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Letters to the Editor

Dear Sir,

South African sport psychology (SP) is facing a crisis. As a sport science it must mobilize itself to facilitate South Africa's re-entry into the world of international sport. Yet the current picture of SP in this country is a fragmented and disorganized one. As a discipline, SP lacks unity and resolve – hardly a situation conducive to the provision of consistent and satisfactory services to South Africa's sporting fraternity.

The most important step towards the establishment of SP as a credible discipline, is the formation of a single, widely recognized SP organization. This will help solve a number of problems currently facing South African SP:

i. It will protect the discipline from those "jumping on the band wagon" who lack adequate training, and will in turn protect the public which uses SP services.
ii. It will present a unified SP front. This will help establish the discipline's credibility not only in the eyes of the public, but also amongst related medical professionals, not surprisingly most of whom don't know either when or how to refer to a "sport psychologist".
iii. It will provide a much needed forum for the resolution of other important issues, such as ethics in SP, the gulf between research and application of SP principles etc.

Currently a number of disparate SP organizations/interest groups exist, none of which however boasts a membership proportional to actual interest in the field. The two such groups which offer the most appropriate frameworks for the future formation of a unified SP organization, are the new Psychological Society of South Africa (PsySSA) which has a division of SP, and the South African Sports Medicine Association (SASMA) which has a SP interest group. Unfortunately the prime movers in the two camps have not always aspired to common goals.

One reason for the disparity of interests is that individuals working in SP have typically come from a variety of backgrounds. This is partly due to the fact there is legally no such thing as a "sport psychologist" in South Africa (hence the inverted commas). Australia is in fact one of the very few countries who do legally recognize the title. In Australia, SP now enjoys greater acceptance and credibility than probably in any other country (no fewer than six psychologists accompanied the Australian Olympic team to Barcelona). I believe South Africa should follow their lead in having the title "sport psychologist" recognized by the psychology profession.

Not only does this title best describe the areas covered by the discipline, but it is also the name by which the discipline is know to all. Additionally it is only a psychologist who is legally, and presumably also practically able to fulfill all the functions which may be required of a "sport psychologist". Hence I believe that SP should, and ultimately will fall under PsySSA as a separate category of registration. The implications of such a move would be that only registered psychologists will be able to register as "sport psychologists".

Clearly this is a very sensitive issue. If and when it happens many non-psychologists working in the field of SP will feel marginalised. Recently there has been a huge upsurge of interest expressed in the field by potential future "sport psychologists". Therefore the longer these issues remain unresolved, the more people will be involved and consequently the messier it will get. In contemplating the future of SP training and registration in South Africa, two facts need to be taken into consideration:

i. Human movement studies departments are an excellent source of vital knowledge concerning both the physiological and psychological aspects of human performance.
ii. Psychology departments offer vital training in counselling and clinical practice, as well as the possibility for ultimate registration as a psychologist.

Based on these realities, I suggest that students interested in SP be advised where possible to do an undergraduate degree with majors in both psychology and human movement studies. Thereafter a student should look towards doing post-graduate honours and masters degrees in psychology to enable him/her to practice legally as a psychologist.

The criteria for full membership of a unified SP organization should include both registration as a psychologist, and some knowledge of human movement studies. Applied sport psychologists should additionally be able to demonstrate having had practical experience in an applied sport setting. Finally, some sort of grandfather clause may be necessary to cater for the non-psychologists with years of experience in the field.

PsySSA is a new organisation which is currently experiencing teething problems of its own. It is still viewed with caution and even skepticism by some psychologists. In light of this, it would also be quite acceptable that a united SP organization be formed under SASMA until such time as PsySSA firmly establishes itself as the controlling body of psychology in South Africa. If this SP organization was later reconstituted under a parent psychology organization, the strong links already established with SASMA would be a bonus. For if there is one thing to be learnt from countries in which SP has developed smoothly and successfully, it is the importance of recognition not only by the parent discipline of psychology, but also by the various sports organisations.

This letter represents just one person's ideas on the subject. Undoubtedly there are many more opposing ones. The point though, is that now is the time to put individual cause to rest, and for the good of the discipline, to voice and hopefully resolve the opposing ideas within a unified SP organization.

Clinton Gahwiler

Diagnosis and management of tarsal stress fractures

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"The success or failure in the diagnosis and management of stress fractures is largely dependent on the enthusiasm with which it is pursued."[1]

Tarsal stress fracture is a common condition in sports medicine[2-4] and its management has improved greatly since the 1920s when patients suffering metatarsal stress fracture had tarsal amputations because of suspicion of neoplasia.[5-7] However, certain tarsal stress fractures continue to be diagnosed late and cause prolonged down time for athletes.[8-10]

This paper reviews reports of the less common tarsal stress fractures in athletes and in military populations. It aims to provide guidance to clinicians by answering the following questions:

i) What is the relative frequency of the different tarsal stress fractures?
ii) What are the clinical features that suggest the presence of a tarsal stress fracture?
iii) What imaging should be used to confirm the diagnosis?
iv) What is the recommended management for each stress fracture?
v) When is the fracture healed?

Introduction

Repetitive physical loading below the single cycle threshold can produce a microfracture in bone.[11] This bone pathology is a continuum often referred to as "bone strain".[2,12,13] The radionuclide scan is a very sensitive tool to diagnose the presence of bone strain.[14-17] By combining clinical features and imaging findings patients' bony injuries can be accurately categorised along the bone strain continuum.[18] Those athletes with symptoms, radioisotope scan evidence of bone strain but without Xray or computerized tomography (CT) evidence of bony pathology are deemed to have a "stress reaction". They would be expected to recover more quickly than those with frank Xray or CT evidence of bony change. This concept is depicted diagramatically in Fig 1. More recent literature does not include "stress reactions" as cases of "stress fracture"[10] but earlier publications included some cases of what may more accurately be labelled "stress reaction" rather than "stress fracture".[19,20]

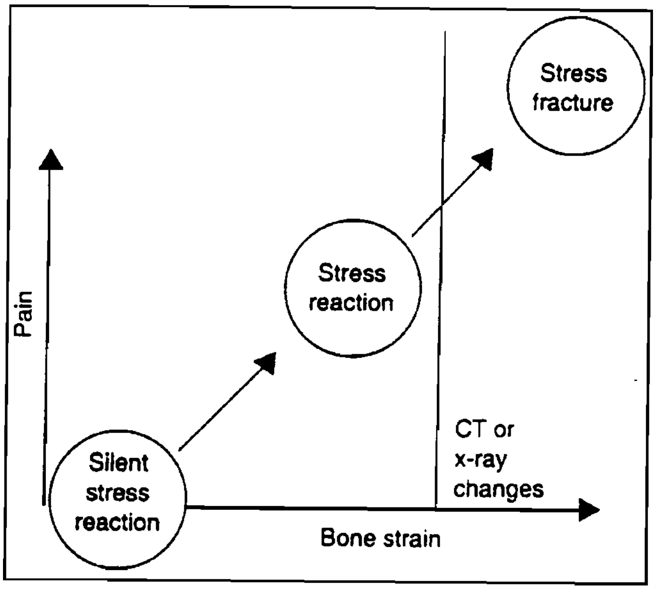

Figure 1. *The continuum of bony stress – from stress reaction through to stress fracture (adapted from Roub et al (1979) and Matheson et al (1987a)).*

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What is the relative frequency of the different tarsal stress fractures?

Stress fractures may comprise as much as 10% of all sports injuries and up to 15% of injuries to runners.[4,20,21] This paper reviews stress fractures that cause tarsal pain: talar, navicular, calcaneal, cuboid and the three cuneiforms as well as the stress fractures of the base of the 2nd and 5th metatarsals. Other metatarsal stress fractures[22,23] and sesamoid and phalangeal stress fractures are not reviewed.[24]

Studies that have reported a sizeable number of stress fractures provide a guide to the relative frequency of stress fractures. In a series of 320 stress fractures in athletes, 81 (25%) were in the foot.[20] Most runners' stress fractures are metatarsal and the frequency of true tarsal bone stress fracture is probably less than 2% of all stress fractures.[25] In a study of 28 stress fractures in élite track athletes there were 12 navicular, 4 talar and 4 cuneiform stress fractures.[26] In a military survey of stress fractures of the lower limb, 367 were in the calcaneus, 17 in the navicular, 9 at the base of the fifth metatarsal, 2 in the talus and one in the cuboid.[27]

Differences in relative frequency of stress fractures between papers result from the nature of the referral population (eg, type of athlete, military population) as well as lack of uniformity in diagnosis of stress fracture (plain Xray, bone scan).

What are the clinical features that suggest the presence of a tarsal stress fracture?

The keys to diagnosis of stress fracture of the tarsal bones are (i) to have a high index of suspicion for the condition, (ii) to know which stress fractures are most common in the particular sport being played and (iii) to understand tarsal anatomy. Pain is the primary presenting symptom of stress fracture. It is worse with running, better with rest, and gradually increases in the absence of reduced mileage or reduced stress.[25] Symptoms start at a low level, gradually becoming more intrusive.[1]

If the diagnosis of stress fracture is suspected, examination aims to detect tenderness at specific sites. In one study 80% of athletes with tarsal stress fracture had point localised tenderness.[20] Another study reported 88% presence of tenderness in stress fracture.[28] Swelling was a much less common sign, being present in 25% of stress fractures in the above studies.[20,28] The importance of accurate localisation of the site of maximum tenderness cannot be overemphasised.[1]

Additional signs such as pain that is worse with ultrasound treatment[20,29] may suggest the diagnosis, but its absence does not rule out a stress fracture.

What imaging should be used to confirm the diagnosis?

For accurate diagnosis in musculoskeletal medicine the sports physician and the radiologist must work together.[29] This is particularly true in the investigation of tarsal stress fractures because the earliest imaging changes are subtle.[30] Plain Xray has been the first investigation for stress fractures but it has low sensitivity.[2] However, it does provide information about the bone structure, for example, the presence of accessory ossicles, osteophytes, tarsal coalition and may exclude other pathologies such as malignancy and infection.[31,32]

The tarsal bones are essentially composed of trabecular bone[33] which reacts differently to stress than does cortical bone. Trabecular bone responds to stress with an increase in sclerosis (Fig 2)[30,34,35] and this is thought to be due to compression causing an increase in transverse callus.[36,37]

Cortical bone such as in metatarsal shafts develops cortical fissures and increased callus of periosteal new bone formation. This is the appearance seen in classic metatarsal "march fracture".[38] This appearance results from the different bone type,[30,39] and distraction causing cortical fissures.[36,37]

The diagnosis of a stress fracture is sometimes delayed because some clinicians adhere to the misapprehension that if Xrays fail to detect a fracture when the patient first presents, a repeat Xray at 10-14 days will generally reveal any stress fracture present.[31] Although this dictum may be the case on occasions, it has been demonstrated in many studies that it is not reliable.[10,30,41,42,43,33,34,35,41,44,46] In two separate longitudinal studies of calcaneal stress fractures, Xray changes occurred between one and 10 weeks after symptoms had commenced.[30,43] Navicular stress fractures have been undetectable on plain radiography as long as 18 months after diagnosis with radionuclide scan and CT scan.[10]

Figure 2. *Horizontal CT scan appearance of stress fracture of medial cuneiform showing increase in sclerosis of trabecular bone.*

Radionuclide scan is the most useful single investigation for diagnosis of stress fracture.[2,12,26,39,47-49,34,35] Although there have been case reports of radionuclide scanning failing to detect stress fractures at certain nontarsal sites at early stages,[42,50] the well performed scan is considered to have 100% sensitivity for bony pathology in the foot. A positive bone scan is not specific for stress fracture - osteoid osteoma, malignancy, avascular necrosis, infection all cause increased uptake.[47,51] Generally, a positive radionuclide scan should be followed by a CT scan to enhance the diagnostic specificity so that the clinician can determine whether the lesion is merely a "stress reaction" or truly a "stress fracture".

The CT scan is a highly specific test and with bone windows optimally set, can provide precise anatomical information.[52] Generally the CT scan will differentiate stress fracture from other pathologies that produce increased uptake on bone scan.


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MRI is a useful diagnostic tool, but is still not readily available in many centres. Moreover, it is relatively expensive compared with radionuclide scan or CT alone. MRI has the advantage over either CT or bone scan alone that it combines anatomical detail with a measure of bone activity.[53] Particularly using the STIR image, bone strain/stress can be detected,[39,53] much as a bone scan can show increased uptake. MRI provides the anatomic detail of CT simultaneously.[54] MRI does not emit radiation, benefiting patients who may need multiple investigations and providing the potential to screen high-risk athletes (eg track athletes, ballet dancers) for specific stress fractures during periods of heavy workload. Thus, one may diagnose a silent stress reaction prior to the athlete having symptoms and thus minimise any time off.

What is the recommended management for each stress fracture?

Management of tarsal stress fractures, and indeed all stress fractures can be simplified by categorising them as either (a) simple or (b) difficult stress fractures according to whether they are likely to have delayed union or nonunion with routine management. Simple stress fractures are those that generally heal without complication when the cause of the stress fracture is removed. Examples of simple stress fractures include fractures of the neck of the fourth metatarsal or the fibula. Patients with simple stress fractures in the lower limb are permitted to continue walking. Simple stress fractures of the tarsal bones discussed in this review are: calcaneal, talar neck, cuneiforms and cuboid. Management consists of correcting any underlying predisposing factors and reducing the precipitating activity[20] (Table 1).

Stress fractures may be "difficult" or "high risk". These difficult stress fractures[25] require more aggressive intervention than merely removal of the offending activity. These stress fractures are prone to nonunion and delayed union.[8] In a study of difficult stress fractures in the entire body, 38% of those that went on to nonunion or delayed union were at foot sites.[8] When navicular stress fractures were treated as simple stress fractures the nonunion rate was greater than 50%; when treated correctly, the data suggests that the successful outcome is about 85%.[55]

TABLE I
SIMPLE STRESS FRACTURES[1]

SIMPLE
Calcaneal
Talar neck
Medial cuniform
Intermediate cuniform
Lateral cuniform
Cuboid

Simple stress fractures are those stress fractures generally requiring management by reduction of activity.

