality, stress fracture, 35% irregular poorly defined uptake, 12% normal	B	2
Batt et al.[18] (1998)	Prospective cohort	Exercise-induced lower leg pain, pain on palpation >5 cm on posteromedial tibial border	MRI/bone scan/x-ray	23: 41 symptomatic tibias, 4 asymptomatic athletes	Athletes and students, 14–58 y; 48% F, 52% M	x-Ray: 9% periosteal elevation; bone scan: 88% tibias abnormal; MRI: 83% abnormal	B	2
Gaeta et al.[19] (2005)	Case control	Lower leg pain <1 month; x-ray normal, clinical exam not stated	MRI/bone scan/CT scan	42: 50 tibias; 10 asymptomatic	Recreational and competitive athletes, 16–37 y; 38% F, 62% M	MRI: 88% abnormal; CT: 42% abnormal; bone scan: 74% abnormal; MRI and CT normal in asymptomatic athletes	B	2
Gaeta et al.[20] (2006)	Case control	Exercise-related pain at posteromedial tibial border	High-resolution CT scan	20 asymptomatic athletes, 10 asymptomatic non-athletes, 11 symptomatic (14 tibias)	Distance runners, 18–26 y; 32% F, 68% M	Asymptomatic non-athletes: 95% tibias normal. Asymptomatic athletes: 45% abnormal; all 14 painful tibias: abnormal	B	2
Fredericson et al.[21] (1995)	Retrospective cohort	Runners with tibial pain with confirmation of MTSS; tibial stress reaction of tibial stress fracture on bone scan	MRI/bone scan	14: 18 tibias	Runners (track, hurdles, distance runners), 18–21 y; 21% M, 79% F	Grade I and II: periosteal oedema and bone marrow oedema on T2 weighted; grade III and IV: periosteal oedema on T2, marrow oedema on T1 and T2. Correlation bone scan/MRI in 78%	B	3

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Table II. Contd

Study (year)	Study design	Inclusion criteria	Imaging type	No. of subjects	Population/type of activity	Outcome	Methodological quality	Level of evidence
Arendt et al.[22] (2003)	Retrospective cohort	Athletes who underwent MRI with suspicion of stress fracture	MRI	26	Athletes; basketball, running, football, gymnastics, ice hockey, track, tennis, softball; 31% M, 69% F	The more severe the lesion on MRI the longer the time to return to sport	B	3
Rupani et al.[23] (1985)	Case series	Not clearly stated	Bone scan	44	Recreational and competitive athletes, 11–72 y; F/M ratio not clearly stated	Distinguishing tibial stress fractures and MTSS is possible with bone scan	C	3
Nielsen et al.[24] (1991)	Case series	Pain along the posteromedial border	Bone scan/x-ray	22: 29 tibias	Male soldiers (age unknown)	x-Ray: 45% abnormal; bone scan: 83% abnormal uptake; 17% normal	C	3
Anderson et al.[25] (1997)	Case series	Activity-related lower leg pain and tenderness on palpation along the posteromedial tibia	MRI/x-ray	19	Competitive and recreational athletes, 17–54 y; 58% F, 42% M	37% MRI normal, 26% MRI periosteal fluid; 26% MRI bone marrow oedema, 11% stress fracture; x-ray: 5/5 normal	C	3
Matilla et al.[26] (1999)	Case series	Medial tibial pain within 500 m of marching; x-ray normal, pain >5 cm along tibial shaft	MRI	12: 14 tibias	Male soldiers, 17–25 y	93% periosteal oedema; 29% intraosseous bright signal and periosteal oedema	C	3
Aoki et al.[27] (2004)	Case series	Pain in the middle or distal portion of the medial side of the leg; normal x-ray	MRI	14 MTSS, 8 stress tibial fracture	Athletes (runners, basketball, volleyball, kendo, soccer players), 13–33 y; 59% M, 41% F	14/14: linear abnormally high signal along posteromedial border, 50% abnormally high signal of bone marrow, 36% both abnormal signals seen. After 4 wk, with continued exercise, MRI signals diminished in 5 patients	C	3

CT = computed tomography; **F** = female; **M** = male; **MRI** = magnetic resonance imaging; **MTSS** = medial tibial stress syndrome; **T1** = T1 weighted; **T2** = T2 weighted.

Table III. Study characteristics and quality of studies concerning **intrinsic** risk factors

Study (year)	Study design	Inclusion criteria	No. of subjects	Population	Risk factors (specification of determinant)	Outcome	Methodological quality	Level of evidence
DeLacerda[28] (1980)	Prospective cohort	Pain along the posteromedial aspect of the tibia	81	Female, physical education students, 18–21 y	Navicular displacement weight bearing/ non-weight bearing	Incidence MTSS 37%. Navicular drop 8.90 ± 2.89 mm in MTSS group, control 5.56 ± 2.32 mm	A2	2
Bennett et al.[3] (2001)	Prospective cohort	Pain with palpation over the distal 2/3 of the posterior medial tibia	125	Cross-country runners, 14–17 y; 46% M, 54% F	Navicular drop test	Navicular drop test ($p = 0.01$), female sex ($p = 0.003$)	A2	2
Burne et al.[29] (2004)	Prospective cohort	At least 1 wk medial tibial pain on exertion and >10 cm pain on palpation at distal 2/3 of posteromedial tibia	158	Military cadets, 17–21 y; 77% M, 23% F	Men only: greater internal and external hip ROM, leaner calf girth	Incidence MTSS 15%. Incidence 15% F, 10% M. Greater internal and external ROM ($p < 0.05$), leaner calf girth ($p = 0.04$)	A2	2
Yates and White[4] (2004)	Prospective cohort	Pain, due to exercise along the posteromedial tibial border, on palpation diffuse >5 cm	125	Naval recruits, 17–35 y; 75% M, 25% F	Female sex (RR 2.03), more pronated foot type (RR 1.70)	Incidence MTSS 36%. Incidence 53% F, 28% M	A2	2
Plisky et al.[30] (2007)	Prospective cohort	Pain along the distal 2/3 of the tibia exacerbated with repetitive weight-bearing activity	105	Cross-country runners, 14–19 y; 56% M, 44% F	Higher BMI (RR 5.0)	Incidence MTSS 15%. 4.3/1000 athletic exposures (F), 1.7/1000 athletic exposures (M)	A2	2
Hubbard et al.[31] (2009)	Prospective cohort	Exercise-related pain along the posteromedial side of the tibia for at least 5 cm with diffuse pain on palpation	146	Collegiate athletes from NCAA division I and II, 20 ± 1.7 y; 45% M, 55% F	Athletic activity <5 y, previous history of MTSS/ stress fracture, use of orthotics	Incidence MTSS 20%. Incidence 11% F, 31% M	A2	2
Gehlsen and Seger[32] (1980)	Case control	Not clearly stated	10 symptomatic, 10 control	Female athletes, age not stated; 10 symptomatic, 10 control	Increased plantar flexor strength. Decreased inversion flexibility (right ankle), increased angular displacement (Achilles tendon/calcaneus)	Increased plantar flexor strength ($p < 0.05$). Decreased ankle inversion ($p < 0.05$). Increased angular displacement ($p < 0.05$)	B	3

