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Editorial

The contents of the first edition of the *South African Journal of Sports Medicine* for 2001 reflect the multi-disciplinary nature of sports medicine. The study by Nurok and colleagues on the athletic ability of young Kenyan athletes reaches the conclusion that the superior running ability of the Kenyans can perhaps be explained by specific inherited characteristics. However, it is a difficult theory to prove and it is going to take many more studies in disciplines ranging from sociology to molecular biology and genetics before we can conclude with confidence that factors governing success in athletics are inherited.

The study by Weston and her colleagues on heart rate as a marker of exercise intensity during mini-trampoline exercise contributes to the knowledge in the field of applied exercise physiology. This study shows that the oxygen consumption/heart rate relationship during mini-trampoline exercise is not always linear. Clearly, under these conditions heart rate is not an accurate marker of exercise intensity and therefore should be used with caution in prescribing exercise. This study once again exposes the predicament that we have in the fields of sports medicine and exercise physiology. For example, on the one hand we have devices for measuring heart rate which are highly sophisticated and can measure heart rate under free living conditions with a high degree of accuracy. Furthermore, the heart rate data can be stored for several days before being transferred to a computer for analysis. On the other hand we have an emerging understanding of how heart rate changes during exercise under various conditions. We know that factors such as environmental temperature, state of hydration, mode of exercise, duration of exercise and competition all have a significant effect on the heart rate/exercise intensity relationship.

More recently it was shown that as physical fitness increases, maximum heart rate decreases (Zavorsky G S. Evidence and possible mechanisms of altered heart rate with endurance training and tapering. *Sports Med* 2000; **29:** 13 - 26.) This finding has important implications in the health industry where exercise participants are encouraged to monitor their training intensity according to their heart rate expressed as a percentage of maximum heart rate. Clearly if the decrease in maximum heart rate with increasing fitness is not taken into account, then the relative intensity of the training sessions will become harder and harder as fitness improves. In summary, the study by Weston and her colleagues is important in that it contributes to narrowing the large gap between the technical capabilities of heart rate monitors and the understanding of how heart rate responds to exercise. This gap has to be narrowed even further before heart rate monitors can be used to their full potential.

The study by Marino and Booth addresses the question of whether precooling before endurance exercise in moderate and high environmental temperatures has any ergogenic effect. Research into this area is fascinating for two reasons. Firstly, the underlying physiological mechanisms of the 'precooling' effect are not fully understood. Secondly, the applied spin-offs of this research may result in marathon race organisers moving their jacuzzis to the start of the race rather than the end!

The article on the popliteal vascular entrapment syndrome describes a possible cause of leg pain in young athletes. This article points out that the syndrome is more prevalent than previously believed and that a late diagnosis can have serious consequences for the patient. In contrast, an early diagnosis and surgical correction result in prompt and lasting relief of the symptoms. This article will surely contribute significantly to more clinicians making the correct diagnosis of the condition thus sparing their patients much discomfort, frustration and expense.

Identifying the competitive edge in sport is always a popular topic. Therefore the article on creatine supplementation will be interesting for a wide range of readers. Although the study in this journal focussed primarily on the performance-related effects, the side-effects that a large proportion of the subjects experienced in this study should not go unnoticed.

In summary, this edition of the *Journal* should have something of interest to cater for the needs of all the health professionals and scientists who belong to a multi-disciplinary sports medicine association. This *Journal* is a vehicle for new ideas in sports medicine. You are encouraged to read it, enjoy it and hopefully learn something which can be used to improve performance and reduce the risk of injury!

Mike Lambert
Editor-in-Chief

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ORIGINAL RESEARCH ARTICLE

Cardiovascular responses to self-paced running in warm humid conditions following whole-body precooling

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Abstract

Objective. This study examined the extent to which an attenuated cardiovascular strain during prolonged exercise following precooling might be attributed to changes in either plasma or blood volume.