The concept of high risk means that there is a high risk of the stress fracture having complications, generally nonunion and persistent pain;[8] it does not imply that the stress fracture has a high prevalence. Difficult stress fractures may be more difficult to image than simple stress fractures. The difficult tarsal stress fractures are: navicular, base of fifth metatarsal, base of second metatarsal and posterolateral talus. (Table II)

TABLE II
DIFFICULT STRESS FRACTURES[1]

DIFFICULT	MANAGEMENT (see text)
Navicular	6 weeks nonweight-bearing cast immobilisation
Posterolateral talus	6 weeks nonweight-bearing cast immobilisation
Base of second metatarsal	4 weeks nonweight-bearing
Base of fifth metatarsal	Screw fixation

Difficult stress fractures are those stress fractures which have a high rate of nonunion or delayed union unless managed in a specific manner.

Stress fracture of the talar neck

The talus can suffer several bony insults. Talar dome fracture is an acute traumatic injury[56] and beyond the scope of this review. The classic site of talar stress fracture is the talar neck.[30] Recently an unusual stress fracture of the posterolateral process of the talus has also been reported.[57]

Talar neck stress fractures have been reported in runners and in military personnel.[30,58-61] The patients present with pain across the dorsum of the ankle which is aggravated by plantarflexion and dorsiflexion. Tenderness is found around the lateral aspect of the talar neck.[58] When plain Xray is positive it shows an increased linear density through the neck of the talus[30,58] but the diagnosis generally made on bone scan.[60,62]

Treatments reported range from (i) 6 weeks in a weightbearing cast and orthotics,[30,58] through (ii) 3-4 weeks nonweightbearing with a splint[59] to (iii) 4 weeks on crutches but without any immobilisation followed by a further 4 weeks avoiding sporting activity (Khan, unpublished case). Generally, the athlete with this stress fracture is expected to return to sport at 2 to 3 months from the time of diagnosis if managed with restriction of activity. Immobilisation is not necessary.

Stress fracture of the body of the talus

Stress fractures causing posterolateral talar pain and tenderness have been reported in two pole vaulters, a basketballer and a footballer. The stress fracture has a characteristic, comma-shaped bone scan appearance and it is clearly evident on CT.[57] Differential diagnosis in that sites includes subtalar joint stiffness and osteoarthritis. From the small series reported, it would appear that 6 weeks nonweightbearing cast immobilisation is the treatment of choice (Bradshaw, personal communication).

Calcaneal stress fracture

This is an important stress fracture in the military, usually occurring in recruits undergoing induction.[1] Patients complain of posterior heel pain. Diffuse swelling of the heels extends superiorly to obliterate the malleoli. With early diagnosis this swelling is less marked.[40,43,63-65] Fracture is virtually always confined to the posterior half of the cancellous bone.

Treatment for calcaneal stress fracture has included bed rest for one week, four-point weight bearing with crutches, half inch sponge and rubber heels in both shoes. Crutches were discontinued at 4-6 days when symptoms permitted. All patients returned to duty in 8 weeks, although some still had some swelling and pain, but these features were insignificant compared with the original symptoms. There was no displacement of any of the fractures.

The military tendency to "dig in" may contribute to the development of these stress fractures. "Digging in" is a method of marching with one or other heel being slammed down on the macadam of the drill fields. In addition, the new recruits must walk "double time" everywhere during the first two weeks of training and is on his or her feet for 8-10 hours per day.[43] As calcaneal stress fracture is not a "difficult" stress fracture it should be managed symptomatically.

Navicular stress fracture

Stress fracture of the tarsal navicular bone has curtailed many promising athletic careers for a number of reasons.[66] The athlete with early navicular stress fracture presents with vague pain which is generally not well localised,[3,67-69] plain film imaging has extremely low sensitivity[70,71] and patients

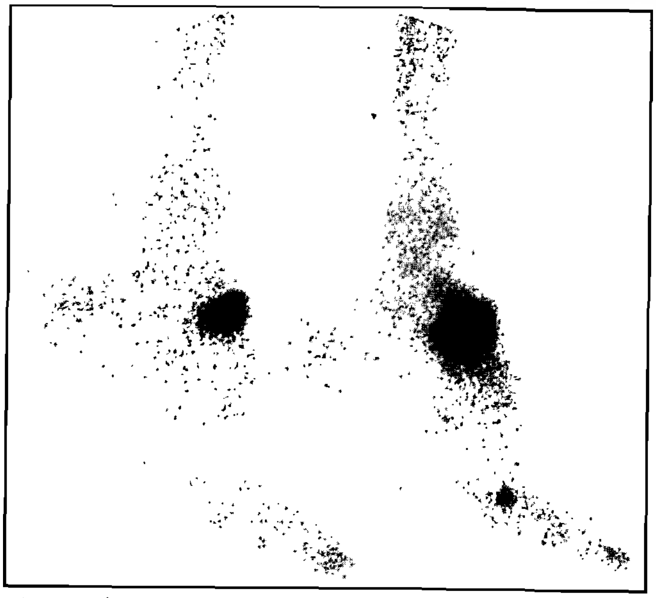

Figure 4. *Bone scan appearance of both feet of an athlete with bilateral navicular stress fracture.*

and doctors often feel reluctant to undertake the proven, but inconvenient treatment method of nonweightbearing cast immobilisation.[10,70] All of these factors contribute to the high incidence of delayed union and nonunion of this fracture.[8]

If a patient, usually a sprinter or multi-event athlete, presents with midfoot pain, palpation of the 'N-spot', the proximal dorsal point of the navicular in the midline reveals tenderness in stress fracture[10] (Fig 3). This site corresponds with the location of the fracture.[52,72,73]

Investigation requires radionuclide scan and CT scan. Techniques to improve the sensitivity of plain radiography in navicular stress fracture have been reported[71] but even with this technique most small stress fractures remain undetectable.[10] Radionuclide scan in navicular stress fracture generally reveals focal increased uptake throughout the entire bone (Fig 4). The CT scan

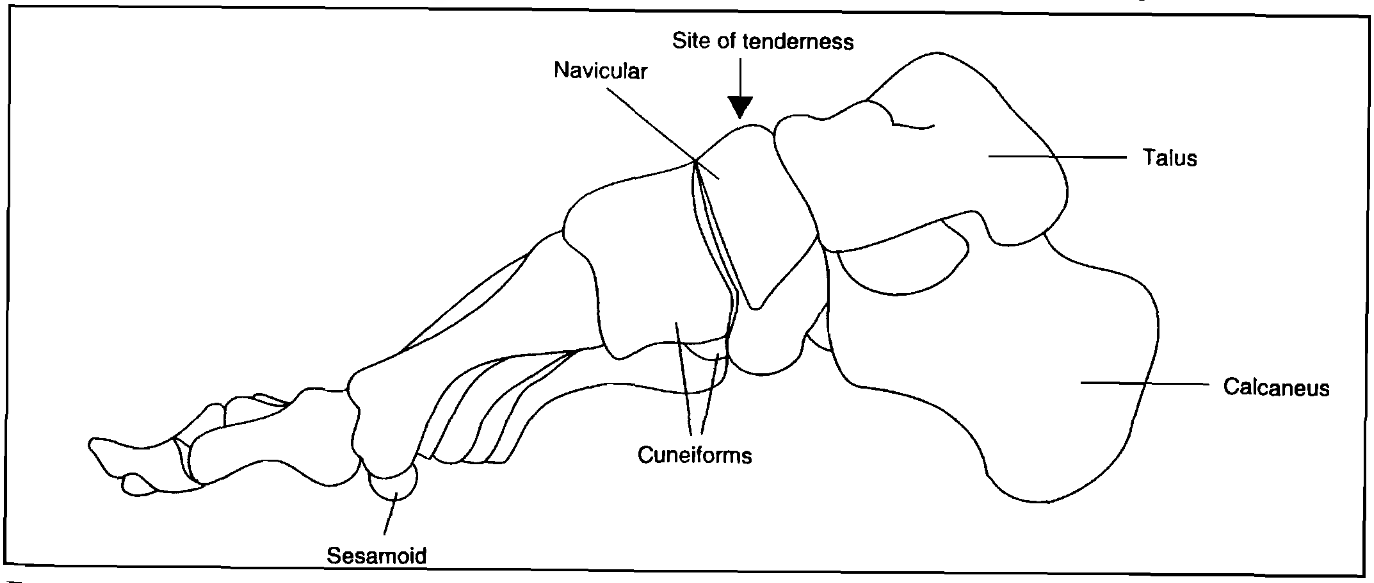

Figure 3. *Location of 'N-spot', the site of tenderness in navicular stress fracture.*

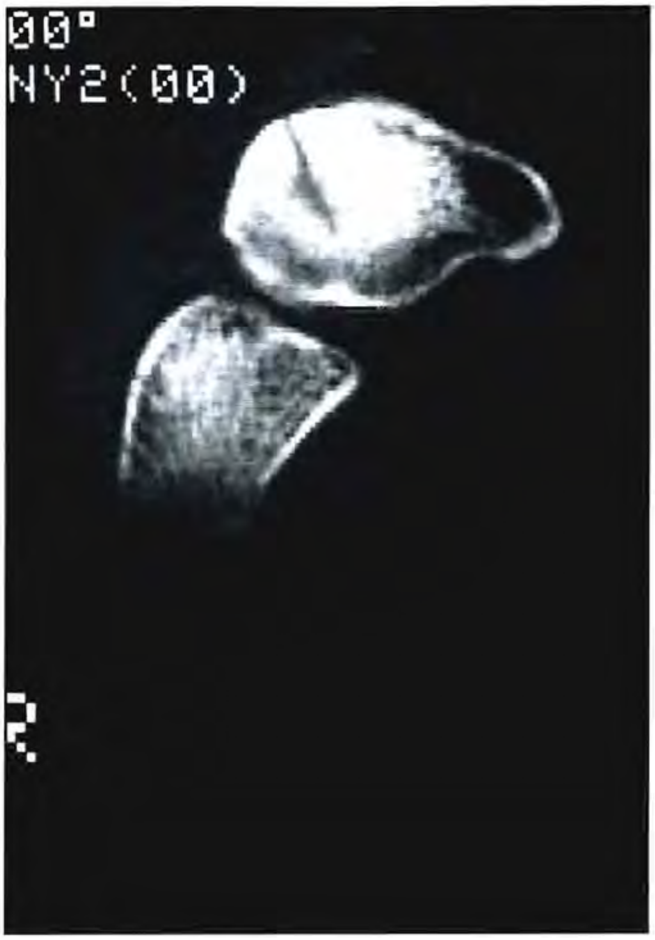

Figure 5. *Axial CT scan appearance of stress fracture of the navicular showing the characteristic saggital cortical defect extending inferiorly from the 'N-spot'.*

appearance of navicular stress fracture is characteristic - a sagittal defect in the middle third of the bone extending distally and inferiorly from the 'N-spot' at the proximal navicular (Fig 5).

In a review of 142 cases of navicular stress fracture in the literature, the success rate of nonweightbearing cast immobilisation was 89%, while that of weightbearing rest (limitation of activity) was just 24%.[55] Because 6 weeks of nonweightbearing cast immobilisation is an inconvenient treatment, the clinician must take time to explain the risk of nonunion in order to encourage compliance.

Joint stiffness, muscle tightness and nerve tethering that results from plaster cast immobilisation requires high quality post-cast rehabilitation.[1,55] Podiatry assessment is necessary. Successful navicular stress fracture management requires the team approach![74]

Stress fracture of the cuneiform bones - medial, intermediate and lateral

The first reports of stress fractures in each of these bone were in the military.[37,75,76] In each case the patient presented with midfoot pain; localisation of the site of stress fracture by palpation is the key. The case reports illustrate the increased sclerosis typical of stress fracture in cancellous bone[37,75,76] but in today's practice, radionuclide scan is generally required to make the diagnosis. Changes on CT scan or plain films changes may become evident at a later stage.

One case of medial cuneiform stress fracture in an athlete was documented with bone scan, CT and MR. This recreational athlete presented with medial foot pain and tenderness over the medial aspect of the bone. The patient recovered in three months by ceasing running without requiring any significant immobilisation.[77] Two cases of stress fracture of the medial cuneiform bone are reported in the military literature.

A plumber who commenced military training developed intermediate cuneiform stress fracture, responded to several weeks rest in bed treatment and was pain free after 8 weeks.[76] In an athlete where diagnosis was delayed several months, surgical treatment has been reported.[78]

Two cases of lateral cuneiform stress fracture both made uneventful recoveries at about one month after onset of symptoms.[10,37]

Cuboid stress fracture

Cuboid stress fractures are exceedingly rare. There are only a few published cuboid stress fracture case reports[28,41,79,80] compared to those involving a bone such as the navicular where over 150 cases have been reported.[55] Athletes with cuboid stress fractures present with lateral foot pain and tenderness on the dorsal surface of the bone. Cuboid stress fractures require standard simple stress fracture management. Because these are such rare stress fractures, it behoves the clinician contemplating the diagnosis to seriously consider differential diagnoses such as cuboid syndrome,[81-83] before making the diagnosis of stress fracture.

Base of second metatarsal stress fracture

The pathology in this stress fracture is an oblique fracture of the proximal portion of the second metatarsal involving Lisfranc's (second tarsometatarsal) joint. At this joint the second metatarsal articulates with all three cuneiform bones. Stress fracture of the base of the second

metatarsal is a common stress fracture in dancers,[84] presumably because the first two metatarsals bear most of the bodyweight in demipointe and pointe work of ballet.[85,86] This stress fracture is occasionally seen in track athletes. It is felt that a short first metatarsal or a long second metatarsal (Morton's foot) may be a risk factor for this specific stress fracture although it is not a risk factor for metatarsal stress fractures in general.[87]

The dancer or athlete typically presents with gradual onset of soreness in the mid foot and later pain becomes evident with pointe and demipointe work.[84] Examination reveals tenderness at the base of the second metatarsal that does not respond to passive joint mobilisation. Investigation requires radionuclide scan and CT or MRI as the injury is not evident early on plain radiography.[84] Although cast immobilisation has been reported for treatment of this stress fracture after delayed diagnosis[88] a more recent report recommends activity modification for 6-8 weeks without immobilisation[84] as long as the diagnosis has been made promptly. Those authors recommended that for the first 4 weeks of treatment the dancers used crutches to unload the second metatarsal.