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Table III. Contd

Study (year)	Study design	Inclusion criteria	No. of subjects	Population	Risk factors (specification of determinant)	Outcome	Methodological quality	Level of evidence
Viitasalo and Kvist[33] (1983)	Case control	Regular or long lasting pain on the medial border of the distal 2/3 of the tibia	13 controls, 13 with frequent and long-lasting MTSS, 22 slight MTSS	Male distance runners, judo, soccer, skiing, boxing, basketball. Age: control 30.6 ± 7 y, frequent 23.8 ± 7 y, slight 19.8 ± 5 y	Increased mobility of inversion, eversion and sum. Achilles tendon angle displacement smaller during full support phase	Passive mobility inversion ($p < 0.01$), eversion ($p < 0.05$), sum ($p < 0.001$). Angular displacement during full support ($p < 0.01$)	B	3
Sommer and Vallentyne[34] (1994)	Case control	Regular or long lasting pain on the medial border of the distal 2/3 of the tibia	25 subjects: 15 controls, 10 cases of which 4 bilateral	Amateur folk dancers, 15–25 y; 80% F, 20% M. 10 previously diagnosed with MTSS, 15 controls	Combination of forefoot and hindfoot varus alignment. Standing foot angle <140°	Forefoot and hindfoot varus ($p = 0.047$). Standing foot angle <140° ($p = 0.0001$)	B	3
Madeley et al.[35] (2007)	Case control	Exercise-induced leg pain of the posteromedial border of the tibia. Pain on palpation >40 mm on 100 mm visual analogue pain scale	30 symptomatic (with 59 painful tibias), 30 controls	Athletes, 17–47 y; MTSS group 53% M, 47% F; control group 53% M, 47% F	Standing heel-rise repetitions	Standing heel rise ($p < 0.001$)	B	3
Tweed et al.[36] (2008)	Case control	Exercise-induced pain on the posteromedial border of the tibia for at least 4 cm and pain on palpation	12 control, 28 with MTSS	Runners, 18–56 y; MTSS group: 43% F, 57% M; control group: 42% F, 58% M	Early heel lift, abductory twist during gait, apropulsive gait	Early heel lift (EOR 27), abductory twist (EOR 123), apropulsive gait (EOR 823)	B	3
Bandholm et al.[37] (2008)	Case control	Exercise-induced pain on the posteromedial tibial border and pain on palpation >5 cm	15 control, 15 with MTSS	Athletes, 20–32 y; MTSS group 60% F, 40% M; control 60% F, 40% M	Larger navicular drop and MLAD during stance. Larger MLAD during gait	Larger navicular drop during stance ($p = 0.046$), larger MLAD during stance ($p = 0.037$), MLAD during gait ($p = 0.015$)		
Taunton et al.[38] (2002)	Retrospective cohort	Not clearly stated	2002 running injuries	Runners, mean age MTSS subgroup 30.7 y; 43% M, 57% F	Below average activity history (OR 3.5 M, OR 2.5 F)	Incidence 5% MTSS	B	3

BMI = body mass index; **EOR** = estimated OR; **F** = female; **M** = male; **MLAD** = medial longitudinal arch deformation; **MTSS** = medial tibial stress syndrome; **NCAA** = National Collegiate Athletic Association; **OR** = odds ratio; **ROM** = range of motion; **RR** = relative risk.

Table IV. Methodological quality of randomized controlled trials according to the Delphi criteria (treatment) [reproduced from Verhagen et al.,[39] with permission]

Study (year)	No. of subjects	Population	Intervention	Outcome	Delphi items[a]									Total score	Methodological quality	Level of evidence
					1a	1b	2	3	4	5	6	7	8			
Andrish et al.[2] (1974)	2777	First-year male midshipmen; age not stated	Five groups: ice application; aspirin (acetylsalicylic acid) and ice; phenylbutazone and ice; heel-cord stretching and ice; walking cast	Incidence MTSS 4%. No significant differences	+	+	+	+	–	–	–	–	–	4/9	A2	2
Nissen et al.[40] (1994)	23 experimental, 26 control	Soldiers; age not stated	Two groups: active laser treatment; placebo laser	No significant differences between groups in VAS score and days to return to active duty	+	+	+	–	–	+	+	–	–	5/9	A2	
Johnston et al.[41] (2006)	7 experimental, 6 controls	Soldiers; 18–37 y; sex not stated	Two groups: leg orthosis and walk-to-run programme; walk-to-run programme	No significant differences between groups in days to recovery ($p = 0.575$)	+	+	+	–	–	–	–	+	+	5/9	A2	2

a Delphi items (+ indicates 'yes', – indicates 'no'):[39]

1a: Was a method of randomization performed?

1b: Was the treatment allocation concealed?

2: Were the groups similar at baseline regarding the most important prognostic indicators?

3: Were the eligibility criteria specified?

4: Was the outcome assessor blinded?

5: Was the care provider blinded?

6: Was the patient blinded?

7: Were point estimates and measures of variability presented for the primary outcome measures?

8: Did the analysis include an intent-to-treat analysis?

MTSS = medial tibial stress syndrome; **VAS** = visual analogue scale.

Table V. Methodological quality of randomized controlled trials according to the Delphi criteria (prevention) [reproduced from Verhagen et al.,[39] with permission]

Study (year)	Population	Intervention	Outcome (incidence)	Delphi items[a]								Total score	Methodological quality	Level of evidence
				1a	1b	2	3	4	5	6	7	8			
Andrish et al.[2] (1974)	2777 first-year male midshipmen; age not stated	Five groups: control; heel pad of foam rubber; heel cord stretches; heel pad and stretches; graduated running programme prior to training	Control group 3.0%, heel pad group 4.4%. No significant difference was found	+	+	+	+	–	–	–	–	–	4/9	A2	2
Bensel and Kish[42] (1983)	2841 army basic trainees; age 16–41 y; 73% M, 27% F	Two groups: 1 hot weather boots; 2 black leather combat boots	1. M 0.27%, F 1.18%; 2. M 0.22%, F 1.17%. Not significant	+	+	+	+	–	–	–	+	–	5/9	A2	2
Bensel[43] (1986)	555 female soldiers; age unknown	Three groups: urethane foam insole; moulded network of lever-like projections attached to material in grid form; standard plastic mesh with nylon	MTSS with different insoles, varying from 5.9% to 7.4%. Not significant	+	+	+	–	–	–	–	+	–	4/9	A2	2
Schwellnus et al.[44] (1990)	1388 military recruits; 17–25 y; sex not stated	Two groups: neoprene impregnated with nitrogen bubbles covered with nylon; no intervention	MTSS: control 6.8%, experimental 2.8% ($p < 0.05$)	+	+	+	–	–	–	–	–	–	3/9	A2	2
Schwellnus and Jordaan[45] (1992)	1398 male military recruits; age < 25 y	Two groups: 800 mg/day calcium supplementation; no supplementation	MTSS: control 20.4%, calcium group 33.3%. Not significant	+	+	+	–	–	–	–	–	–	3/9	A2	2
Pope et al.[46] (2000)	1538 male army recruits; age 17–35 y	Two groups: stretching gastrocnemius, soleus, hamstring, quadriceps, hip adductor and hip abductor muscle groups; no stretching. Both groups same physical protocol	MTSS 1.6%. No effect of stretching on injury risk. LR = 1.24, HR 1.23	+	+	–	+	–	–	–	+	+	5/9	A2	2
Larsen et al.[47] (2002)	146 military conscripts; 145 men and 1 woman; age 18–24 y	Two groups: custom-made biomechanic shoe orthose; no intervention	MTSS: control group 38%, intervention group 9% ($p = 0.005$). RR 0.2; cost per prevented case $US101 (2002 values)	+	+	+	–	+	+	–	+	+	7/9	A2	2
Brushöy et al.[48] (2008)	1020 military recruits training for Royal Danish Life Guard; 19–26 y; sex not stated	Two groups: strength, coordination and stretching exercises of the legs; strength and stretching exercises of upper body	MTSS: leg training 4.5%, upper body training 4.9%. Not significant	+	+	+	–	+	–	+	+	–	6/9	A2	2