Design. Seven subjects performed a 30-minute self-paced treadmill run in warm (32°C) humid (60% relative humidity) conditions following whole-body precooling (PC) or no precooling (control (CON)) in a counterbalanced fashion. All subjects were moderately trained and had a mean peak pulmonary uptake (VO_{2peak}) of 60.3 ± 2.4 ml/kg/min/. Blood samples were collected pre and post-exercise for the determination of haemoglobin (Hb) and hematocrit (Hct). Heart rate (HR), rectal temperature (T_{re}) and mean skin temperature ($\overline{T}_{sk}$) were monitored continuously during exercise. Total body sweating (l/h) was calculated from changes in nude body mass and corrected for fluid ingestion.

Results. The distance covered at the end of CON was 6 912 ± 345 m and increased following precooling to 7 263 ± 389 m ($P < 0.01$). On completion of the run CON T_{re} increased to 39.4 ± 0.4°C, while PC T_{re} increased to 38.8 ± 0.4°C ($P < 0.03$). The end of exercise $\overline{T}_{sk}$ was 34.5 ± 0.6°C and 35.6 ± 0.5°C ($P < 0.01$) for PC and CON, respectively. The HR response was lower ($P < 0.05$) for PC at 5 minutes of exercise but not for the remainder of the run. The changes in Hb and Hct resulted in percentage changes in plasma volume (% ΔPV) of –6.9 ± 3.6 for CON and –3.0 ± 5.4 for PC, and percentage changes in blood volume (% ΔBV) of –3.4 ± 1.2 for CON and –1.8 ± 4.1 for PC; these changes were not significantly different between conditions.

Conclusions. Although the subjects significantly increased their performance in warm humid conditions following precooling with an attenuated cardiovascular strain, it is unlikely that changes in either plasma or blood volume contributed to the attenuated cardiovascular strain.

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Introduction

When exercise is performed in a hot environment a severe strain on the cardiovascular system is observed.[1] This strain is usually reflected by the changes in heart rate (HR), stroke volume (SV) and cardiac output (Q).[1,7,23] These cardiovascular dynamics change in order that a finite Q satisfies the metabolic demands of the working muscle and that the skin is highly perfused in order for the body to deal with the accumulating body heat. Moreover, during exercise in the heat cardiovascular drift is exacerbated due to a substantially reduced cardiac filling and SV which require a higher HR in order to maintain Q.

Progressive dehydration as a consequence of exercise, particularly in the heat, can have a significant effect on the cardiovascular system resulting in haemoconcentration and a reduction in blood volume (BV).[8] A reduced BV has been shown to compromise the cutaneous circulation,[5] which diminishes convective heat transfer from the body core to the skin.

Haemoconcentration resulting from running exercise is widely reported.[10] However, variable responses have been shown where some subjects displayed transient haemoconcentration while others displayed transient haemodilution.[3,17,20] Nevertheless, Fortney *et al.*[6] have shown that during 30 minutes of exercise at 60% of maximum aerobic power, a 10% reduction in BV resulted in significantly greater heat storage and core temperature (T_c) with substantially reduced SV and Q and an elevated HR compared with individuals with a maintained BV. Moreover, it is generally accepted that a greater loss of plasma volume (PV) is asso-

ciated with greater increases in T_c, tachycardia and hypotension during exercise heat stress.[14] This is particularly important as a reduced PV has been shown to limit exercise in concert with decreased plasma osmolality, skin blood flow and sweating rate.[19,24] In addition, SV is lower and HR higher in the early stages of exercise when PV is reduced following hypohydration.[22,27] Therefore, minimising the thermal strain during exercise in the heat is extremely important for attenuating a decrement in exercise performance and increasing the safety of exercise under more extreme conditions.

Several studies[12,15,26] have shown the precooling strategy to be beneficial in enhancing endurance performance during moderate and high environmental temperatures. Generally, these studies show cardiovascular and thermoregulatory strain to be reduced substantially during exercise following precooling. However, it is still unclear whether precooling reduces cardiovascular strain during exercise heat stress as a consequence of an attenuated reduction in either PV or BV. Therefore, this study examined to what extent prolonged exercise performance might be improved following precooling and whether that improvement might be in part due to an attenuated cardiovascular strain.