Stress fracture of the base of the fifth metatarsal

Three different fractures around the base of the fifth metatarsal need to be distinguished because each requires different treatment. There are two acute fractures and one stress fracture. The acute avulsion fracture of the base of the fifth metatarsal occurs commonly and usually requires symptomatic treatment only, even if the joint surface is involved.[89,90] The acute fracture of the diaphysis, which Sir Robert Jones himself sustained while dancing around a tent at a military party in England,[91] requires 6 weeks nonweightbearing cast immobilisation.[9,90,92,93]

The stress fracture of the base of the fifth metatarsal, probably should not be called a Jones' fracture as it is not the injury sustained nor reported by Jones and it needs to be managed differently to the acute Jones' fracture.[94] The stress fracture has a high rate of delayed union and refracture when managed conservatively, particularly in basketballers.[90,93,95] This stress fracture can present with sudden onset of symptoms,[9,90] therefore the proximal fifth metatarsal must be scrutinised for radiological evidence of medullary sclerosis, lateral cortical hypertrophy and beaking[96] as this suggests that conservative management is less likely to be successful. Surgical treatment of stress fracture of the base of the fifth metatarsal is by intramedullary curettage and either screw fixation or bone graft.[97] Although some surgeons advocate bone graft[9] screw fixation provides a more rapid return to sport.[94,98]

When is the fracture healed?

Clinical features, particularly loss of tenderness at the fracture site, are the best guide to stress fracture healing (Fig 6). Plain radiographs are often not sufficiently sensitive to detect the original tarsal fracture. Although signs of the fracture (sclerosis in the case of trabecular bone, cortical defects in the case of cortical bone) may develop with time, plain radiographs are rarely useful in monitoring fracture healing. In fact, when a fracture is evident in cortical bone on Xray at diagnosis there may well be a period of apparent increase in the fracture line width during a period of immobilisation due to demineralisation and resorption associated with fracture healing.[70]

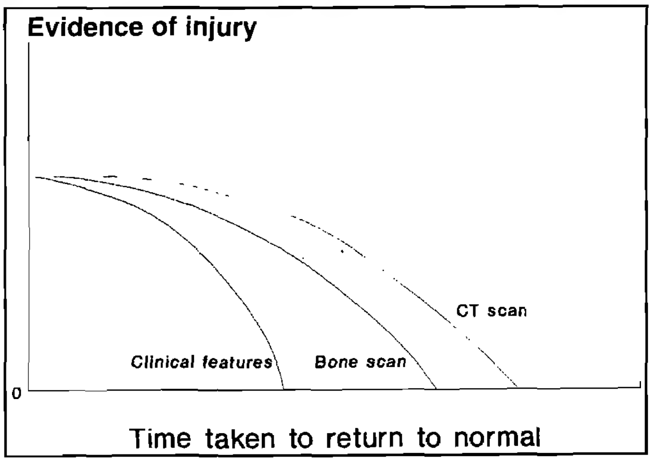

Figure 6. *Diagrammatic representation of the different rate of clinical features and imaging modalities to return to normal after stress fracture.*

Radionuclide scan is not useful for monitoring fracture healing as the scan remains positive well in excess of 6 months after clinical union.[48] CT scan will not necessarily show complete obliteration of the fracture line after stress fracture even though the patient is asymptomatic.[10] Similarly it has been shown that decreased T2 signals persist on MR after fractures have healed clinically.[39]

As a general rule, after stress fractures at any site, not just the tarsal bones, routine imaging is not indicated and clinical examination of point tenderness should be used to monitor a graded return to sports.[10] In particular, post-immobilisation CT appearances should not be used as an indication for surgery for nonunion in any asymptomatic patient.[10,52] Thus the clinician must have a good understanding of the sites of tenderness in stress fractures eg, the N-spot in navicular stress fracture[10] and comparable sites in other bones.

After periods of rest and particularly after immobilisation, joint stiffness can cause pain and this must not be confused with pain from the fracture. Knowledge of the anatomy of stress fracture sites and excellent palpatory skills should enable differentiation of the two conditions. In addition a trial of mobilisation can often permanently remove pain resulting from joint stiffness, whereas pain from delayed union would persist or return quickly after joint mobilisation.

Summary

Seven practices to ensure successful stress fracture management

1. Have a high index of suspicion as early symptoms can be slight.
2. Careful palpation is very important - focal bony sites of tenderness requires investigation.
3. Understand the limitations of Xrays - and proceed to radionuclide scan without hesitation if clinical suspicion of stress fracture exists.
4. Develop good rapport with a sports radiologist - the ability to diagnose may be limited by inappropriately taken or incorrectly interpreted films.
5. Instigate appropriate treatment of difficult stress fractures rather than hoping limitation of activity will be enough.
6. Use clinical features to monitor healing and subsequent return to sport.
7. Use the team approach to rehabilitation - the biokineticist, physiotherapist all have essential roles to play.

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Exercise and bone mass in mature premenopausal women

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Introduction

The adult skeleton has completed longitudinal growth, but is still metabolically active and constantly undergoing remodeling.[1] Bone remodeling is a combination of the sequential processes of activation, resorption, formation and quiescence. Bone may be resorbed as part of the normal physiological functions of maintaining plasma mineral homeostasis, repairing microfractures or altering bone shape in response to mechanical demands.[2] Bone turnover rate is approximately 15 % per year, but is higher in trabecular bone (20 %) than in cortical bone (5 %) at any particular time.[3] A positive bone balance exists when bone formation rate exceeds bone resorption rate and is particularly high during adolescence,[4] whereas nett bone loss is a result of increased bone resorption rate without a concomitant increase in bone formation rate.

It is generally accepted that bone loss occurs with increasing age and that menopause is responsible for a period of accelerated bone loss in women and places them at a higher risk for subsequent osteoporotic fractures. Indeed, the cumulative loss of vertebral BMD from early adulthood to late old-age may be as high as 47 % in women compared with only 14 % in men.[5] However, it is not only the rate of bone loss which may influence fracture risk, rather it has been hypothesized that postmenopausal osteoporosis may result partly from a relatively low peak bone mass in women compared with men.[6] Data from both Riggs et al (1981)[5] and Seeman et al (1989)[7] support the hypothesis that a relatively low vertebral BMD in young adulthood is an independent risk factor for a relatively low vertebral BMD in later life. Also, a longitudinal study on 521 female volunteers over a 15 year span has shown that a single bone mass measurement can predict the probability of hip fracture over that time period.[8]

Peak adult bone mass

The data discussed above highlight the importance of achieving and maintaining a high peak adult bone mass in order to reduce the risk of spontaneous fractures in later life. However, there is controversy about when peak bone mass is achieved, when bone loss begins and what factors affect the peak bone mass attained.[9,10] Some data have shown no significant increases or decreases in bone density between 20 and 40 years of age, and suggest that a peak is reached earlier than previously believed.[10,11,12] In contrast other research has suggested that the average premenopausal female may be able to increase her lumbar spine BMD by as much as 10-15 % during the years preceding menopause.[13] Still other evidence suggests that bone loss can occur in premenopausal women prior to the disruptions in ovarian function which indicate the onset of menopause.[9]

These conflicting findings are likely due to the fact that peak bone mass, the age at which bone loss begins, and the rate of bone loss are controlled both by inheritance and a variety of lifestyle factors. A large genetic component appears to determine the maximum bone mass achievable, however several nutritional and lifestyle factors will determine whether an individual is able to achieve this level or not. These factors include, amongst others, current menstrual status and menstrual history, habitual levels and type of physical activity, dietary calcium intake, lactation and parity, and oral contraceptive use. This review will discuss the current status of knowledge on these issues.

Influence of genetics on peak bone mass

Genetic potential has a relatively higher impact than environmental factors in the development of premenopausal bone mass.[11] Peak bone size, bone mass, and bone density in young women are all strongly influenced by genetic information not only from the mothers but also from fathers.[14] Matkovic et al (1990)[14] found a resemblance between the mean bone size, bone mass, and bone density of the appendicular and axial skeleton of both the parents and that of their daughters, and concluded that by the age of 16 years, daughters had accumulated 90-97% of the bone mass of their premenopausal mothers. This relationship seems to hold for older generations as well, since another study reported a significantly lower lumbar spine BMD in the premenopausal daughters of osteoporotic mothers compared to women of the same age whose mothers did not have osteoporosis.[7]

However, it has been hypothesised that there is an interaction between genetic and lifestyle factors which influence bone and, in particular, that

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negative lifestyle factors will prevent the accrual of the genetically determined potential peak bone mineral density.[15] Also, lifestyle factors may influence the age and rate at which bone loss begins, or predispose to an episode or several episodes of premenopausal bone loss.

Does either exercise training or regular physical activity, or both, influence bone?

It is unknown whether bone loss in athletic women follows a different trend relative to the general population. Studies with relatively small numbers of subjects, suggests that physical activity may prevent age-related premenopausal bone loss.[16,17] Indeed "age-related premenopausal" bone loss may be a misnomer. Rather premenopausal bone loss may be simply due to an age-related decline in physical activity. Brewer et al (1983)[16] concluded from their study comparing marathon runners to sedentary controls that bone mineralisation may be enhanced in premenopausal middle-aged (30-49 years of age) women who participate in moderate to intense exercise, with an inferred consequence being a lower risk of fracture later in life. Several studies have found that athletes involved in weight-bearing activities, such as running, volleyball and basketball, have higher bone densities than non-athletes.[16,18,19] However, a contentious issue is still whether women with relatively higher BMD choose to participate in regular physical activity or whether their higher BMD is a result of their exercise habits. Although Smith and Gilligan (1987)[20] concluded from their review that weight-bearing activities such as walking, running, and racquet sports are more effective in maintaining integrity of the neck of the femur and the spine than nonweight-bearing activities such as bicycling and swimming, only longitudinal studies using exercise as intervention will be able to definitively solve this issue. Even then, variables which may affect the outcome of intervention studies will include e.g. previous habitual exercise patterns and starting level of BMD.

The effect of physical activity on bone mass is not only relevant to the athletic population. Rather the effects of the normal range of activity most likely to be undertaken in daily living and leisure time is possibly even more important, since the athletic population is relatively small. A study by Kanders et al (1988)[13] on a sample of non-athletic women with a stable lifestyle showed a highly significant correlation between BMD in the lumbar spine and overall level of physical activity. A similar study by Halioua (1989)[21] investigated lifetime physical activity habits of a group of healthy premenopausal women (n=181, 20-50 years of age) and found significant relationships between lifetime physical activity habits and BMD and BMC of the distal radius and the mid-radius. They concluded that good exercise habits during the formative years of adolescence and early adulthood should maximise the genetic potential for bone mass of the individual.

Although these cross-sectional studies can lay scientifically sound foundations for hypotheses, controlled intervention studies are required to substantiate them. Most intervention trials to date have been done on postmenopausal women. For example, Dalsky et al (1988)[22] found that in a group of healthy, sedentary, postmenopausal women who completed a 9-month programme of weight-bearing exercise, BMC of the lumbar spine increased significantly. The increase in lumbar spine BMC was maintained with a longer term program. However, after a 13 month period of detraining, mean BMC had returned to just above baseline. Although these data were collected in postmenopausal women, they suggest that the skeleton is indeed responsive to physical activity, but that the level of activity should be maintained or the benefit will be lost. Snow-Harter et al (1992)[23] conducted an 8 month intervention trial on a group of 31 healthy, premenopausal women (mean age 19.9 years) who were randomly assigned to a control group, or to progressive training in jogging or weight-training. The study found a significant increase in lumbar spine BMD in the runners and weight-trainers, in comparison to the control subjects in whom BMD did not change. There was no significant difference in lumbar BMD between the two exercising groups. To our knowledge no controlled exercise intervention trials have been done in women between 30 and 45 years of age.

The relationship between body mass and bone mass

If weight-bearing activity is such an important determinant of BMD it seems logical that body weight may also play a role in the attainment of peak bone mass, either as a function of a larger skeleton or in conjunction with weight-bearing exercise. Different studies have noted a positive relationship between several parameters of body stature and bone mineralisation,[16,24,25,26,27] indicating that larger women do indeed have greater bone mass. Conversely, women with smaller stature have lower bone mass. A study by Drinkwater et al (1990)[28] in athletic premenopausal women aged 18-38 years, found body weight to be a significant predictive variable for bone density at all of seven sites measured, including lumbar spine, femoral neck and shaft, tibia, fibula, and the distal radius and shaft of the radius. The authors suggested two other possible explanations for this relationship. Firstly, the skeleton will respond to the stress placed on it by the additional body mass, and secondly, there is increased conversion of androgens to estrone in the adipose tissue of the heavier women. But body weight predicted BMD of weight-bearing as well as non weight-bearing sites in this study, indicating that the relationship is more complex than a simple gravitational response. A strong positive correlation between body weight and BMC has also been shown in a group of perimenopausal women.[6] However, these authors suggested another potential variable which may explain this association: they hypothesised that there may be a later onset of menopause in women with a higher % body fat. It is therefore clear that the effect of body weight on BMD is multifactorial.

Muscle mass/strength

Similar to the positive relationship between body mass and bone mass, a positive correlation has been demonstrated between muscle mass and bone mass.[29] This may be due to the relationship between body mass and muscle mass and therefore merely another method of relating body size to bone mass. However cross-sectional studies showing higher BMD in weight-lifters than in other athletes, suggest that weight-training may provide a better stimulus for improving bone status than running or swimming.[17,30,31] In a group of regularly menstruating female resistance- and endurance-trained athletes, fat-free body weight was the best predictor of bone mineral content.[30] Therefore the relationship between muscle mass and bone mass may be due to skeletal loading during weight-training or other activities that cause muscle hypertrophy. Indeed, bone remodeling has been shown to occur in response to site-specific mechanical loading.[17,32] For example, Davee et al (1990),[17] showed that lumbar BMD was greatest in women who supplemented their aerobic exercise with muscle-building activities, including arm, leg, chest and back exercises. They concluded that exercise affects bone remodelling by working at the site where mechanical loading is maximal. However, Snow-Harter et al (1990)[33] concluded that although muscle strength is the most significant independent predictor of BMD and may account for 15-20 % of the total variance in bone density in young women, it is more complex than a simple site-specific relationship between the muscle attachments and the bone. In that study biceps strength was an independent predictor of BMD at the hip, and grip strength best predicted lumbar spine density. It is unknown whether this is due to a systemic hormonal effect of exercise, or whether the biceps curl is a specific exercise which is most representative of exercise habits influencing the whole body.