a Delphi items (+ indicates 'yes', – indicates 'no'):[39]

1a: Was a method of randomization performed?

1b: Was the treatment allocation concealed?

2: Were the groups similar at baseline regarding the most important prognostic indicators?

3: Were the eligibility criteria specified?

4: Was the outcome assessor blinded?

5: Was the care provider blinded?

6: Was the patient blinded?

7: Were point estimates and measures of variability presented for the primary outcome measures?

8: Did the analysis include an intent-to-treat analysis?

F = female; **HR** = hazard ratio; **LR** = likelihood ratio; **M** = male; **MTSS** = medial tibial stress syndrome; **RR** = relative risk.

Beck et al.[51] dissected 50 legs and concluded that if a traction was implicated in MTSS, the soleus muscle and the flexor digitorum longus muscle rather than the tibialis posterior muscle could be involved. During dissection, no fibres of the tibialis posterior muscle were found on the distal half of the posteromedial border of the tibia. In the upper half of the distal tibia, fibres of the soleus muscle and flexor digitorum longus muscle were abundant on the medial border. While MTSS complaints are commonly felt in the distal third of the tibia, few muscle fibres of the soleus muscle or any other muscle were found at this site.[51]

Garth and Miller[52] concluded, after performing a case-control study in 17 athletes, that the flexor digitorum longus muscle caused the complaints. In the symptomatic group he found a decreased flexion range of motion of the second metatarsophalyngeal joint and weakness of the toe flexors. He suggested that this was caused by permanent increased activity of the flexor digitorum longus muscle.

Traction of the above-stated muscles on the periosteum is thought, by some authors, to cause MTSS. The traction explanation was first published in the 1950s.[7] This states that complaints are due to repeated traction on the periosteum of the fibres of the tibialis posterior, soleus or flexor digitorum longus muscles. However, symptoms are not always felt at the site of distal attachment of the tibialis posterior, soleus and flexor hallucis longus muscles.[51] The traction explanation has only recently been investigated. Using three cadaver specimens, traction on the periosteum during soleus, posterior tibial and flexor digitorum longus activity was measured.[53] As tension on the tendons of the aforementioned muscles was increased, strain in the tibial fascia, which in our opinion refers to the periosteum, increased in a linear manner.

2.2 Biomechanics

Several explanations for the development of MTSS are found in the literature, of which the traction explanation is one. Another explanation for which much evidence exists states that repeated tibial bending or bowing causes MTSS.[54,55] This mechanism has similarities to the aetiology of a tibial stress fracture.[56] Animal studies showed that repeated bending causes adaptation of the tibia, predominantly at the site where bending forces are the greatest.[57,58] The site of most profound bending is where the tibial diaphysis is narrowest[59] – approximately at the junction of the middle and distal thirds. The goal of adaptation is to strengthen the bone to resist future loading. The adaptation is described in Wolff's law and the Utah paradigm;[60-64] loads on bones cause bone strains that generate signals that some cells can detect and to which they or other cells can respond. Normally, bones can detect and repair small microdamage caused by strains that stay below the microdamage threshold. Strains above the threshold can cause enough microdamage to escape repair and accumulate.[62]

The first clinical study to provide evidence for an altered bending mechanism in MTSS was provided by Franklyn et al.[65] in a recent cohort study. Tibial scout radiographs and cross-sectional computed tomography (CT) were used to study bone characteristics of aerobic controls, MTSS subjects and subjects with tibial stress fractures. These authors showed that male subjects with MTSS and tibial stress fractures had a smaller cortical area than aerobic controls. They also calculated that aerobic controls were better adapted to axial loading, torsion, maximum and minimum bending rigidity, and pure bending than subjects with MTSS and tibial stress fractures.[65]

In addition, animal and human studies showed that diminished muscle forces negatively influence the bone adaptation process, when weaker muscles opposing tibial bending allow more bending to occur.[66-69] A recent *in vivo* study showed greater tibial strain when muscles were fatigued.[70]

A combination of the traction and bony overload explanation is another hypothesis. The adaptation to loading of the tibia is further challenged by the traction of the soleus and flexor hallucis longus muscles on the periosteum.[56]

3. Histology

Histological evidence for periostitis is sparse. Two studies from the 1980s describe three

patients with inflammation or vasculitis found in the fascia after biopsy.[10,49] In the study by Michael and Holder[49] a thickened periosteum was seen, and termed 'periostitis'. The study by Mubarak et al.[10] showed two patients with microscopic inflammation and vasculitis of the periosteum. In larger studies, inflammatory cells were not often found in the periosteum.[71,72] Inflammatory changes were found in the crural fascia in 13 of 33 athletes upon biopsy.[72] In the same specimens, one biopsy sample showed evidence of plasma cell infiltration surrounding wide lymphatics in the periosteum, along with a thickened periosteum and increased osteoblast activity. This was also found by Bhatt et al.,[71] who also found fewer osteocytes compared with normal bone, although this finding just failed to achieve statistical significance.[71] They did not describe the activities of their patient population.

Evidence in recent literature is accruing that osteocytes play a major role in mechanotransduction, a mechanism through which bone senses mechanical stimuli.[73,74] Osteocytes probably promote bone remodelling in response to a direct mechanical stimulus or to bone microdamage.[75] In bone remodelling, apoptosis of osteocytes is seen and this apoptosis may influence osteoclast formation and/or function.[76]

In patients with MTSS, low regional tibial bone density has been found compared with healthy athletes.[77] Bone density in the mid- to distal tibia, measured by dual energy x-ray absorptiometry (DXA), was 23% ± 8% (mean ± SD) less in patients with MTSS. The bone density regained normal values when the athletes had recovered after a mean of 5.7 years (range 4–8 years).[78]

4. Patient Examination

4.1 History

Patients with MTSS present with exercise-induced leg pain. The pain is located along the posteromedial border of the tibia, usually in the middle or distal thirds. Initially symptoms are present on starting activity and subside with continued exercise, but later on pain continues to be present during activity. If symptoms worsen, then the pain can be felt even after the activity ceases.[79,80] This has also been described in stress fractures, so the physician should be cautious when interpreting this symptom. In severe cases, even performing activities of daily living will provoke symptoms.