Materials and methods

The thermoregulatory and performance aspects of this study have been published in a companion paper.[2]

Subjects and experimental design

Seven subjects (five men and two women) volunteered for the study. All were competitive runners and apparently in good health as reported by a health history questionnaire and an exercise stress test. None of the subjects reported heat exposure within the preceding 2 months of the study. The mean (± SD) for age, mass, height, body surface area, peak pulmonary uptake (VO_{2peak}) and maximum HR were 25 ± 4.5 years, 66.2 ± 9.5 kg, 171.0 ± 8.8 cm, 1.76 ± 0.15 m^2, 63.5 ± 2.6 ml/kg/min, and 188 ± 7 beats/min, respectively. The experiment was approved by the Ethics in Human Research Committee of Charles Sturt University and all subjects gave written informed consent.

All participants refrained from vigorous exercise, caffeine and alcohol ingestion for at least 24 hours before reporting to the laboratory. During the initial visit the subjects were familiarised with treadmill running, anthropometric measurements were recorded and a maximal incremental treadmill test to exhaustion was undertaken. VO_{2peak} was defined as the highest VO_2 (ml/kg/min) attained over a 1-minute period.

The first subject was randomly assigned to either a run in the heat (control (CON)) or a run in the heat following whole-body precooling (PC). All subsequent subjects were assigned in a counterbalanced fashion. The ambient temperature (T_a) and relative humidity (RH) were set at 32°C and 60%, respectively. Testing was scheduled at least 3 - 7 days apart but at the same time of day so that circadian variation could be minimised. On the day of testing participants reported to the laboratory and rested quietly for approximately 20 minutes, after which a pre-exercise blood sample was drawn and nude body mass measured. A rectal probe was inserted and HR transmitter and skin thermistors secured. Subjects then either commenced their performance run or were immersed in a water bath for whole-body precooling as previously described.[16] During the run subjects ingested a controlled volume of distilled water in an attempt to control for a progressive dehydration effect. Once the run was completed a post-exercise blood sample was drawn, subjects were towelled dry and nude body mass was re-measured.

Precooling manouvre

The method of whole-body cooling has been previously described.[16] Briefly, however, subjects reclined in a water bath to the level of the neck. The initial water temperature was set at 28 - 29°C. After an accommodation period water was siphoned and replaced with cold water (approximately 13°C) until water temperature reached 23 - 24°C. The immersion protocol lasted for 60 minutes or until continuous shivering was observed. Once subjects left the water bath they were towelled dry, prepared for exercise, and commenced running within 3 minutes.

Performance run

The subjects ran on a motorised treadmill. The speed was set by the experimenter to the nearest 0.5 km/h and increased or decreased on demand through previously rehearsed signals. The aim of the test was for subjects to run as great a distance as possible within the allotted 30 minutes. On completion of the run the total distance travelled was recorded. During and following the run participants were not given any feedback regarding their performance other than the self-selection of running speed.

Thermoregulatory measurements and calculations

Rectal temperature (T_{re}) was monitored and measured with a 12-gauge rectal thermistor (Mon-a-therm, Mallinckrodt Medical Inc., St. Louis, MO) inserted 10 cm beyond the anal sphincter. Skin temperature (T_{sk}) was measured at four sites (chest, arm, thigh and calf) with thermistors (427 series, Yellow Springs Instrument, OH) secured with transpore tape. All thermistors were connected to an eight-channel telethermometer (Zentemp 5000, Zencor, Australia). Temperatures were monitored continuously and recorded pre-exercise and at the end of exercise. Mean skin temperature ($\bar{T}_{sk}$) was calculated using the area weighted formula[25]: $\bar{T}_{sk} = 0.3\ (T_{chest} + T_{arm}) + 0.2\ (T_{thigh} + T_{leg})$. Heat storage (S) was calculated from $\bar{T}_{sk}$ and T_{re} using the formula[15]: $S = 0.97 \cdot m \cdot \Delta \bar{T}_B \cdot A_D^{-1}$, where $\bar{T}_B = (T_{re} \cdot 0.65) + (\bar{T}_{sk} \cdot 0.35)$, 0.97 is specific heat of body tissue (W/kg), m is body mass (kg) and A_D^{-1} is surface area (m^2).