Menstrual status and reproductive history in premenopausal women affects bone mass

a. *Menstrual status: current and history*

The rapid loss of BMD following menopause illustrates the fact that normal reproductive hormonal status is one of the most important requirements for maintenance of bone mineral density, and consequently is highly likely to influence the attainment of peak adult bone mass. Research in premenopausal women has found that estrogen status (determined by a combination of age at menarche, average length of menstrual cycles since menarche, use of birth control pills, and circulating estrogen levels) is the most important determinant of bone mass in Caucasian women.[11,12] Values within the normal range for these factors, along with age and previous pregnancy, are determinants of reproductive maturity and have been shown to be protective against development of menstrual dysfunction in premenopausal women.[34] Women athletes are a population at risk of developing menstrual dysfunction,[35,36] particularly those participating in sports which place emphasis on leanness.[37] Since one of the important potential benefits of regular physical exercise is increased bone mineral density,[19,38] it is incongruous that the beneficial effects of exercise are negated or reversed in those athletes who experience menstrual cycle disruption and hormonal deficiencies.[39-41] Many studies have identified a significant, direct relationship between current menstrual status and bone mineral density in sedentary and exercising subjects.[18,26-28,39,41-47] In summary, currently amenorrheic athletes (usually defined as 3 or fewer periods per year) have lower spinal BMD than currently oligomenorrheic athletes (4-9 periods per year), who in some, but not all studies. also have lower BMD than exercising eumenorrheic athletes. The significance of this bone loss has been highlighted in a study by Myburgh et al (1990)[48] which showed that athletes with stress fractures had significantly lower lumbar spine BMD, a higher incidence of current menstrual irregularity and a lower incidence of oral contraceptive use than the age- and exercise-matched control athletes who had never had a bone injury.

It is important to note that not only amenorrhea is associated with low BMD. Many female runners who may be classified as eumenorrheic, due to a regular number of cycles per year, may be experiencing short luteal phases, anovulation or both.[49] A study by Prior et al (1990)[50] on 66 premenopausal (21-42 years) women of varying activity levels, found that the inadequate production of progesterone, which occurs in cycles of short luteal phases and anovulatory cycles, was associated with accelerated bone loss, despite normal production of estradiol and regular menses. Also, Lloyd et al (1988)[12] showed that even moderate oligomenorrhea (6-7menses/year) is sufficient to cause reduced BMD.

Some cross-sectional studies have also looked in more detail at the overall history of menstrual dysfunction instead of focussing only on the current status.[28,43,47,51] Drinkwater et al (1990)[28] found a correlation between a score (category 1-9) derived from combining current and previous menstrual status and current BMD of the lumbar spine with those subjects who scored 1 (always regular) tending to have the highest BMD and those subjects scoring 8 (current amenorrhea with a history of oligomenorrhea) and 9 (primary amenorrhea) tending to have the lowest BMD. Interestingly, a study by Myburgh et al (1993)[47] which calculated the number of years of amenorrhea, the number of years of regular menstruation and the estimated total number of periods missed found that the most robust predictor of lumbar spine BMD was the number of years of regular menstruation. In that study no statistical correlation was found between lumbar spine BMD and years of amenorrhea or total periods missed since menarche. Their explanation for this was that the BMD deficit noted in amenorrheic women may not necessarily only reflect bone loss, but may instead also be due to inadequate bone gain during adolescence and early adulthood. Other methods of quantifying menstrual history have estimated the overall average number of periods per year, so that years of regular menses are taken into account.[43,51] The calculated menstrual history indexes corroborate the findings of both Drinkwater et al (1990)[28] and Myburgh et al (1993).[47]

It is therefore clear that women athletes with menstrual irregularities are at risk of not achieving their peak adult bone mass due to a combination of factors relating to their current and previous menstrual status.

b. *Trabecular bone vs cortical bone*

Several studies have found a significant difference in trabecular bone density between regularly menstruating athletes and athletes with secondary amenorrhea, with no difference in cortical bone mass between the two groups.[18,39,42,44,46] Brewer et al (1983)[16] have stated that bone mineral deficits are greater in trabecular bone than in compact bone due to the greater surface-to-volume ratio in trabecular bone. The bone surface is exposed to the hormonal milieu and similar to the hypoestrogenic state associated with menopause, lower circulating estradiol levels have been found in amenorrheic young women compared to women with regular menstrual periods.[39,42,44] A positive linear correlation between lumbar spine BMD and serum estradiol[44] supports this hypothesis. A study by Young et al (1994)[25] concluded that predominantly cortical, weight-bearing regions will benefit more from weight-bearing exercise and suffer less from hypogonadism. Their explanation for this was that cortical bone may be more responsive to loading as it is normally adjacent to, or receives, direct muscle insertions while trabecular bone is more central and encased by cortical bone. However, cortical bone may be less sensitive to estrogen deficiency than trabecular bone because it has a lower inherent turnover rate. Conflicting results from a study by Myburgh et al (1993)[47] found that cortical bone is not necessarily protected from mineral loss as BMD of the femoral neck (where the proportion of cortical bone may be as high as 50%) was significantly lower in the amenorrheic group than the regularly menstruating group, despite similar body weight.

c. *Age at menarche*

Delayed menarche and training at a young age may also contribute to subsequent development of amenorrhea and a higher incidence of bone injuries, as some studies have shown a later age at menarche in amenorrheic runners.[26,28,44,46,47,52] Marcus et al (1985)[18] showed that the amenorrheic athletes in their study had commenced training within 1 year of menarche, whereas the cyclic athletes did not begin training until around 5 years later. These findings may be explained by the hypothesis that intensive endurance training affects the immature hypothalamic-pituitary function more than it would after complete maturation.[18] Delayed menarche in a normal, non-athletic population of women may also result in reduced BMD as revealed in the results of a study by Armamento-Villareal et al (1992)[11] which found an inverse relationship between vertebral bone density and age at menarche, and concluded that reaching sexual maturity earlier in life will increase the exposure of the skeletal tissue to the beneficial effects of estrogen. Similarly Bachrach et al (1990)[53] have shown that BMD is highly significantly lower in amenorrheic anorexic teenagers than in age-matched controls. Therefore both adequate nutritional status and adequate estrogen is important for bone mineralisation during the rapid growth phase.

d. *Resumption of menses*

It is apparent that young women with a history of irregularity generally have lower BMD than age-matched controls. In a study by Grimston et al (1990)[51] runners were grouped according to their menstrual history. Those runners reporting a history of menstrual irregularity at any time had a significantly lower lumbar spine BMD than those runners with a history of menstrual regularity. The question is whether the resumption of normal menses in these women is sufficient to counterbalance the negative effect of transient hypoestrogenism within the fertile period.[11,38] Drinkwater et al (1986)[40] have shown that after resumption of regular menses, previously amenorrheic athletes show a significant increase in lumbar spine BMD over a 15 month period, with no change in the cyclically menstruating women over this period. However, the level attained was still significantly lower than control athletes. The resumption of menses in this group of previously amenorrheic runners coincided with a decrease in mileage due to injury or illness, and the substitution of running with other activities. There was also a significant increase in body weight during the 15 month period and five of the former amenorrheics reported an increase in consumption of dairy products and/or calcium supplementation over the previous year. Similarly, research by Lindberg et al (1987),[54] also over a 15 month period in which previously amenorrheic athletes took supplemental calcium and reduced their running distance, showed that along with an increase in body weight, increased estradiol levels and eumenorrhea, there was a 6.6% increase in lumbar spine bone density. Our study[43] in somewhat older women showed that any history of menstrual irregularity was more predictive of BMD than was current menstrual status, indicating that BMD may never be regained in sufficient quantity to match women who have always been eumenorrheic. If these data are repeatable, it is unknown why BMD does not recover to normal premenopausal levels.

e. *Oral contraceptive use, lactation history and parity*

Other factors related to reproductive history such as use of oral contraceptives and parity may have a positive influence on peak adult bone mass, although a long history of lactation may be a negative influence. Although most studies have found no relationship between the use of oral contraceptives and BMD,[24] oral contraceptive use in women athletes has been shown to protect against the development of stress fractures and other musculoskeletal injuries,[48,55] as well as early age-related bone loss[56] and may therefore have an effect on achieving peak bone mass. It is still uncertain however whether oral contraceptives used specifically to correct menstrual irregularities will improve bone mass or merely prevent bone loss. In a study by Lindquist et al (1981)[6] on women from three different age strata (46, 54 and 62 years), there was no relationship between parity and bone mineral content.

Sowers et al (1993)[57] found that women who breast-fed for more than 6 months have reduced BMD of the lumbar spine and the femoral neck. This is in contrast to research by Koetting and Wardlaw (1988)[58] who found no association between a history of long-term lactation and low bone density in the lumbar vertebrae and the femoral neck. However, this discrepancy in results may be explained by the fact that BMD of the lumbar spine has been shown to return to baseline levels within 12 months of stopping breast-feeding.[57] The return of BMD to baseline levels may also be influenced by dietary calcium intakes. Athletic women tend to have paradoxically low energy intakes and some studies have also documented low intakes of calcium.[44,48,59] There is as yet little information regarding the combination of breast-feeding and dietary habits of mature athletic women.

f. *Training and menstrual function*

A relationship has been found between distance run, ie. mileage per week, and chronic menstrual dysfunction.[28,35,39-41,45] A study by van Gend and Noakes (1987)[60] found that all of the amenorrheic and 3 of the oligomenorrheic women in their study were running over 75 km/wk, while no one who ran under 35 km/wk experienced any changes in their menstrual pattern. Other studies have not found a relationship between menstrual irregularity or amenorrhea and weekly mileage,[36,44,47] however a study by Schwartz et al (1981)[35] found that the group of amenorrheic runners had been running for a longer period of time than those runners with regular cycles. Running mileage should however not be considered in isolation as chronic menstrual dysfunction may be the result of a combination of lifestyle factors associated with running, such as sustained heavy training, frequent intensive competition and chronic low body weight due to a kilojoule-restricted diet. Indeed, Myburgh et al (1992)[61] have shown that an athlete's risk of menstrual dysfunction increases with the presence of more risk factors and that no single risk factor is more important than another. Therefore those factors which promote menstrual dysfunction may also indirectly promote bone loss and prevent the achievement of the genetically predetermined potential peak adult bone mass.

Nutrient intake

As stated above, women athletes report a reduced kilojoule intake relative to their high energy expenditure.[18,44,60] Our previous research has found a significant relationship between total energy intake and the Menstrual History Index, a measure of menstrual irregularity, and therefore suggests that there is a relationship between current energy intake and the history of menstrual dysfunction which may be related to a history of chronic undernutrition.[43] Results of a study by Drinkwater et al (1984)[39] showed a daily energy intake of 1623 kcal in a group of amenorrheic athletes compared to 1965 kcal in their control group of eumenorrheic athletes. Thus, previous and current energy intake, specifically in women athletes, may also be indirect determinants of peak bone mass.

Adequate calcium intake during the adult years is necessary to maintain calcium balance and a strong skeleton and therefore calcium deficiency contributes to bone loss.[2] Not only current calcium intake but also the history of calcium intake, ie. during adolescence and in the early 20s, is important in the attainment of peak adult bone mass.[14,21] Calcium intake may also be particularly important in exercising adults since Myburgh et al (1990)[48] have shown that athletic subjects (men and women) with stress fractures had a significantly lower current intake of dietary calcium and also a lower estimated intake of dairy products since leaving school, than did control athletes who had never had a bone injury. Even shin soreness without frank tibial stress fractures have been associated with low dietary intakes of calcium.[59] However it would appear that the relationship is not simple. A cross-sectional study by Kanders et al (1988)[13] on 60 premenopausal women, with stable endocrine status, found that although there was not a significant linear relationship between lumbar spine BMD and calcium intake, when the sample of women were divided into those with a calcium intake above the RDA (800 mg/day) and those with a calcium intake below the RDA, both vertebral and radial BMD were significantly greater in the high calcium intake group. Although Grimston et al (1990)[62] found that current calcium intake was not shown to be related to current BMD, they did find a positive relationship between estimated history of calcium intake during the formative years of bone growth, and current BMD of the lumbar spine. Calcium requirements increase with the onset of puberty at which time calcium intake is often reduced,[63] placing the individual at risk of inadequate calcium retention resulting in reduced peak bone mass and the subsequent risk of developing osteoporosis in later life.

It is important to remember however that effective calcium intake is not only a result of adequate calcium in the diet. A study by Heaney and Recker (1982)[64] found that the most prominent determinant of calcium balance was calcium absorption, with dietary calcium intake being the next prominent factor. Thus, factors such as absorption efficiency, retention efficiency, various nutrient-nutrient, drug-nutrient, and disease-nutrient interactions all affect the availability and utilization of the calcium taken in the diet.[63] Therefore, in healthy women excessive intake of certain nutrients such as nitrogen, phosphorus and caffeine, as well as the abuse of certain drugs such as diuretics, antacids and alcohol, may impact negatively on absorption.