4.2 Physical Examination

Few articles were found concerning physical examination and MTSS. V. Ugalde has performed non-published research (personal communication 2006). In this research, an attempt was made to determine the sensitivity and specificity of physical examination tests. Symptomatic athletes and control athletes were included. The gold standard in this study was bone scintigraphy. Three tests were examined: diffuse posteromedial pain on palpation, pain on hopping, and pain on percussion. Diffuse posteromedial pain on palpation was the most sensitive test.

During physical examination, pain is present on palpation of the distal two-thirds of the posteromedial tibial border. Mild swelling of the tibia can sometimes be present.[79-81] The risk factors for MTSS are described in section 5 and should be considered during clinical examination.

The differential diagnosis of exercise-induced leg pain consists of medial tibial stress syndrome, tibial stress fracture, exertional compartment syndrome, and to a lesser extent popliteal artery entrapment and nerve entrapment.[81]

It is our opinion that differentiation between MTSS, tibial stress fracture and exertional compartment syndrome can usually be accomplished without additional imaging. Patients presenting with exertional compartment syndrome complain of cramping, burning or aching pain, and tightness in the leg on exercising. A tight feeling in the muscles and sometimes neurological symptoms such as sensory abnormalities can also be present. Palpation at rest is usually not painful. During exercise the leg is painful, but upon stopping the pain disappears quickly. The diagnosis can be confirmed by intracompartmental pressure measurements.[81] In the 1970s and 1980s some thought that MTSS was caused by elevated leg

compartment pressures. Puranen and Alavaikko[82] studied this in 1981 by measuring the pressure in 22 patients with pain on the medial side of the leg. They found that on exertion, patients had significantly higher increases in pressure than controls. On exertion the pressure ranges in patients and controls were 70–150 mmHg and 15–30 mmHg, respectively. Other researchers failed to find elevated pressure.[10,83,84] In one study, 14 track runners with MTSS showed no elevated pressure present in any compartment.[84] In a series of 12 patients with MTSS, compartment pressures were measured and compared with diagnosed chronic compartment syndromes. In the MTSS group, pressures were lower compared with diagnosed compartment syndrome during exercise (84 mmHg [mean value] vs 112 mmHg, respectively).[10] In 12 patients with MTSS the pressures during exercise were compared with pressures in the compartment syndrome. Values in the compartment syndrome group were higher (28 mmHg and 70 mmHg, respectively).[83] All four studies examining compartmental pressure in MTSS examined relatively few patients, and were of poor methodological quality.

Some claim that medial tibial stress syndrome and compartment syndrome may coexist, but apart from the sole study of Puranen and Alavaikko[82] in 1981, no evidence exists.

The differentiation between stress fracture and MTSS can sometimes be challenging, especially since radiographs for stress fracture can be false negative with sensitivities as low as 26–56%.[23,85,86] In stress fractures, pain is usually more focal, while in MTSS the pain is more diffuse. Also, night pain and pain on percussion are not usually present in MTSS. Evidence has shown that in persons with stress fractures, plain radiographs are often normal in the first few weeks and may later show callus formation.[6] Bone scintigraphy and magnetic resonance imaging (MRI) are widely used to confirm the diagnosis.[87]

4.3 Imaging

There is a fair amount of literature on MTSS and imaging. In most of the imaging studies the clinical diagnosis is used as the gold standard when establishing sensitivity and specificity of imaging modalities.[18-20] The fact that history and physical examination is used as the gold standard confirms that the diagnosis is made clinically and that the role of additional investigations is limited.

Table II describes the study characteristics, methodological quality and results of the imaging studies.

4.3.1 Radiograph

Imaging MTSS with radiograph is not appropriate, with most authors reporting normal radiographs.[25,27,77,88] Callus formation is seldom seen on the medial side of the tibia. In one study, four of 46 patients with pain on palpation for at least 5 cm along the posteromedial tibia showed periosteal elevation on radiograph.[18] In other research describing callus formation, the inclusion criteria for the study were less clear.[10,49]

4.3.2 Bone Scan

In 1984, Holder and Michael[16] were the first to examine MTSS with three-phase bone scans (angiograms, blood-pool images and delayed images) in a prospective study. On delayed images, longitudinal tibial lesions of the posterior cortex, involving one-third of the length of the bone, were seen (figure 1). They suggested that MTSS was a condition in which the periosteum is irritated and osteoblasts are activated. Some years later other researchers studying different athletic populations reached the same conclusion.[17,24] Prospective studies on bone scans by Batt et al.[18] and Gaeta et al.[19] showed a sensitivity of 74–84%. Batt et al. found a 33% specificity (positive likelihood ratio [LR+]/negative likelihood ratio [LR–] 1.25/0.48). The low specificity is explained by the high number of positive scans in asymptomatic athletes and controls.

In 1987 and 1988, respectively, Zwas et al.[89] and Matin[90] developed a grading scale for the severity of abnormalities found on bone scan for bone stress injuries. They divided scintigraphic findings into four or five grades. Although the study of Zwas et al. was aimed at stress fractures of the tibia, the results of his study were later used to distinguish between stress fracture and MTSS.

Fig. 1. Bone scintigraphy. Arrows show abnormal longitudinal uptake in lateral view (reproduced from Aoki et al.,[27] with permission).

Suggestions of a continuum between MTSS and stress fracture were already made in 1979.[91] Differentiating between these two entities has proved difficult with bone scans.[17,18]

Batt et al.[18] found in their prospective study, including mainly dancers and runners, that four out of five asymptomatic athletes had abnormalities on bone scans. Other studies also showed false positive bone scans.[16,17] A study of 100 athletes presenting with back complaints, where bone scans were performed, examined the incidence of abnormalities in the lower leg:[92] 34% of the athletes had abnormalities in the lower extremity. None of the regions of abnormal lower extremity uptake was symptomatic at the time of initial evaluation. They were referred to a sports medicine clinic and remained asymptomatic after 8–14 months of follow-up.

4.3.3 Magnetic Resonance Imaging

In the last decade MRI has increasingly been used for studying MTSS. On MRI, periosteal oedema and bone marrow oedema can be seen (figure 2a and b).[25,26]

Only two studies were found that prospectively examined the sensitivity and specificity of MRI in MTSS. Researchers found that MRI had a 79–88% sensitivity and 33–100% specificity and LR+/LR– of 1.18/0.64 for the diagnosis of MTSS.[18,19] The 100% specificity Gaeta et al.[19] described is based on ten asymptomatic athletes with no abnormalities on MRI.

Fredericson et al.[21] and Arendt and Griffiths[5] both developed a grading system for MTSS on MRI, in which Arendt's system was modified from Fredericson's. In this grading system MTSS and stress fracture are separated and the severity is graded. In stress fractures more bone marrow oedema and sometimes a fracture line is seen compared with MTSS.