Body mass, fluid intake and total body sweating

Change in nude body mass was measured to the nearest 10 g on an electronic precision balance (HW - 100KAI, GEC, Avery Ltd., Australia). Before commencing the performance run a drink bottle was filled with a known quantity of distilled

water (range 500 - 600 ml) so that subjects could drink *ad libitum*. In the subsequent trial subjects were only permitted to drink a similar volume of water to that consumed during the initial trial. At the end of the trial the remaining water volume was subtracted from the initial water volume and recorded. This value was also used to adjust the nude body mass measurement. Water volume was measured to the nearest 1 ml using a graduated 100 ml cylinder.

Heart rate

HR was monitored continuously and recorded pre-exercise, at 5-minute intervals during exercise and at the end of exercise using a Sports Tester (Polar Electro, Oy, Finland).

Blood sampling, analysis and calculations

Pre and post-exercise blood samples were drawn from a superficial vein on the dorsal aspect of the hand using a 21-gauge needle. The pre-exercise blood draw was obtained while subjects were seated. The post-exercise blood sample was obtained within 2 minutes of completion of exercise. In all cases the blood samples were collected while the subject remained seated. Blood was collected in vacutainers containing EDTA for determination of haemoglobin (Hb) and haematocrit (Hct). Haematological variables were quantified using the Coulter principle with a Coulter STER analyser. The percentage changes in blood volume (%ΔBV) and plasma volume (%ΔPV) were calculated using the following equations[4]: $\%\Delta BV = (Hb_1/Hb_2 - 1) \times 100$ (equation 1); and $\%\Delta PV = (((Hb_1)x\ (1 - Hct_2)/\ (Hb_2)\ x\ (1 - Hct_1)) - 1)x\ 100$ (equation 2); where Hb_1 and Hct_1 are pre-exercise values and Hb_2 and Hct_2 are post-exercise values.

Statistics

Statistical analyses were performed using an SPSS for Windows (release 7.5.1) software package (SPSS Inc., 1996). Descriptive statistics were generated for all variables. Student's paired *t*-tests were used to compare pre and post-exercise measurements for within treatments and between conditions. Continuous measurements such as HR were analysed using analysis of variance (ANOVA) for repeated measures on time. When significant main effects were detected Tukey's HSD (honestly significant difference) *post-hoc* procedure was employed to locate the source of significance. Statistical significance was set at $P < 0.05$. Values are reported as mean ± standard deviation (± SD).

Results

Running performance

The distance covered at the end of CON was 6 912 ± 345 m. This result was significantly improved following precooling to 7 263 ± 389 m ($P < 0.01$). The running speeds at each 5-minute interval are shown in Fig. 1. The running speeds were only different at 30 minutes when subjects were able to accelerate from 14.6 km/h in CON to 16.8 km/h ($P < 0.03$) in PC. Although the running speeds were only different at 30 minutes, participants were able to maintain a higher average