Some research has found that a high protein intake in excess of dietary need may enhance bone loss through increased urinary calcium excretion due to reduced retention of absorbed calcium.[63] There is also a strong relationship between caffeine consumption and bone mineral density, as a high caffeine intake has been found to increase the risk of osteoporotic fractures in middle-aged women as a result of increased urinary excretion.[65] Other dietary factors that may influence calcium balance include nitrogen and phosphorous.[64] Large amounts of fibre have also been found to have an affect on calcium balance, however it is unlikely that the fibre content of most Western diets is sufficient to cause negative calcium balance, however some individuals may be at risk.[63]

There appears to be an interaction between exercise and calcium intake, since exercise training in the presence of low calcium intake, may minimise the adaptation of the bone to exercise training.[2] In contrast, adequate levels of both will result in maximal bone mineralisation, which will apparently not be increased further by even higher calcium intakes or excessive physical activity levels.[21]

Conclusion

In conclusion, it is clear that many factors influence peak adult bone mass, but that in premenopausal women, as in postmenopausal women, estrogen deficiency is the major factor. Some risk factors can be manipulated by either dietary intervention, altered training or estrogen replacement therapy. However, the highly competitive amenorrheic premenopausal athletes may be resistant to reducing training and gaining weight to regularise their menstrual cycles. The current data would suggest that their only other alternative is to take estrogen replacement therapy and this should be strongly recommended at an early stage since it is still unknown whether or not bone loss due to prolonged menstrual irregularity can ever be fully regained prior to menopause. Until data is available to definitively show that women in their late premenopausal years who had menstrual irregularity in their twenties, can regain their bone mass, it must be assumed that such women are at risk of developing osteoporosis in later life. However, the data discussed in this review would suggest that regular weight-bearing exercise or weight-training, or both, in conjunction with adequate calcium intake should benefit the peak adult bone mass of regularly menstruating women who have a family history of osteoporosis or other significant risk factors for low bone mass.

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The force absorption characteristics of cricket batting gloves at four impact velocities

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ABSTRACT

Injuries to the fingers, as a result of being struck while batting, fielding and catching, make up a significant percentage of the injuries to South African club and provincial cricketers and schoolboy cricketers, respectively. An experiment was conducted to compare the impact characteristics and the efficiency to absorb impact forces of four types of cricket batting gloves (G1, G2, G3 and G4) at four impact velocities: Slow-medium (S1), Fast-medium (S2), Fast (S3) and Express (S4). The impact forces were measured using the drop test where a weighted ball was dropped vertically onto the surface of the batting glove with the vertical forces measured by pressure sensors in a cricket bat handle. A two-way analysis of variance, with Tukey's method of multiple comparison, were used to test for significant differences ($p<0.05$) between the gloves at the four impact velocities. Significant differences were found at all the impact momentums. At S1, G4 showed significantly less ability to absorb the impact forces than the other three pairs of batting gloves. At S2 significant differences were found between G1 and G3. At S3, G4 showed the greatest ability to absorb the impact forces, while at the greatest impact velocity (S4), G3 and G4 showed the greatest ability to absorb the impact forces. These differences were as a result of the structure and composition of the protective part of the batting glove, which differed for all four makes of gloves. From the results it would appear that the batting gloves best able to absorb forces at the fastest impact velocities were those that had a thin plastic reinforcement over an extra padded layer of the fingers, plus the normal padding on the fingers.

Introduction

Cricket has traditionally been regarded as a sport that is relatively injury free, although it has been classified as a sport with a 'moderate' injury risk.[1] Over the past two decades the case reports and studies of the incidence of injuries in cricketers indicate an increase in injuries in cricket players. The major areas of concern are: (i) impact injuries, particularly to the fingers which were found to be the most vulnerable site of injury and consisted primarily of fractures, dislocations and contusions while batting and fielding,[2,3,4,5,6,7] and (ii) injuries to the back.[6,8,9,10]

In batting the top- and middle-order batsmen were found to be more susceptible to impact injuries to the phalanges and metacarpals than the lower-order batsmen.[9] Of the 62 upper limb injuries sustained by provincial cricketers, the phalanges, metacarpal and lower arm injuries made up 50 (81%) of the total upper limb injuries. Thirty (60%) of these were caused while batting with the top- and middle-order batsmen sustaining 24 (48%) injuries. At the beginning of the innings the top-order batsmen face the fast bowlers when they are rested and during the early part of the innings the ball and pitch usually possess more bounce tending to favour the fast bowlers thus making the batsmen more susceptible to impact injuries.

In a study of the seasonal injuries sustained in club and provincial cricketers, upper limb injuries accounted for 34,1% of the total injuries sustained and occurred predominantly in the fingers (20,5%) as a result of being struck by the ball while fielding, catching and batting.[11] Five injuries to batsmen were as a result of being struck on the hand and lower arm by fast bowlers. Similarly, among schoolboy cricketers upper limb injuries accounted for 24,6% of the total injuries sustained, with 15,8% of these injuries as a result of being struck on the fingers by the ball while batting, fielding and catching.[7]

The structure and composition of the protective part of the gloves over the index and middle fingers differed in all the makes of batting gloves assessed (Figure 1). In all four the batting gloves the fingers were protected by individual padding over each finger. However, three of the pairs of gloves (G1, G2 and G3) had an additional protective layer, which covered both the index and middle fingers.

The outer protective layer of both G1 and G2, which covered the first two fingers, consisted of sponge/polyurethane which was 10 mm thick. The outer protective layer of G3 consisted of sponge/

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polyurethane which was 2 mm thinner than that for G1 and G2, while its density, although not measured, appeared to be less than the other two gloves. The primary difference, however, was that G3 had additional protection in the form of with a flat plastic reinforcement 3mm thick over the outer protective layer over the index and middle fingers.

The structure and composition of the inner protective layer of the gloves varied with G2 made up of a similar component, 16 mm thick, to its outer layer. G1 and G3 were made up of a layer of tightly compacted stuffing in each of the fingers, with a thin (2 mm) sponge covering forming the innermost layer of protection. Additional padding was also provided by a sponge or polyurethane layer on the medial side of the index finger in G1 and G2.

G4 differed from the other three gloves in that each finger was protected individually. This consisted of three layers. The outermost part consisted of a thin (1 mm) plastic reinforcement, with a 2 mm foam covering on the outer side, over each finger. Secondly, the middle layer consisted of a 10 mm thick composition similar to the outer and middle layers in G2. Finally, the inner most layer, 7 mm thick, was made from a component similar to that used in the outer protective layer in G3 although it appeared to be less dense.

Taking into account the seriousness of the impact injuries to the fingers in cricket batting, the primary aim of this study was to compare the impact characteristics and the efficiency to absorb impact forces of four types of cricket batting gloves at four different impact momentums. A secondary aim was to identify the reason, if any, for these differences.

Method

Four new pairs of cricket batting gloves (G1, G2, G3 and G4) were used in this study. These

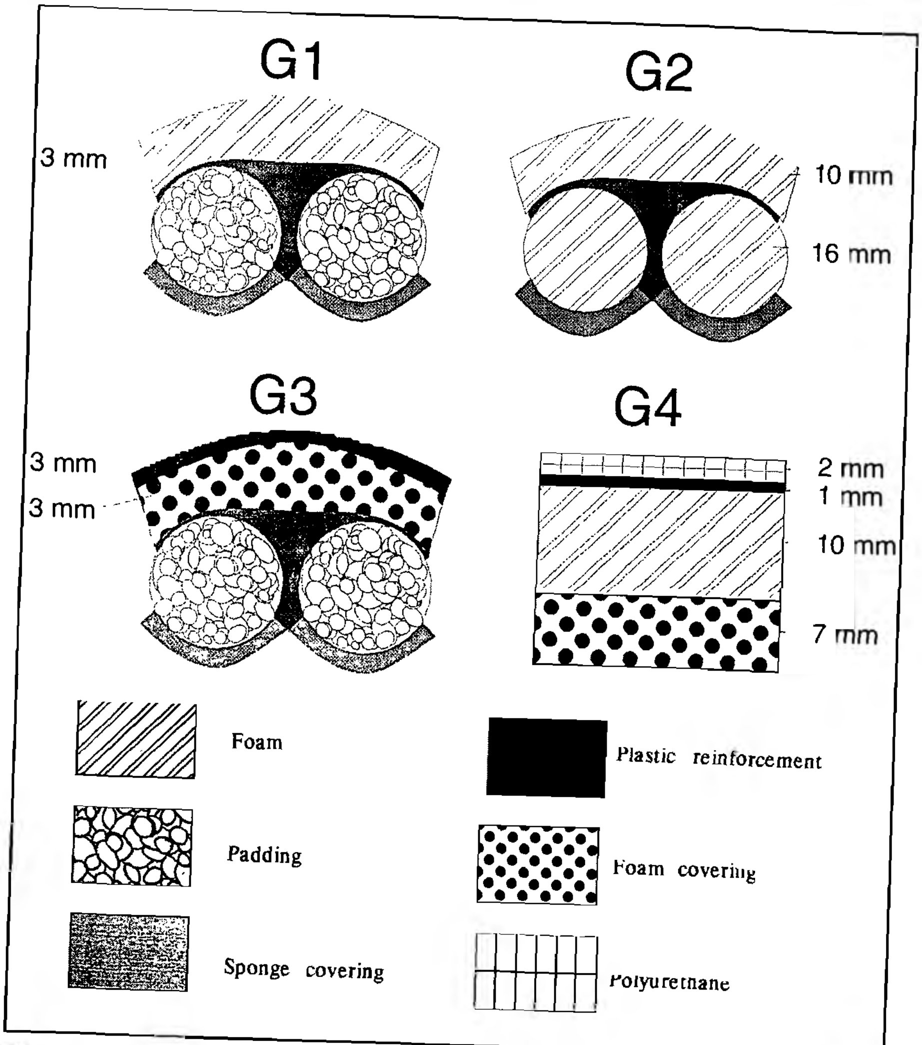


Figure 1: *Schematic representation of the protective layers in the batting gloves.*

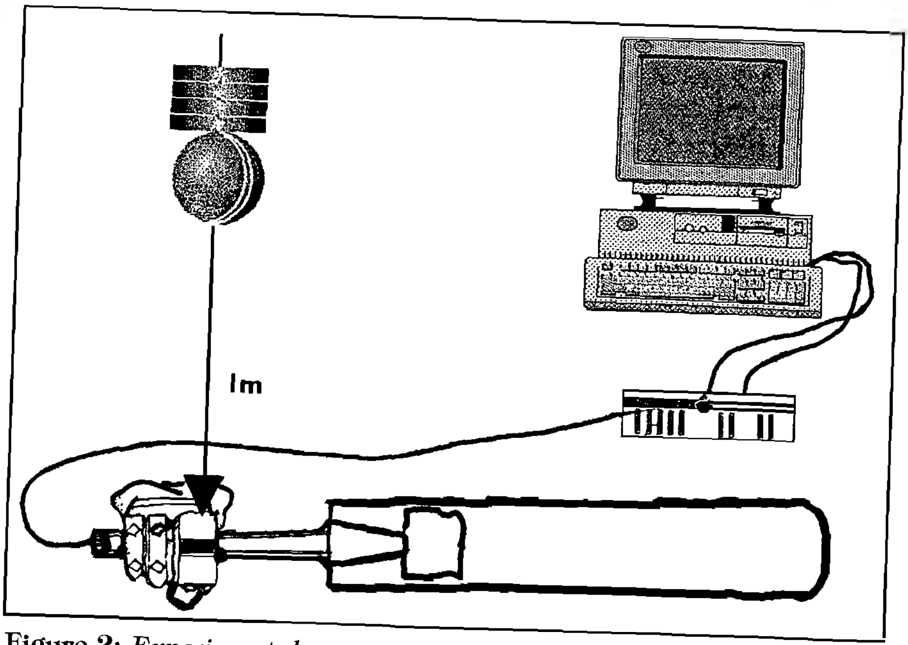


Figure 2: *Experimental setup.*

were supplied by four different manufactures and were rated as their top of the range batting gloves. The palm of each right-hand glove was removed so that it could be placed on the testing apparatus. The procedure used in the assessment of the cushioning properties of the batting gloves was based on the drop test.[12] In this test the dropping mass falls onto the surface of the batting glove with the vertical forces measured by the pressure sensors in the cricket bat handle (Figure 2).

The vertical forces were recorded using the standard sized short-handle Slazenger cricket bat which was instrumented to respond to hand

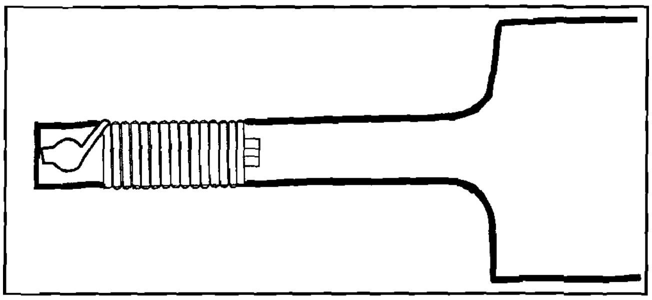


Figure 3: *Position of the pressure sensors in the bat handles.*

grip pressure during the execution of the batting stroke in cricket.[13] Forces were measured by one of the two pressure sensors (Motorola MPX 10 GP) with a pressure range of 0,0 to 1,5 PSI (0,0 to 10,3365 kPa). The pressure sensor uses a single piezoresistive element diffused on an etched silicon diaphragm to sense the stress induced on the diaphragm by an external pressure. The sensor is rugged, provides a clean resistance change and is almost a totally non-invasive force measurement. The effect of temperature is a maximum of ± 1% of full scale over a temperature range of 0° to 85°C. The pressure sensor was linked, via the side pressure port, to a silicon rubber tubing that was positioned around the bat handle. The open end of this tube was sealed with silicon rubber.

The pressure sensor was positioned into a hollow cut into the bat handle, with a small channel cut out for the pressure port and silicon rubber tubing to pass along (Figure 3). A 1,72 metre silicon tube with an inner diameter of 3 mm was attached to the pressure port of the pressure sensor and wound around the bat handle fifteen times. Where the tube was wound around the bat handle the diameter was reduced slightly in order to maintain the original diameter of the bat handle. This method ensured that the pressure applied by all parts of the glove in contact with the bat was recorded. Wires attached to the pressure sensor tab passed through the handle to run along a small channel cut into the back of the handle. These wires passed upwards along the handle to a termination board positioned at the top of the handle. From here the electrical leads exited through a hole drilled from the termination board through the top of the handle. After mounting the pressure sensor the rubber grip was replaced on the bat handle. A 5 mm thick lead then passed to an amplifier where the voltage frequency from the pressure sensor was amplified. This was connected via an interface cable to a computer which was fitted with an analogue to digital converter card (P.C. 30) to convert the impulses for storage and later retrieval and analysis. The circuit is powered from the computer via the interface cable. The system is fitted with an auto-zeroing circuit for cancelling zero drift due to temperature and pressure effects on the material of the silicon rubber tubing around the bat handle.