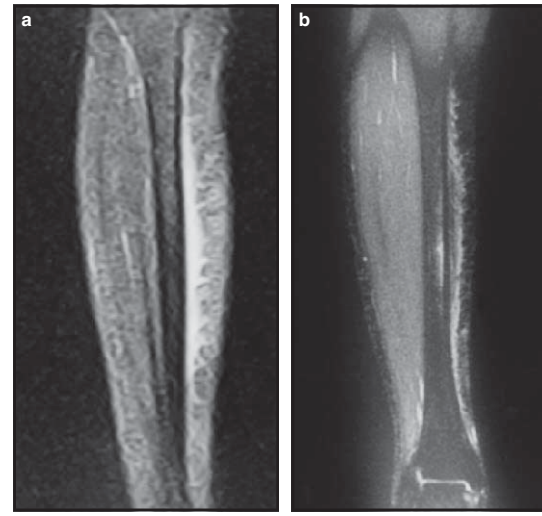

Fig. 2. Coronal magnetic resonance image showing an abnormally high signal on (**a**) the medial side of the tibia and (**b**) along the medial border and the medial side of the bone marrow (reproduced from Aoki et al.,[27] with permission).

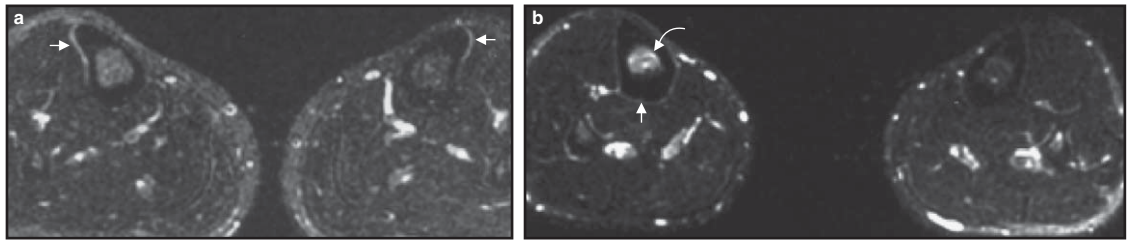

Fig. 3. Axial T2 weighted images in asymptomatic runners showing (**a**) periosteal oedema (straight arrows) and (**b**) bone marrow oedema (curved arrow) [reproduced from Bergman et al.,[93] with permission].

Arendt and Griffiths[5] found, in a retrospective study, that MRI can estimate the time to return to sport. To estimate this, they used an MRI grading scale, previously developed by Fredericson et al.[21] Grade I (positive short T1 inversion recovery [STIR] image) returned to sport in about 4 weeks; grade II (positive STIR and positive T2 weighted) returned to sport in about 6 weeks.

It is questionable whether grading on bone scan and MRI can be compared. Batt et al.[18] found a positive correlation between the two imaging techniques in 23 athletes where both bone scan and MRI were performed. Fredericson et al.[21] found no correlation when the MRI and bone scan were compared in 14 athletes with MTSS.

Research from Japan[27] points out that MRI can distinguish between stress fracture and MTSS soon after the beginning of tibial complaints. No MRI scans of patients with MTSS showed a signal extending throughout the whole bone marrow, which was present in stress fractures. In MTSS a linear abnormally high signal along the posteromedial border of the tibia and the bone marrow was seen. This study also showed that five athletes with MTSS, who were followed up by MRI 4 weeks after initial MRI, and who continued sports activity, did not develop a stress fracture. In chronic cases (defined as complaints for >46 months in a study investigating athletes, mainly runners) MRI scans were normal in seven patients.[25]

Despite abnormalities found on MRI in symptomatic patients, Bergman et al.,[93] in a study with 21 distance runners, showed that 43% had a tibial stress reaction while asymptomatic (figure 3a and 3b). These runners ran 80–100 km a week for 8 weeks and continued doing this. None of these runners developed complaints.

4.3.4 High-Resolution Computed Tomography Scan

With high-resolution CT scan, Gaeta et al.[19,20] showed osteopenic changes in the tibial cortex and few resorption cavities (figure 4). A case-control study reported a sensitivity and specificity of 42% and 100%, respectively (LR 0.58).[19] In ten asymptomatic non-athlete controls, one tibia showed mild abnormalities (slightly reduced cortical attenuation). In 20 asymptomatic runners, 18 of the 40 tibias showed abnormalities (ranging from slightly reduced cortical attenuation to cortical osteopenia). All

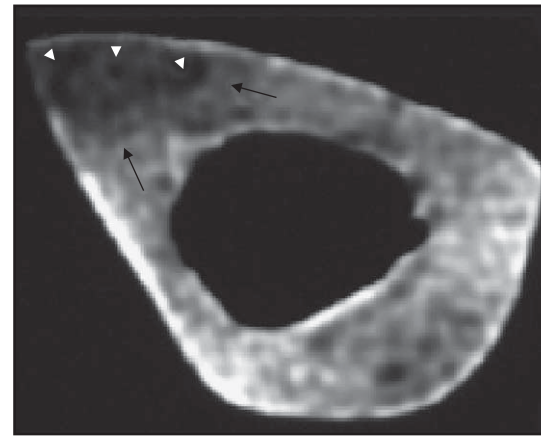

Fig. 4. Axial CT scan showing cortical osteopenia (black arrows) and small resorption cavitations (white arrows) [reproduced from Gaeta et al.,[20] with permission].

symptomatic tibias in patients with MTSS showed cortical osteopenia.[20]

4.3.5 Imaging Summary

The diagnosis of MTSS should be made clinically. In cases where the diagnosis is unclear the physician may perform a bone scan or an MRI, which have approximately the same sensitivity and specificity. Compared with these values the sensitivity of CT scanning is lower, with a higher specificity.

5. Risk Factors

5.1 Risk Factor Studies

A number of prospective case-control and retrospective studies have examined intrinsic risk factors. Extrinsic risk factors have been poorly studied. The methodological quality and results of the risk studies are described in table III.

One of these intrinsic risk factors is overpronation.[4,32,33] However, the definition of pronation in different articles varies. Pronatory foot type was shown to be a risk factor in a prospective military study by Yates and White (relative risk [RR] 1.70),[4] using the Foot Posture Index.[94,95] Gehlsen and Seger[32] and Viitasalo and Kvist[33] found increased pronation upon heelstrike to be a risk factor in two athlete case-control studies. In the study by Gehlsen and Seger,[32] the angular displacement between the calcaneus and the midline of the leg while running was significantly greater ($p < 0.01$) in the MTSS group compared with the non-MTSS group. Viitasalo and Kvist described the same finding as Gehlsen and Seger.[32] The angle between the lower leg and calcaneus at heel strike was higher for the symptomatic group ($p < 0.01$).