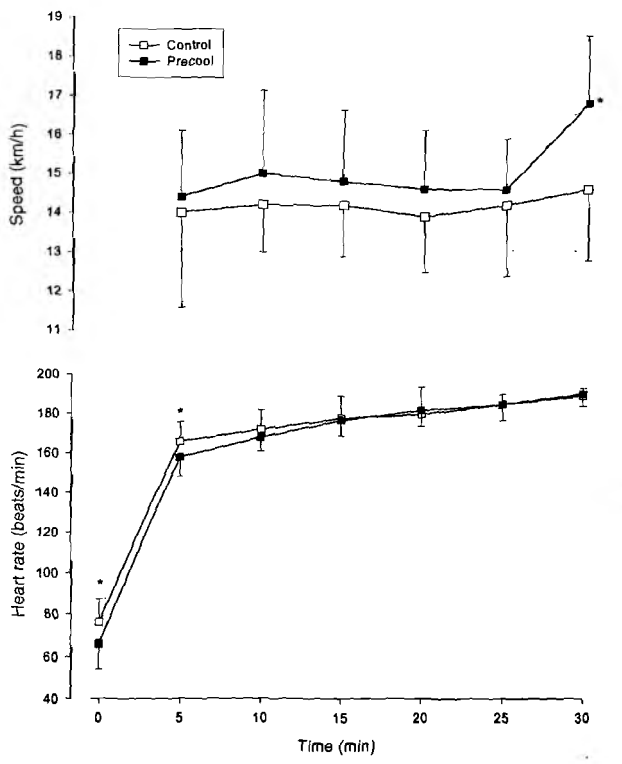

Fig. 1. Mean heart rate response and running speeds at pre-exercise (0 minutes), 5-minute intervals and end-exercise (30 minutes) for control and precool conditions. *P < 0.05 compared with precool.

running speed throughout PC at 15.1 ± 1.5 km/h compared with 14.3 ± 1.5 km/h ($P < 0.01$) for CON.

Thermoregulatory responses

While the pre-exercise T_{re} for CON was 37.4 ± 0.1°C, whole-body precooling reduced pre-exercise T_{re} from 37.3 ± 0.1°C to 36.6 ± 0.6°C ($P < 0.0001$) so that precooled subjects started the exercise bout with a significantly reduced T_{re}. On completion of the run CON T_{re} increased to 39.4 ± 0.4°C, while during PC T_{re} increased to 38.8 ± 0.4°C ($P < 0.03$, Fig. 2). Precooling reduced the pre-exercise $\bar{T}_{sk}$ from 34.1 ± 0.20°C to 28.8 ± 1.6°C ($P < 0.0001$), while the pre-exercise $\bar{T}_{sk}$ for CON was 34.4 ± 0.28°C. The end of exercise $\bar{T}_{sk}$ was 34.5 ± 0.6°C and 35.6 ± 0.5°C ($P < 0.01$) for PC and CON, respectively (Fig. 2). The end of exercise heat storage increased from 62.8 ± 12 W/m² for CON to 124 ± 23 W/m² for PC ($P < 0.05$).

Body mass and total body sweating responses

The pre-exercise values for nude body mass were 63.8 ± 3.1 kg and 63.9 ± 3.1 kg ($P = 0.34$) for CON and PC, respectively. The end of exercise body mass was adjusted for water ingestion and was 63.0 ± 3.0 kg for CON and 63.1 ± 2.9 kg ($P = 0.76$) for PC. The change in body mass for both trials was 0.8 kg amounting to an equal and total body sweating of 1.6 l/h.

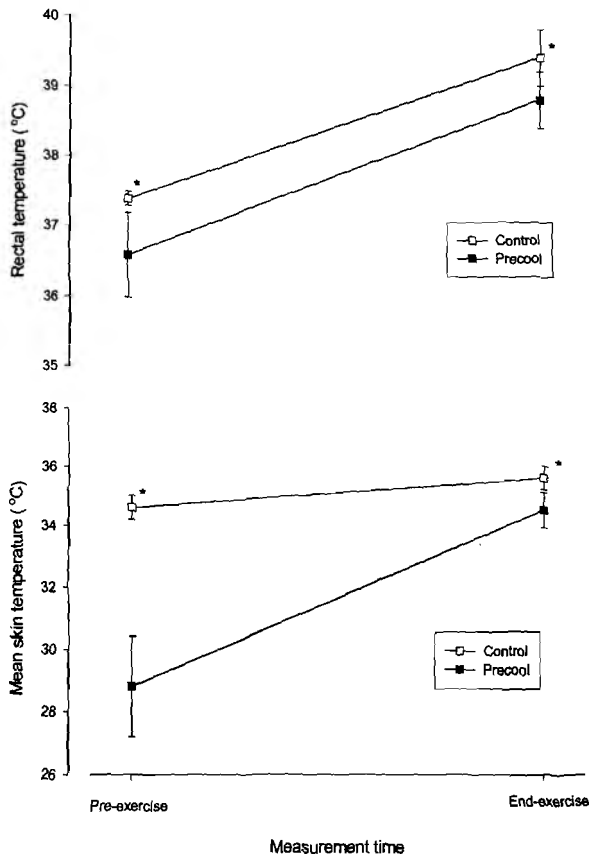

Fig. 2. Mean changes in rectal and mean skin temperatures at pre-exercise and end-exercise.*P < 0.01 compared with precool condition. Pre- versus end-exercise values are all significantly different.