The pressure sensor was calibrated after installation into the bat handle using the same circuit, computer and programme used in the actual experiment. The equipment was calibration by loading and unloading the pressure sensor from 0 N to 260 N while voltage and load cell data was sampled to 1000 Hz. The pre-experiment calibration was used to make the first discrete calibration file (1 volt = 26.4703 N), while the pre- and post-experiment calibration trials showed pressure sensor output consistency (r = 0.999).

The impact velocities used in this study were based on the release velocities in a study on elite fast bowlers[14] and taking into account a drop-off of 14,3%.[15] These were classified as slow-medium (S1), fast-medium (S2), fast (S3) and express (S4) (Table 1).

Table I
RELEASE AND IMPACT VELOCITIES OF THE BALL UNDER MATCH CONDITIONS AND THE CALCULATED MASS OF THE EXPERIMENTAL "BALL"

	Release Velocity ($m.s^{-1}$)*	Impact Velocity ($m.s^{-1}$)**	Mass of Experimental "Ball" (g)
Slow-medium (S1)	17,90	15,34	630,8
Fast-medium (S2)	26,80	22,97	944,4
Fast (S3)	35,80	30,68	1 262,8
Express (S4)	40,20	34,45	1 416,6

* Calculated from Abernethy (1981)
** Calculated from Penrose et al (1976)

The impact velocity of the ball under match conditions was simulated using a new Duncan Fearnly cricket ball which was weighted and dropped vertically onto the batting glove from a height of 1 metre. The mass of the experimental "ball" was determined as described below:

The impact momentum of the ball under match conditions for the different impact velocities was computed from the following:

Momentum = mv

where:

m = Mass of ball (156 g)

v = Velocity of the ball at impact from Table I

The velocity of the experimental "ball" dropped from a height of 1 metre was computed from the following:

Velocity = $\sqrt{2gd}$ (ii)

where:

g = Gravity (9,8050 m/s^2)

d = Distance which remained constant at 1 metre

The mass of the experimental "ball" dropped from a height of 1 metre was computed from the following:

Mass = $\frac{m}{v}$

where:

m = Impact momentum of the ball at various impact velocities under match conditions, calculated from (i)

v = Velocity of ball at impact, calculated from (ii)

The gloves were subjected to ten impacts at each of the velocities with the weighted "ball" to simulate impact at each of the four release velocities. The gloves were positioned on the bat so that impact would occur over the first two digits of the index and middle fingers of the right (bottom) hand. At this site all the manufacturers ensured that maximum protection occurred by providing additional padding and/or placing a thin plastic reinforcement into the padding of the glove.

The statistical package Statistica was used to compute single variable statistics. A normal probability plot (Figure 4) was done for the data and showed it to be approximately normally distributed. Since the data was approximately normally distributed and homogeneity was assumed, a two-way analysis of variance was used to examine whether any inter-glove differences occurred or whether the gloves showed

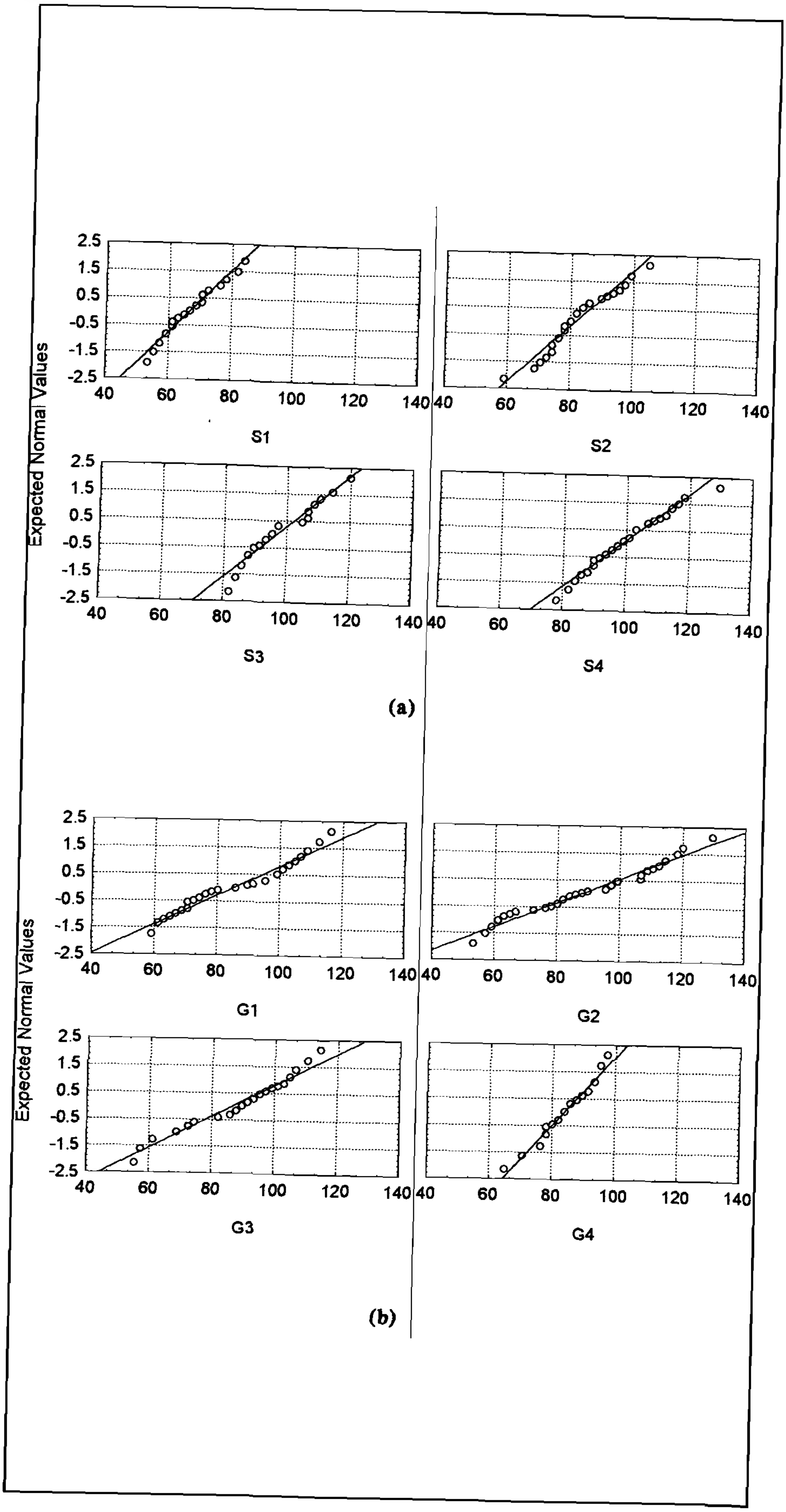


Figure 4: *Normal probability plot for the force data (N) at (a) the four impact velocities and (b) the four types of batting gloves.*

similar impact characteristics at the different impact velocities. Tukey's method of multiple comparison[16] was used to establish the significance of the differences. The five percent level of significance was used for all tests.

Results

The mean impact forces for the different batting gloves at the four velocities are shown in Figure 5. These results are summarized in Table II which shows means and standard deviations (SD). The 2-way analysis of variance showed a significant interaction effect (Table III, Figure 6 and Figure 7). Tukey's multiple comparison tests showed which gloves differed significantly at each of the impact velocities (Table IV).

At S1, G4 differed significantly from all the other pairs of batting gloves, showing the greatest forces recorded, meaning that it had the least ability to absorb the impact forces from the ball. At S2, G2 and G3 showed similar abilities to absorb the impact forces of the ball, while significant differences were found between G1 and G3. At S3, both G2 and G3 differed significantly from G4, with G4 showing the greatest ability to absorb the impact forces. At S4, G1 and G2 showed significantly less ability to absorb the impact forces than G3 and G4 which were non-significant in themselves.

Discussion

The principal finding of this study was that there is a significant difference between the impact forces of cricket batting gloves at various impact velocities. These differences would be as a result of the differences in the structure and composition of this protective part of the gloves. The manufacturers are, however in the difficult position of having to balance the impact absorption characteristics of the gloves with the comfort required to wear them for long periods at a time.

From the findings it would appear that the batting gloves best able to absorb the impact forces of a ball bowled at fast to express velocities would be those with a plastic reinforcement over the padding of the fingers, preferably in the outer section of padding that covers the first two fingers. This rigid surface would serve to dissipate the impact forces of the cricket ball from its point of application over as wide an area as is possible, thus reducing the pressure on the phalagens to tolerable levels.

Secondly, it would appear that the density of the material used should decrease from the outer layer inwards thus offering the best cushioning of the impact forces. This more flexible state will enable the protective layers of the batting gloves to act as a dampening spring. This would allow an increased time for the deceleration of the ball to occur and consequently softer impact on the fingers by this phased deceleration of the ball. These results are as a result of

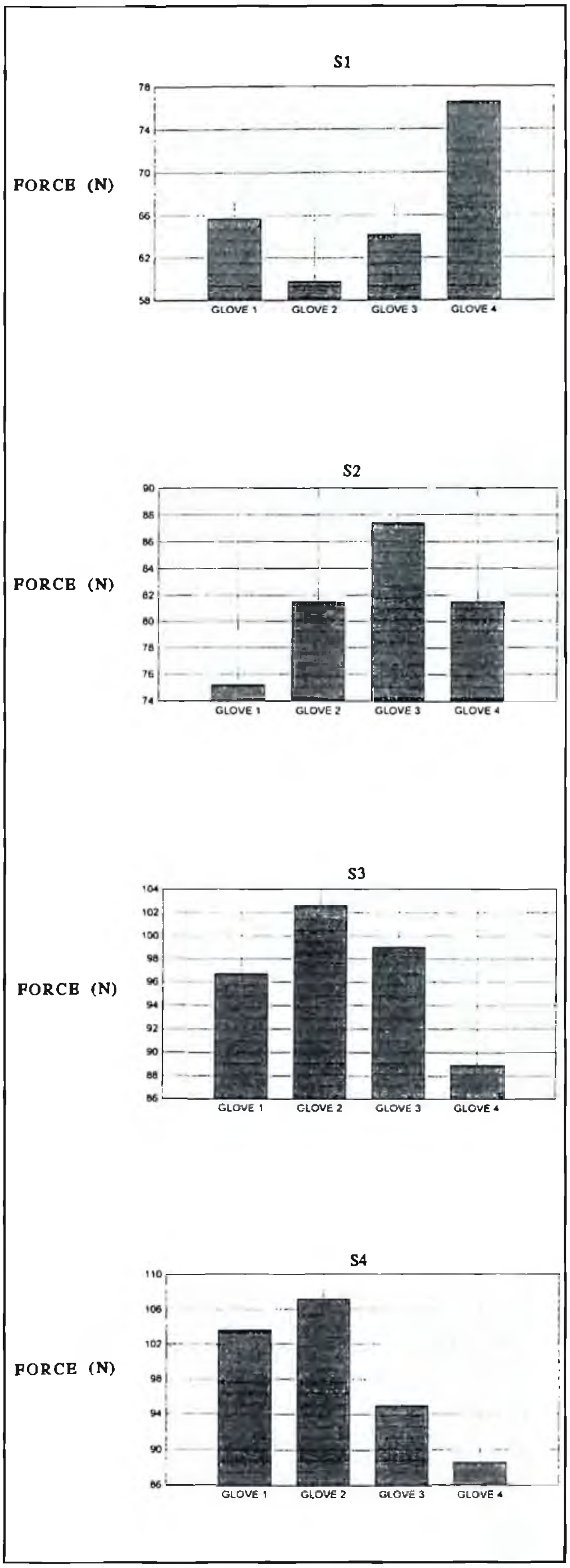
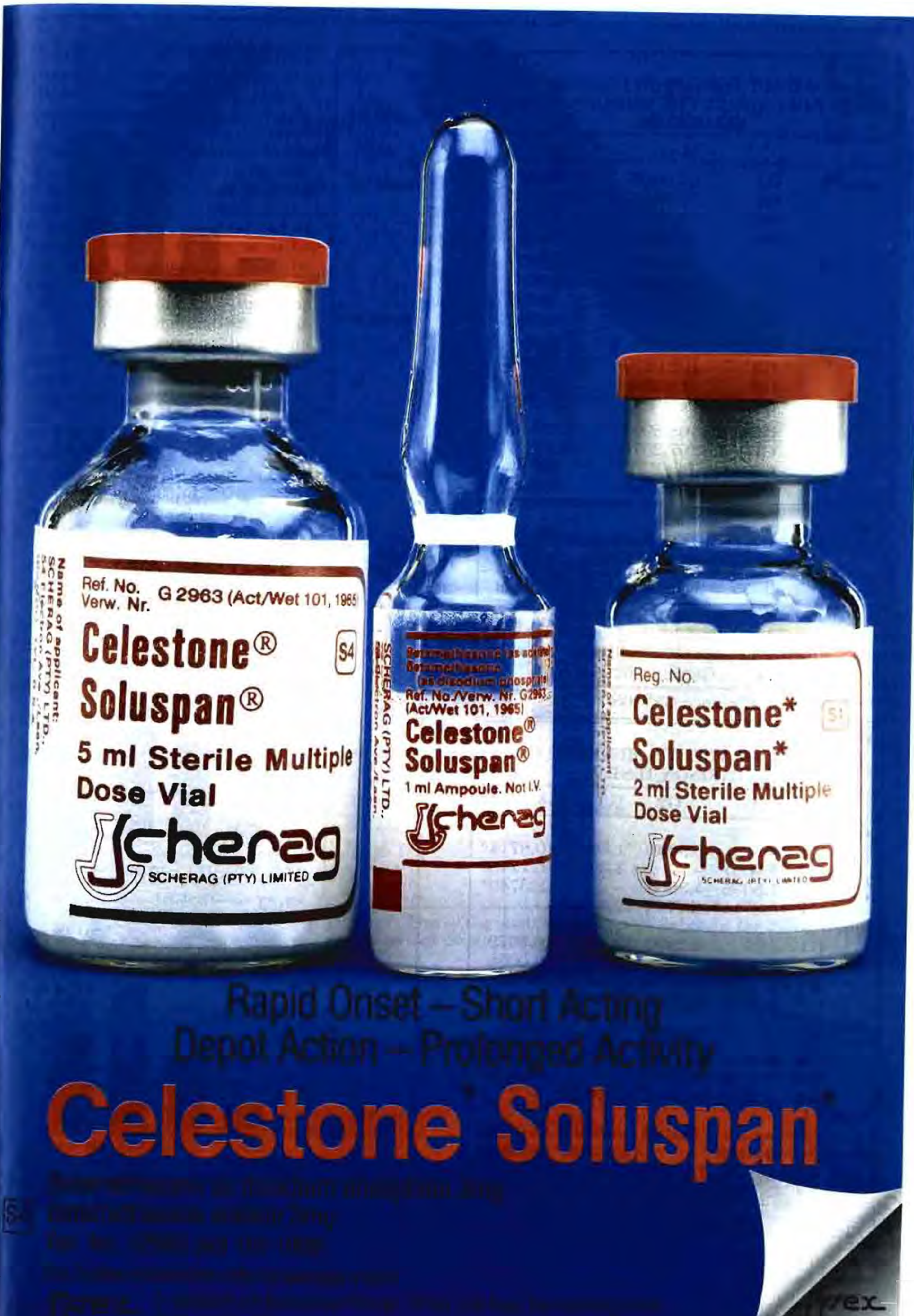