Equivalents of pronation, measured with the navicular drop test and the standing foot angle, have also been studied. Four prospective studies were published examining the navicular drop test (the difference in distance between the lower border of the navicular and the ground – loaded and unloaded).[3,28,30,31] The navicular drop test is an indicator of midfoot pronation. Attention to the navicular prominence is also paid in the Foot Posture Index.[94,95] The navicular drop test was measured in the study by Bennett et al.[3] of 125 runners. The mean drop distance in runners with complaints was 6.8 mm (± 3.7 mm), compared with 3.7 mm (± 3.3 mm) in the asymptomatic group ($p = 0.003$). In the second study, a significant correlation was found between navicular tuberosity displacement and the incidence of MTSS (8.9 ± 2.9 compared with 5.6 ± 2.3 mm) [$p < 0.01$].[28] A recent case-control study conducted among athletes showed a significant difference ($p = 0.046$) in navicular drop between loaded and unloaded groups (MTSS group 7.7 ± 3.1 mm, control group 5.0 ± 2.2 mm).[37] A third and fourth prospective study failed to find a significant relationship between navicular drop and MTSS.[30,31]

The standing foot angle measures the angle between medial malleolus, navicular prominence and first metatarsal head. Sommer and Vallentyne[34] found that a standing foot angle <140° was predictive of MTSS ($p < 0.0001$). The 140° cut-off value was chosen because this led to the best sensitivity and specificity (71.3% and 69.5%, respectively).

Recently, two case-control studies examined the functional foot posture in MTSS patients during gait[37] and while running.[36] Using three-dimensional gait analysis, the group of recreational athletes with MTSS showed increased medial longitudinal arch deformation during gait compared with healthy controls ($p = 0.015$).[37] This study also showed increased medial longitudinal arch deformation upon standing compared with the controls. In the study by Tweed et al.,[36] athletes were videotaped during running with and without shoes. Of the variables tested during running, three were significantly different between the groups: early heel rise ($p = 0.003$; estimated odds ratio [OR] 27), abductory twist of the forefoot ($p = 0.003$; estimated OR 123) and apropulsive gait ($p < 0.001$; estimated OR 827).

The role of passive inversion and eversion was investigated in a case-control study by Viitasaalo and Kvist[33] among male athletes, showing increased passive inversion (19.5 ± 8.6°) and eversion (10.7 ± 4.4°) in the ankle to be an intrinsic risk factor ($p < 0.05$). The inversion and eversion were measured manually and repeatedly. The correlation coefficient for this measurement was 0.84.

Although suggested in the literature, reduced ankle dorsiflexion has not been shown to be an intrinsic risk factor in a prospective study.[29] Ankle dorsiflexion for males and females was 32° and 29°, respectively, in the case group and 31° and 27° in the control group ($p > 0.05$). Another prospective study showed that increased plantar flexion range of motion was associated with MTSS ($p = 0.004$). This study was conducted among collegiate athletes.[31] A case-control study published in 1980 reported significantly increased plantar flexion strength values ($p < 0.05$), using cable tension procedures, in ten athletes with MTSS compared with ten healthy athletes.[32]

In an Australian military prospective study by Burne et al.,[29] greater internal and external ranges of hip motion was a risk factor ($p = 0.01$–0.04 for left and right hip). This was measured with the hip and knee flexed to 90°, with the hip rotated until a firm end feel. The extra amount of internal and external hip ranges of motion among patients was 8–12°.

In the same study[29] the lean calf girth (the maximal calf perimeter corrected for skin thickness) was 10–15 mm less among symptomatic cadets compared with asymptomatic cadets. This finding was only significant among males ($p < 0.04$). Leaner calf girth may also be biomechanically (see section 2.2) associated with MTSS due to reduced shock-absorbing capacity.[66-69] However, lean calf girth is not strictly correlated with calf muscle strength.[96]

In a case-control study, Madeley et al.[35] found a significant difference in the number of heel raises that could be performed. MTSS patients succeeded with 23 repetitions per minute compared with 33 in the controls ($p < 0.001$). The study demonstrated muscular endurance deficits in athletes with MTSS.

A higher body mass index (BMI > 20.2) was shown to be an intrinsic risk factor in the prospective study by Plisky et al. (OR 5.3).[30] The study investigated risk factors in a group of cross-country runners.

Female sex is also an intrinsic risk factor.[3,4,29] In a prospective study of naval recruits in Australia the incidence was 52.9% in females compared with 28.2% in males (RR 2.03).[4] The incidence of MTSS in a group of high school cross-country runners in another prospective study was 19.1% in females and 3.5% in males ($p < 0.003$).[3] A prospective study among the Australian Defence Force Academy also showed female sex to be a risk factor (MTSS incidence: females 30.6%, males 9.8%; OR 3.1).[29]

A retrospective Canadian study found that a below-average activity history (<8.5 years) was an extrinsic risk factor (OR 3.5 in males, 2.5 in females).[38] Prior to analysis of the data, the activity history was divided between >8.5 or <8.5 years. The study evaluated the medical records of 2002 running-related injuries between 1998 and 2000. This study was confirmed by a prospective study that showed that athletes with MTSS had been running less years (5.3 ± 1.8 years) than the control group (8.8 ± 4.0 years), who did not develop MTSS ($p = 0.002$).[31]

The same prospective study found that athletes with a previous history of MTSS were more likely to develop MTSS than those who had not developed MTSS in the past ($p = 0.0001$).[31]

Risk factors such as increased running intensity, running distance, change in terrain, change of shoes and running with old shoes are often mentioned,[79] but there are no scientific studies supporting these claims.

5.2 Risk Factor Summary

For the intrinsic risk factors, there is level I evidence for excessive pronation and female sex. Level II evidence is available for the risk factors increased internal and external hip ranges of motion, higher BMI, previous history of MTSS and leaner calf girth.

6. Therapy

6.1 Conservative

Only three randomized controlled trials have been conducted on treatment of medial tibial stress syndrome (table IV). All three studies were conducted among military populations.

In the first study, by Andrish et al.[2] in 1974, 97 marine recruits who developed MTSS were randomized into five groups. The range of the

duration of pain prior to inclusion was 1–14 days. Marines in group one did not run until they were pain free, and applied ice over the painful area three times a day. In group two, aspirin (acetylsalicylic acid) [650 mg four times daily] was added for 1 week. In group three, phenylbutazone (100 mg four times daily) was added for 1 week. In group four, additional calf muscle-stretching three times a day for 3 minutes was added. In group five, a plaster walking cast was applied for 1 week.

The number of days that the marines were not capable of performing at full activity was recorded. The marines were considered recovered if no pain or tenderness remained or when 500 m running was completed comfortably. The time to recovery for the separate groups was: group 1 – 6.4 days; group 2 – 9.4 days; group 3 – 7.5 days; group 4 – 8.8 days; and group 5 – 10.8 days. The mean time to recovery was 8.6 days. No significant difference was found between the intervention groups.

The second study was published in 1994 and had a double-blinded design.[40] Cadets with pain on the posteromedial side of the tibia and pain on palpation of this area were included. The duration of the complaints was not reported. The authors state that other causes besides MTSS of posteromedial pain in the tibia were excluded, without mentioning the specific exclusion criteria. Seventy-two cadets were assessed for inclusion, of which 23 were not eligible or were excluded during the study. The most common reason for exclusion during follow-up was not showing up for treatment. Cadets were randomized into two groups. The first group (n = 26) were treated with a placebo laser probe, while the other group (n = 23) were treated with a functioning laser probe. Both groups received a maximum of six treatments with the probe on the affected part of the tibia. The laser used was a gallium-aluminium-arsenic laser of 830 nm wavelength and 40 mW intensity per 60 seconds per affected centimetre of the tibia. Visual analogue scale (VAS) scores were recorded before every treatment.

After 14 days or a maximum of six treatments a physician decided, based on patient history and physical examination, if the cadet could return to duty or not. In the placebo group 19 of 26 cadets (73%) were able to return to duty, and 18 of 23 cadets (78%) in the laser group. There was no significant difference between the two groups for return to duty and VAS scores. Just before statistical analysis began, the double-blinded design of the study was changed to single-blinded, due to an accidental breakage of the blinding code.

The third study was published in 2006.[41] In this study a leg orthosis was compared with relative rest. The orthosis was an elastic neoprene sleeve with a padded aluminium bar designed to be centred over the most symptomatic portion of the medial leg. Exclusion criterion was any sign of stress fracture on bone scan. Twenty-five soldiers were included, but half of them did not complete the study. Most of them dropped out of the study because of failure to return for follow-up or because of change in permanent training station. Randomization divided the soldiers into two groups: those with and those without a leg orthosis. Both groups followed an identical rehabilitation programme consisting of activity modification and ice massage. Seven days after enrolment in the study a gradual walk-to-run programme was initiated. VAS scores were recorded before and after running. The endpoint was the time until the soldiers could complete running 800 m without pain. Only 13 soldiers completed the rehabilitation programme. Days to completion of the programme were 13.4 ± 4.5 (mean ± SD) days in the orthosis group and 17.2 ± 16.5 days in the control group. These differences were not significant ($p = 0.575$).

In the literature the following treatment regimens are recommended: calf muscle training, using anti-pronation insoles, massage, maintaining aerobic fitness, electrotherapy[97] and acupuncture.[98] Randomized controlled trials or case series studying these treatment options were not found.

6.2 Surgery

The studies reporting surgery were all of poor methodological quality and none had a controlled design. In all of these studies diagnosis was made clinically, and patients with suspected compartment syndrome were excluded.

Surgery is sometimes performed when complaints persist after conservative treatment fails.

Different surgical approaches have been described. Some authors[99,100] performed a fasciotomy along the posteromedial border of the tibia using only local anaesthesia. Others[101] used the same technique, but under general anaesthesia. Abramovitz et al.,[102] Detmer[14] and Yates et al.[103] added removing a strip of the periosteum from along the inner border of the tibia. The effect of the operation is thought to be less traction on the periosteum.[102]

Regarding the pain, good to excellent results were found in 69–92% of patients: 69% by Yates et al.[103] and 92% by Detmer.[14]

Some of the surgical articles report the rate of return to sport[14,100,102,103] The time to return to sport after the operation is poorly stated. Only Detmer[14] states that patients were able to fully resume their sports 3 months after the operation. The results of success in achieving a chance to return to sports have a broad range: 29–93% of patients returned to preoperative sports level. The study by Abramovitz et al.[102] showed 29% return to preoperative sports activity, Holen et al.[100] reported 31%, Yates et al.[103] reported 41%, while Detmer[14] showed a 93% return to preoperative sports level. As mentioned previously, the results should be interpreted with caution, due to the poor study designs.

7. Prevention

7.1 Prevention Studies

Eight randomized controlled trials were found on the prevention of MTSS (table V), all conducted in military populations. The first study, by Andrish et al.,[2] was part of the study that also studied treatment of MTSS. They divided 2777 soldiers randomly into five groups. Group one served as a control group and performed the normal training regimen. The other four subgroups conducted the same training regimen, but to each a preventative intervention was added: the second group wore a heel pad in their shoes; group three performed heel-cord stretching exercises three times daily for 3 minutes; the fourth group performed the same stretches as group three and wore a heel pad; group five entered a gradual running programme 2 weeks before the start of the training schedule and equalled the rest of the groups after the third week of training – they also performed fitness exercises. No significant difference was found between the different groups in incidence of MTSS. In the control group the incidence was 3.0%, with 4.4% in the heel-pad group, 4.0% in the heel cord stretching exercises group, 3.0% in the heel-pad plus heel cord stretching exercises group, and 6.0% in the group with graduated running programme.

The second study[42] examined the effect of two kinds of boots in 2841 soldiers over an 8-week period. Training consisted mostly of physical training, although this was not further specified. One boot was constructed of leather, while the other boot had a nose of cotton and nylon (a boot used in tropical environments). The study was conducted to acquire data regarding the effect of the two types of boots on type and frequency of leg disorders among soldiers. The incidence of MTSS, defined as pain and tenderness of the tibia due to overexertion, was the same in both groups.

In another study,[43] 555 female soldiers were randomized to wear one of three kinds of insoles. A urethane foam insole and a custom-made insole were compared with a standard insole. During 9 weeks all female soldiers followed the same training programme. There were no significant differences between the groups. A definition of MTSS was not stated.

The fourth randomized controlled trial was published in 1990.[44] 1538 soldiers were included, of whom 237 were randomized into an intervention group. They performed 9 weeks of training. The control group wore standard insoles and the intervention group wore neoprene insoles. After 9 weeks of training, 20.4% of the control group had developed MTSS, although this was not defined, compared with 12.8% in the intervention group. This was a significant difference.

The fifth study was conducted in 2002.[47] 146 soldiers were randomized to receive standard insoles or a semi-rigid insole, which was handmade and was adjusted per foot. After 3 months of training a significant difference ($p < 0.005$) was present. Twenty-four (38%) soldiers with the

standard insole developed MTSS, compared with four (8%) in the intervention group. MTSS was not defined in this study.

Schwellnus and Jordaan[45] examined whether calcium supplementation prevented MTSS, for which no definition was given. Of 1398 soldiers, 247 were randomly selected as an experimental group. Before the study started, dietary assessment took place in a selected number of soldiers in the control and experimental groups not yet taking the calcium. Food supplements and calcium intake were calculated. No dietary differences were found. An additional 500 mg of calcium per day was provided to the experimental group. No significant differences were found in the number of patients with MTSS between the experimental and control groups.

The seventh preventive study[46] examined preexercise stretching: 1538 army recruits were randomly allocated to stretch or control groups. The stretching protocol consisted of 20 seconds of static stretching for the different lower leg muscles. The study revealed no significant effect on the occurrence of MTSS, which was not further defined.

A Danish study[48] examined whether the incidence of MTSS was lowered by a prevention training programme during 12 weeks of military training. Platoons were randomized between two types of training: the prevention training programme and the placebo training programme. The prevention training programme consisted of leg strength and coordination and stretching exercises of the legs, while the placebo training programme consisted of strengthening and stretching exercises of the upper body. MTSS was defined as pain on the medial border of the tibia during running, with pain on palpation of the medial tibial border, not localized to one spot. No significant differences were noted between the training groups.