Figure 5: *Mean impact forces for the different gloves at the various impact velocities.*



Ref. No. Verw. Nr. G 2963 (Act/Wet 101, 1965)
Celestone® Soluspan®
S4
5 ml Sterile Multiple Dose Vial
Scherag
SCHERAG (PTY) LIMITED
Name of applicant: SCHERAG (PTY) LTD.
Ref. No./Verw. Nr. G2963
(Act/Wet 101, 1965)
Celestone® Soluspan®
1 ml Ampoule. Not I.V.
Scherag
SCHERAG (PTY) LTD.
Reg. No.
Celestone* Soluspan*
2 ml Sterile Multiple Dose Vial
Scherag
SCHERAG (PTY) LIMITED
Rapid Onset – Short Acting
Depot Action – Prolonged Activity
Celestone* Soluspan*

Table II
MEAN IMPACT FORCES (N) FOR THE DIFFERENT GLOVES AT THE VARIOUS IMPACT VELOCITIES

		Mean	SD
Speed 1	G1	65.6807	5.10269
	G2	59.7806	4.41392
	G3	64.1772	6.60116
	G4	76.5521	6.28207
Speed 2	G1	75.2100	9.21077
	G2	81.5020	6.58204
	G3	87.3995	11.16700
	G4	81.5073	4.64532
Speed 3	G1	96.7277	8.79259
	G2	102.6279	11.45240
	G3	99.0280	9.40085
	G4	88.9402	4.30283
Speed 4	G1	103.5968	6.63416
	G2	107.2073	12.15657
	G3	95.0177	7.52594
	G4	88.5510	6.35688

Table III
SUMMARY OF THE TWO-WAY ANALYSIS OF VARIANCE TEST

Source	df	Mean Squares	F	P-level
Speed	3	9004.154	143.0325*	0.000000
Glove	3	109.073	1.7326	0.162943
Interaction	9	563.817	8.9563*	0.000002
Error	144	62.952	62.9518	

* Significant difference (P<0.05)

Table IV
SUMMARY OF TUKEY'S MULTIPLE COMPARISON TEST

Glove	G1	G2	G3	G4
Speed 1				
G1	–	5.9001	1.5035	10.8714*
G2	5.9001	–	4.3966	16.7715*
G3	1.5035	4.3966	–	12.3749*
G4	10.8714*	16.7715*	12.3749*	–
Speed 2				
G1	–	6.2920	12.1895*	6.2973
G2	6.2920	–	5.8975	0.0053
G3	12.1895*	5.8975	–	5.8922
G4	6.2973	0.0053	5.8922	–
Speed 3				
G1	–	5.9002	2.3003	7.7875
G2	5.9002	–	3.5999	13.6877*
G3	2.3003	3.5999	–	10.0878*
G4	7.7875	13.6877*	10.0878*	–
Speed 4				
G1	–	3.6105	8.5791*	15.0458
G2	3.6105	–	12.1896*	18.6563*
G3	8.5791*	12.1896*	–	6.4667
G4	15.0458*	18.6563*	6.4667	–

*Significant difference (P<0.05)

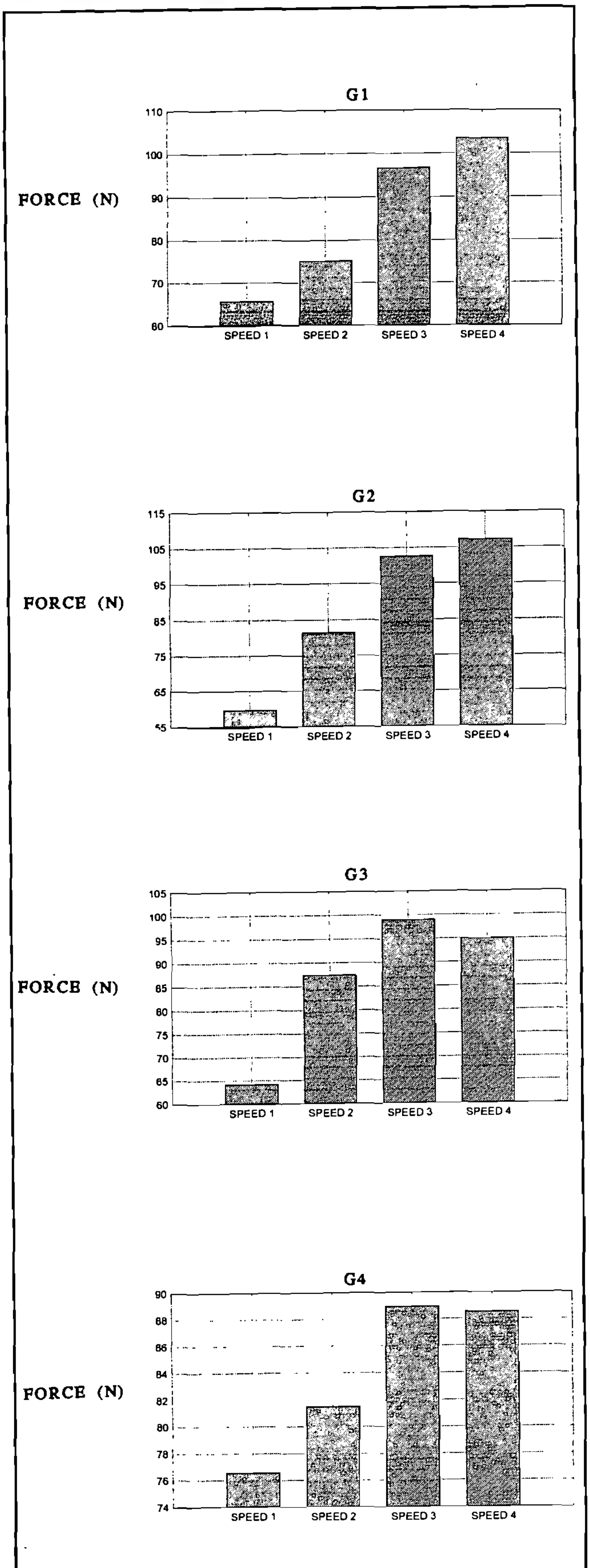


Figure 6: *Mean impact forces for the different gloves at the various impact velocities.*

Newton's second law of motion concurs with the observations in a study evaluating impact absorption when landing from a jump.[17]

Further research needs to be conducted on the protective layers if any attempt by the cricket batting glove manufacturers at trying to reduce the impact forces on the cricket batsman's hand when struck by a fast bowler is to be successful. Attention needs to be paid to altering the structure of the protective material used, as well as the combination of the strucutre of the protective layers used as the reduction of force levels is a result of a complex interaction between the various protective layers. These could be done by further investigating the impact properties of the components or combinations of components use in the protective layers in cricket batting gloves, either using mathematical modelling techniques or by assessing the shock absorption characteristics of the individual layers or combinations of layers. Only through continual research in the design and composition of the materials used in the manufacture of batting gloves, will the risk of a fracture to the fingers while batting in cricket be reduced.

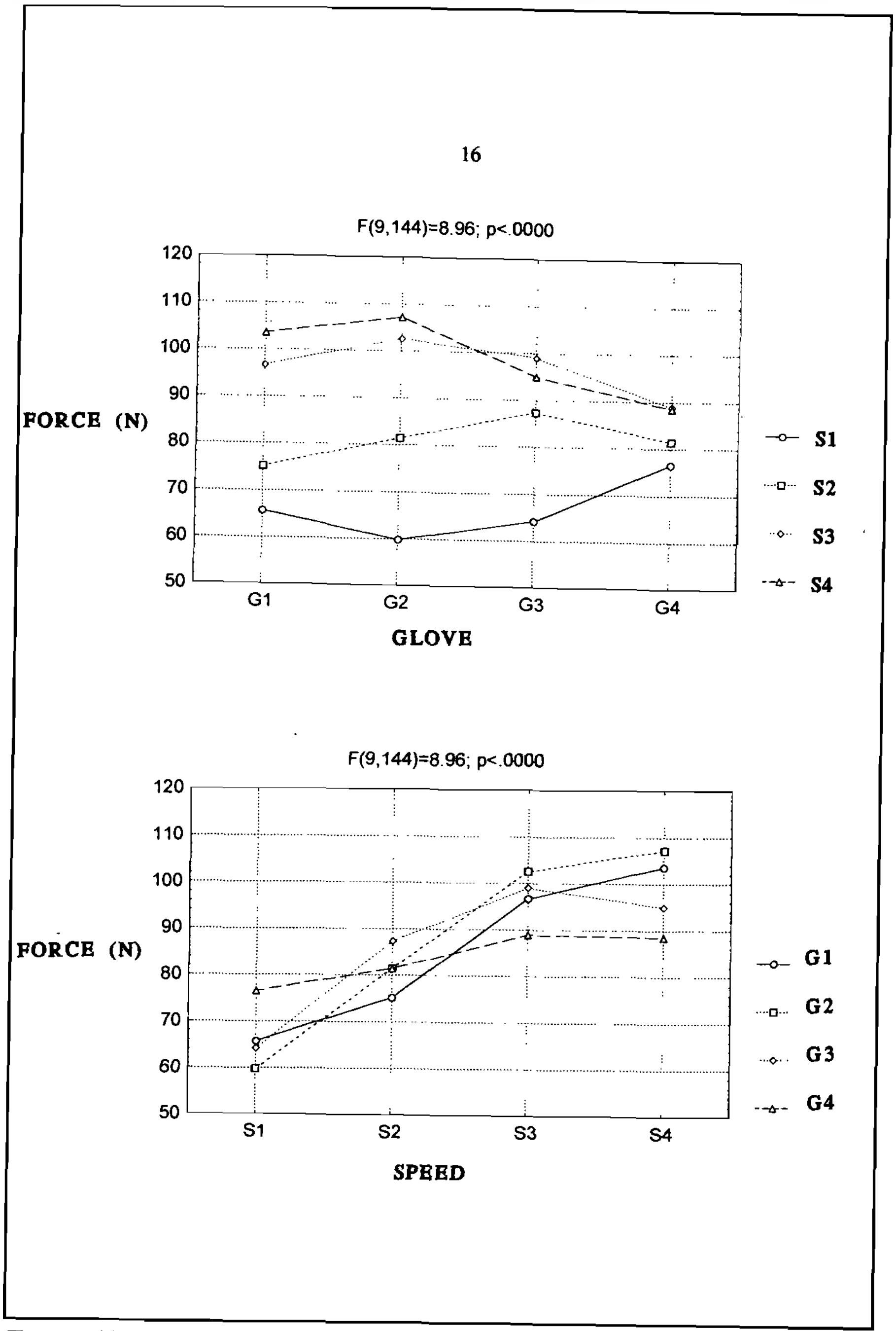


Figure 7: *Plot of the mans of the impact forces for the different gloves at the various impact velocities.*

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Perceptions of Sport Injury Rehabilitation in the Cape Town Sporting Community

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Abstract

A random sample of the Cape Town sporting community completed a questionnaire study, to assess their opinion on treatment they received for injuries incurred whilst playing sport, and their knowledge of the function of various members of the sports health-care team. Questionnaires were distributed to clubs or individuals in a variety of different sports. Of 260 questionnaires, 48 were answered incorrectly and excluded from analysis. Of the 212 remaining subjects who completed questionnaires, the majority were male (86%), between the ages 20-30 (69%) and on medical aid (74%). Of the subjects, 71% had suffered a sport-related injury in the last five years. The body parts most often injured were the leg (19%), knee (18%), shoulder (15%) and ankle (15%). Of those injured, 42% went directly to a physiotherapist for treatment, and 11% directly to an orthopaedic surgeon. There was little difference in the professional chosen to treat the injury by those subjects covered by medical aid and those who were not. None of the injured subjects consulted a biokineticist, and no person in this sample was referred to or managed by a biokineticist during rehabilitation. The majority of people were satisfied with their treatment (78%) and would go to the same person again with the same injury (87%). A large proportion of subjects were not aware of any sport medicine physicians practicing in Cape Town (46%), and there was a high level of ignorance of the function of biokineticists (47%). The results of this study illustrate that, generally, the sport health care team is well received and successful in the management of sporting injuries in Cape Town, with physiotherapists managing the highest proportion of injuries. However, biokinetics is a profession which is not well known to the Cape Town sporting population, and because of this, the multi-disciplinary approach to sport injury management is not being correctly utilised at present. Perhaps better marketing of this concept is required by all professionals involved in sport injury management.