7.2 Prevention Summary

A number of interventions were studied in the various articles about prevention of MTSS, but of these only a shock-absorbing inlay showed a reduction in the incidence of MTSS in two different military studies. For this, level I evidence is available.

8. Discussion

A general weakness when reviewing the literature on MTSS is the confusing terminology and the lack of consensus surrounding the definition, which makes comparison of different studies difficult. No widely used definition of MTSS is available in the current literature. Based on the reviewed literature, the following definition of MTSS is suggested: 'pain felt along the posteromedial border of the tibia'. The pain is aggravated by weight-bearing activity and subsides gradually on stopping. On examination there is recognizable pain on palpation of the posteromedial border over a length of at least 5 cm. This definition distinguishes MTSS from stress fracture, in which the pain is more focal.

It is our opinion that the diagnosis can be established clinically. The high prevalence of abnormal imaging studies in asymptomatic athletes means that these techniques should not be used routinely to establish the diagnosis.

Several studies show that normal bone remodelling involves resorption of bone before the rebuilding of new bone structures occurs.[60-64] Imaging of tibiae of asymptomatic runners shows abnormalities mimicking the abnormalities found in MTSS.[93] This is thought to represent normal remodelling.

From the literature, it is unclear as to whether tibial stress fracture is a continuum of MTSS. In the 1970s Roub et al.[91] were the first to suggest that increased levels of stress to the tibia could result in a spectrum of bony overload. In this spectrum the endstage was a cortical fracture. In the beginning of this spectrum, when bone resorption outpaces bone replacement, MTSS occurs. In both MTSS and stress fractures the same altered bending is present compared with healthy athletic controls.[65]

Although a continuum was suggested in the seventies and in our opinion is attractive, no conclusions can be made. In one study[27] athletes with MTSS kept on running after being diagnosed with MTSS. On follow-up MRI scanning,

there was no evidence of stress fracture. This could mean that MTSS does not develop into a stress fracture, but also that some symptomatic tibiae healed before fracturing. Possibly, bone variations between individuals determine if one person develops MTSS and the other develops tibial stress facture. If MTSS and stress fracture are not two ends of a continuum of bone injury, then further research is needed to identify the unique pathophysiology of the two conditions. For example, histological samples of MTSS could be studied for microcrack patterns and compared with stress fracture findings. Recently, O'Brien et al.[104] and Raesi Najafi et al.[105] studied the behaviour of microcracks in loaded bones. This behaviour could be compared in MTSS and stress fractures. Also, more studies using high-resolution CT scanning comparing findings between MTSS and stress fractures could be conducted. Recently, micro-CT images were obtained to assess bone microdamage.[106] Slices 10 μm thick could be made with this CT device. Highly detailed images of microdamage in MTSS and stress fractures could possibly be studied.

Many controversies surround MTSS. This syndrome has had at least five different names over the past 50 years. Debate still continues as to the underlying cause of MTSS. For decades periostitis caused by traction of the tibialis posterior, flexor digitorum longus or soleus muscles was commonly cited as the mechanism causing MTSS. However, anatomical studies showed that complaints are regularly felt more distal to the most distal attachment of the tibialis posterior, soleus and flexor digitorum longus muscles. Only one study has investigated the role of traction in MTSS and supplied some scientific data to support traction as a possible contributor in the development of MTSS.[53]

Recently bony overload of the medial tibia has been shown to be important as the underlying problem. There are four important findings that support the theory that bony overload forms the primary pathophysiological basis for MTSS: (i) on triple-phase bone scans the last phase is abnormal, showing that the bone and periosteum are involved;[16,18] (ii) on high-resolution CT scan the tibial cortex is found to be osteopenic, as can be seen in patients as well as in asymptomatic athletes as a sign of bone remodelling;[20] (iii) on MRI images, bone marrow oedema as well as a signal along the periosteum can be seen;[19,27] and (iv) in patients with MTSS, bone mineral density is reduced compared with controls,[77] and when symptoms improve, the bone density returns to normal values.[78]

Through prospective studies a number of intrinsic risk factors for MTSS have been established. A pronatory foot type with standing is an intrinsic risk factor.[4] Also, an indicator of midfoot pronation, a positive navicular drop test, is an intrinsic risk factor.[3,28,30] The literature suggests, although opinions vary, that excessive pronation leads to increased internal tibial rotation.[107,108] This could cause higher strains in the tibia and may eventually lead to MTSS. Female sex[3,4,29] is another intrinsic risk factor, in which hypoestrogenism and eating disorders probably play a role. It is well known that hypoestrogenism in menstrual irregularities leads to loss of bone mineral density.[109] Eating disorders, independently of hypoestrogenism, lead to altered modulation of the bone turnover under influence of insulin-like growth factor-1 and leptin hormones.[110] A higher BMI[30] will increase tibial loading and bending, leading to pronounced tibial cortex adaptation[62,64] and increased risk for MTSS. Leaner calf girth is associated with MTSS,[29] because the shock-absorbing capacity of the calf muscles is diminished.[66,67,69] No solid explanation is available as to why greater internal and external hip ranges of motion[29] are intrinsic factors. An external risk factor is the previous history of MTSS, possibly due to individual alterations in individual bone remodelling.[31]

Little research has been conducted on the treatment of MTSS. Only three randomized controlled trials, published 30 years apart, were found.[2,40,41] The result of these studies is that no intervention proved more valuable than rest alone. The use of common therapies such as massage, strengthening exercises for the calf muscles and anti-pronatory orthotics has never been investigated.

Sometimes surgery is performed if conservative treatment fails. The quality of studies

studying surgery for MTSS is poor. These studies show that surgery can be useful for pain reduction, but only few athletes will return to their pre-injury sports level.

Eight studies on prevention of MTSS have been published. Three studies investigated the use of different kinds of insoles. Two studies, using semi-rigid orthotics and a neoprene insole, found a significantly lower incidence of MTSS after this intervention.

9. Conclusions

MTSS is a common overuse injury affecting many athletes and military recruits worldwide. The use of the definition of MTSS first used by Yates and White[4] is recommended: "pain felt along the middle or distal third of the posteromedial border of the tibia". The pain is aggravated by weight-bearing activity and subsides gradually on stopping. On examination there is recognizable pain on palpation of the posteromedial border over a length of at least 5 cm.

It is most probably primarily due to bony overload of the posteromedial tibial border. There is little evidence to support the commonly cited repeated traction-induced periostitis as the primary underlying aetiological factor. Whether or not MTSS and tibial stress fractures are on a continuum is yet to be established and should be investigated further.

MTSS is a clinical diagnosis and the prevalence of abnormal findings in asymptomatic subjects means that results of additional investigations should be interpreted with caution.

There is level I evidence showing that pronatory foot type and female sex are intrinsic risk factors. There is level II evidence showing that BMI, greater internal and external ranges of hip motion, and calf girth are also intrinsic risk factors. Level II evidence is present also for previous history of MTSS as an extrinsic risk factor.

Only three studies have examined the conservative treatment of MTSS. At present there is no evidence that any treatment is superior to rest alone. There is level I evidence that shock-absorbing insoles may help in the prevention of MTSS.

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