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Introduction

During the past two decades, the medical and para-medical services offered to sports participants in South Africa have increased.[1,2] The development of these services appears to have followed the trend which has emerged in other countries.[3] Studies done at hospital casualty departments[4] have defined the types of sport injuries treated by hospital clinicians, and studies of different sporting disciplines have identified injuries common to these sports.[5,6,7,8]

In South Africa, three distinct specialty services available to the injured athlete have evolved. Medical doctors can train to become primary care sports medicine physicians. Although these specialists are not recognised as such by the SAMDC, sports medicine physicians are trained to diagnose orthopaedic sports injuries and metabolic conditions which result from physical activity. Physiotherapists can receive post-graduate training in acute phase injury rehabilitation; and biokineticists, recognised by the SAMDC since 1983, are trained primarily in final phase injury rehabilitation.[9] Therefore, there is a multi-disciplinary team of professionals available to diagnose and treat an injury in the acute and final phase, enabling the athlete to return to sport participation sooner than if these services were not offered.[3]

The extent to which these health professionals are utilised by injured sports participants is not known. The aim of this study, therefore, was to sample a group of sports participants, and through a questionnaire assess the nature of injuries suffered by them, the treatment of these injuries and their perceptions of the function of various members of the sports medicine health-care team. It was decided to conduct the study in the Cape Town region, since this region is representative of the metropolitan regions of South Africa in nature and number of health care personnel available to the injured athlete.[2]

Subjects and methods

A questionnaire study of the sporting community of the Cape Town region was undertaken during 1993/1994. In compiling the questionnaire, draft copies were sent to a statistitian, biokineticist, physiotherapist, and a sports medicine physician for their comments. The questionnaire was translated into Afrikaans and sent to an Afrikaans doctor who checked the translated version. A pilot study was conducted on a random sample of subjects. Any problematical questions in the questionnaire were adjusted.

Participants in the study were randomly selected from rugby, cricket, running, squash, canoeing, weightlifting and aerobics, which represented a broad spectrum of sporting activities. Clubs in each sporting discipline were randomly selected where possible. Once authorisation to distribute the questionnaire had been obtained from the club officials, individual participants completed the questionnaire after being randomly selected at training venues. In the sports with fewer participants such as canoeing, participants were randomly selected from those canoeists training at the different venues in the Cape Town Region. Participants could choose to answer either the English or Afrikaans version of the questionnaire. Although help was supplied for any grammatical queries with the questionnaire, no discussion was allowed on answers to the questions.

The questionnaire sought answers to the following questions in both the injured and non-injured population groups: (i) Personal data – age, gender, language group, occupation, type of sport played and competitive level. (ii) Previous history of injuries suffered from participation in sport (single latest injury in the last 5 years of sport participation) including body part injured, severity of injury, and which health care professional attended to the injury in both the acute and late phase. (iii) Perceptions of treatment received and subjective level of satisfaction derived from the treatment. (iv) Knowledge of the role of various members of the health care team – including sports medicine physicians, physiotherapists and biokineticists. (v) Number of health care practitioners in Cape Town.

Results

Two hundred and sixty questionnaires were distributed using the methods described previously. Forty eight questionnaires were excluded from the final analysis due to missing data. Table I describes the personal characteristics of the subjects (n = 212). A high proportion of the sample were male (86%), between 21-30 years of age (69%), on medical aid (74%) and English-speaking (81%). Table II describes the sporting history of the subjects, with rugby (28%) and cricket (20%) being most represented. A high proportion of subjects (70%) perceived themselves to be competitive athletes. Thirty one percent of all subjects were aware of medical services being offered by their respective clubs, 46% were unaware and 23% were unsure of services at their disposal.

Of the 212 subjects, 71% had suffered a sport-related injury within the last 5 years. Injuries involved most anatomical regions, with the leg (19%), knee (18%), shoulder (15%), and ankle

Table I
DESCRIPTIVE DATA OF SUBJECTS (N = 212).

		Percentage
Gender:	Male	86
	Female	14
Age (years):	10 – 20	14
	21 – 30	69
	31 – 40	13
	> 40	4
Occupation:	Business	48
	Student	33
	Other	11
	Sportsperson *	6
	Medical **	2
Medical Aid:	Yes	74
	No	26
Language:	English	81
	Afrikaans	19

* – Full Time Professional Sportsperson
** – Member of Medical or Paramedical Professions

Table II
DESCRIPTIVE DATA OF THE SPORTS IN WHICH SUBJECTS (N = 212) PARTICIPATED.

	Percentage
Sport Involved In:	
Rugby	28
Cricket	20
Running	14
Squash	11
Aerobics	8
Weightlifting	8
Other	6
Canoeing	5
Total	100
Level of Competition:	
Competitive	70
Social	30
Total	100
Club Medical Services:*	
Yes	31
No	46
Unsure	23
Total	100

* – Medical services perceived by the subjects to be available to them through their respective clubs

Table III
DESCRIPTIVE DATA OF THE INJURIES SUSTAINED BY THE SUBJECTS (N = 212).

	Percentage
Injured	71
Non-injured	29
Body Part Injured:	
Leg	19
Knee	18
Shoulder	15
Ankle	15
Back	8
Wrist	4
Finger	4
Neck	3
Hand	3
Hip	3
Foot	3
Head	2
Arm	2
Elbow	1
Total	100
Severity:	
Continue Sport	53
Halt totally	47
Total	100
Tissue Type Injured:	
Ligament	32
Muscle	23
Bone	17
Unsure	10
Tendon	9
Other	9
Total	100

Table IV
INITIAL MEDICAL CONTACT AND PERSONNEL RESPONSIBLE FOR FINAL MANAGEMENT OF THE SUBJECTS INJURY (N = 150).

	Initial Contact (%)	Final Management (%)
Biokineticist	0	0
Chiropractor	1	3
General Practitioner	21	9
Hospital Casualty	8	3
Orthopaedic Surgeon	11	26
Pharmacist	0	1
Physiotherapist	42	42
Self managed	6	6
Sport Medicine Physician	11	10
Total	100	100

(15%) being injured most frequently (Table III). Just under half of the injuries (47%) necessitated total cessation of sporting activities, whilst the remaining 53% allowed continuation of sport during ongoing therapy. Ligamentous (32%) and muscular tissue (23%) were most often reported by the athletes as being injured, although 10% of subjects were totally unsure of the nature of their injury.

Table IV shows which medical professionals were responsible for both initial contact with the injured sportsperson, and final management of their injuries. The question of which professional the subjects would consult after an injury was first analyzed by splitting the subjects into medical aid and non-medical aid groups. However, the responses from these were so similar that it was decided to report the results of the combined group. Of the 150 injured athletes discussed above, it is interesting to note that 42% consulted a physiotherapist and 11% an orthopaedic surgeon directly, whereas 21% went to a general practitioner, 11% to a sport medicine physician and 8% to a hospital casualty. None of the sample population consulted a biokineticist, either for initial diagnosis or final management of their injury. The majority of patients stayed with a physiotherapist for treatment (42%), although the number of patients treated by orthopaedic surgeons increased from 11% to 26% in the late phase of management. Table V shows that 78% of subjects were satisfied with the treatment they received, and that 87% of them would consult the same person again if they had a similar injury.

Of the 212 subjects, 47% did not know the professional role of a biokineticist, compared to 19% for sports medicine physicians and 8% for physiotherapists. Forty six percent of all subjects were not aware of any sport medicine physicians practicing in Cape Town. Of those subjects who believed they knew what the various functions of the health professionals were, 33% gave incorrect answers regarding the role of a biokineticist, as compared to 10% for sport medicine physicians and 1% for physiotherapists.

Table VI shows the medical health services the non-injured subjects would choose to consult if suffering a major or minor sports injury. For a major injury, 39% of these athletes would go to a general practitioner, 24% to a hospital casualty, and 20% to a sport medicine physician. If suffering a minor injury, 59% of non-injured athletes

Table V
PERCEPTIONS OF TREATMENT RECEIVED BY THE INJURED SUBJECTS (N = 150).

	Percentage
Dissatisfied	15
Unsure	7
Satisfied	78
Total	100
Consult same person with similar injury?:	
Yes	87
No	13
Total	100

would consult a general practitioner, 24% a physiotherapist, and 14% to a sports medicine physician. No subjects would go to a biokineticist with a major injury, whereas 1% would go to a biokineticist with a minor injury.

Table VII shows the number of health practitioners currently practicing in the Cape Town region. There is as expected a high proportion of general practitioners in the region compared to the other members of the health care team. There was a high proportion of physiotherapists (481) relative to biokineticists (11) and sports medicine physicians (11). The number of hospital casualties (4) was taken as the number of casualties with radiographic facilities available to the general public which were not attached to private hospitals.

Discussion

Several points of discussion arise from this questionnaire study. Firstly, the fact that the majority of subjects were on medical aid is important, as this would theoretically allow greater freedom of choice in choosing a member of the health care team when suffering an injury. Surprisingly, this was not the case, as subjects either with or without medical aid had similar responses.

Secondly, the majority of injured people appeared to bypass the hospital casualty and general practitioner and went directly to either physiotherapists or orthopaedic surgeons. Physiotherapists appear to be favoured by the sporting community – certainly in the population in this study – both for initial diagnosis and management of injuries. In contrast, only 11% of the subjects consulted a sports medicine physician for diagnosis of their injuries. This may be related to the finding that approximately half of all subjects were unaware of any sport medicine physicians practicing in Cape Town, a situation which may be exacerbated by the relatively low number of sport medicine physicians practicing in the Cape Town region.

It was important also to note that not one subject went to a biokineticist either on their first visit or for management of their injury. Given that biokineticists are trained for final phase rehabilitation, it is not surprising that there were no initial consultations performed by biokineticians. However, the finding that no patients were referred to a biokineticist for late phase rehabilitation is in contrast to an earlier South African study[9] whose figures for patient referrals to biokineticists showed steady increases over the years 1989-1991. More subjects were totally unaware of the role of biokineticists as compared to the role of sports medicine physicians and physiotherapists. More subjects incorrectly identified the role of biokineticists compared to sports medicine physicians and physiotherapists. This may be caused by the fact that biokinetics is a relatively new profession. Whatever the cause, the above findings suggest that the profession of

Table VI
MEDICAL HEALTH SERVICES THE NON-INJURED SUBJECTS (N = 62) WOULD CHOOSE TO CONSULT IF SUFFERING A MAJOR* OR MINOR** INJURY

	Major Injury (%)	Minor Injury (%)
Biokineticist	0	1
Chiropractor	3	0
General Practitioner	39	60
Hospital Casualty	24	0
Orthopaedic Surgeon	8	1
Pharmacist	0	0
Physiotherapist	6	24
Spots Medicine Physician	20	14
Total	100	100

* – Fracture of a limb requiring cessation of sport and immediate treatment
** – Insidious onset chronic over-use type injury

Table VII
NUMBER OF HEALTH PRACTITIONERS IN THE DIFFERENT DISCIPLINES IN THE CAPE TOWN REGION.

	Number
Biokineticists	11
Chiropractors	34
General Practitioners	526
Hospital Casualties	4
Orthopaedic Surgeons	71
Pharmacists	280
Physiotherapists	481
Sports Medicine Physicians	11

biokinetics is not well exposed to sports participants, certainly in the Cape Town region, and perhaps may require improved marketing.

The nature of injuries reported by the subjects does not differ greatly from those reported in other published papers.[3-6] It is interesting that 10% of the subjects were unsure of the nature of their injury, which suggests a lack of communication from the health care members involved. Although the majority of subjects were satisfied with the treatment they received and would consult the same clinician again with a similar injury, a small percentage of subjects would go to the same person even if they were dissatisfied with the treatment they had received. This is a somewhat puzzling finding.

Despite a large proportion of injured subjects consulting a physiotherapist or orthopaedic surgeon when injured, the majority of non-injured subjects reported that they would go to a general practitioner or hospital casualty if they were to suffer a injury of any type or severity. It would be interesting to see who these subjects would actually consult if they suffered an injury, given the theoretical discrepancy between the injured and non-injured groups.

In conclusion, it would appear that the various disciplines of the sport health-care team are generally well received by those athletes they manage, with physiotherapists appearing to play a major role in the treatment of sport injuries. However, in contradiction to a previous study,[9] biokinetics is a profession which is not well known to the Cape Town sporting population, and because of this, the multi-disciplinary approach to sport injury management in the Cape Town region is not being correctly utilised at present. Perhaps better marketing of this concept is required by all professionals involved in sport injury management.

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Readers' letters concerning articles in the Journal are invited, and will be forwarded to our Editors for consideration for publication.

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Material submitted for publication in the SAJSM is accepted on condition that it has not been published elsewhere. The management reserves the copyright of the material published.

Original articles of 3 000 words or less, with up to six tables or illustrations, should normally report observations or research of relevance to Sports Medicine or related area. Short reports of 1 000 words or less, with one table or illustration and no more than five references, include case reports, side-effects of drugs and brief or negative research findings. Review articles, except those written by invitation, will not be accepted. Authors may request that an article be considered for rapid publication; such articles should be of unusual importance.

Letters to the Editor intended for the correspondence column should be marked "for publication". Letters longer than 400 words may be returned for shortening, and only one illustration or table will be permitted.

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6. If in doubt, refer to "Uniform requirements for manuscripts submitted to biomedical journals (Br Med J 1991; 302: 338-341).

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1. Figures consist of all material which cannot be set in type, such as photographs and line drawings. (Tables are not included in this classification and should not be submitted as photographs.) Photographs (in triplicate) should be glossy, unmounted prints. In no circumstances should original x-ray films be forwarded; glossy prints must be submitted.
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   a. Williams G. Textbook of Sports Medicine. 2nd Edition: Butterworth, 1989: 101-104.
   b. Vandermere P, Russel P. Biomechanics of the hip joint. In: Nordien PE, Jeffcoat A, eds. Clinical Biomechanics. Philadelphia: WB Saunders, 1990: 472-479.
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