Heart rate response

The HRs at 5-minute intervals are shown in Fig. 1. Resting HR following whole-body precooling was reduced from 75 ± 3 beats/min to 62 ± 4 beats/min ($P < 0.05$). The HR at 5 minutes was significantly ($P < 0.05$) lower at 158 ± 10 for PC compared with 166 ± 10 beats/min for CON, after which it was not different for the remainder of the run between conditions. The end-exercise HRs were similar for CON (189 ± 4 beats/min) and PC (190 ± 4 beats/min).

Haematological responses

The Hb and Hct values are given in Table I. Pre-exercise Hb values were similar for both CON and PC. On completion of the run, Hb concentrations were not significantly altered. The pre-exercise Hct values were also similar for both trials. However, the end-exercise CON Hct increased significantly from 45 ± 0.03% to 47 ± 0.03% ($P = 0.006$) compared with 46 ± 0.04% for PC. The percentage changes in BV and PV are given in Table II. The changes in Hb concentration and Hct did not significantly alter the %ΔPV or %ΔBV for either experimental condition.

TABLE I. Mean (± SD) haemoglobin and haematocrit values for pre and post-exercise ($N = 7$)

	CON		PC	
	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise
Hb (g/dl)	15.3 ± 1.2	15.8 ± 1.2	15.3 ± 1.5	15.6 ± 1.4
Hct (%)	45 ± 0.03	47 ± 0.03*	46 ± 0.04	47 ± 0.04

* $P = 0.006$ compared with CON value.
CON = control trial, PC = precooling trial.

TABLE II. Percentage changes for blood (BV) and plasma volume (PV)

	%ΔBV (CON)	%ΔBV (PC)	%ΔPV (CON)	%ΔPV (PC)
Mean	−3.4	−1.8	−6.9	−3.0
± SD	1.2	4.1	3.6	5.4

CON = control trial; PC = precool trial, %Δ = percentage change.

Discussion

Previous precooling studies have shown that endurance exercise either at a fixed $\%VO_{2max}$ or during either self-paced running or cycling improves exercise performance in moderate and warm, humid conditions.[2,12,15,26] Although traditionally endurance performance has been evaluated using protocols at a fixed workload to exhaustion, it is now apparent that the reliability of such protocols is questionable.[18] In addition, it is now thought that self-paced or stochastic protocols are able to give a better representation of performance enhancement because of potentially higher reliability.[11] Therefore, in the present study a self-paced protocol was used in order to evaluate better the magnitude of improvement in running performance, thereby evaluating the practical application of precooling.

Previous precooling studies[2,12,15] indicate that thermoregulatory and cardiovascular strain are attenuated as a result of the precooling manouvre. However, none of these studies report or examine to what extent the attenuated cardiovascular strain following precooling is influenced by changes in BV or PV. The effects of small alterations in BV and PV on circulatory dynamics during exercise in a hot environment can be critical to performance. The demands placed on the circulatory system during exercise in the heat are partly due to an increased blood flow to the skin to facilitate the dissipation of heat. Additionally, fluid is lost from the vascular compartment, which further compromises cardiac filling.[21]

In the present study, exercise endurance was enhanced following whole-body precooling. This improvement was accompanied by a reduced thermoregulatory and cardiovascular strain. The attenuated thermoregulatory strain resulting from precooling has been previously dealt with.[2,12] Briefly, however, it is clear that precooling enhances the capacity to store heat mainly as a result of the large reduction in skin temperature.[12] A larger capacity to store heat, coupled with the capacity for higher exercise intensity, would enable subjects to complete more work. In fact, the subjects maintained a higher average running speed throughout the run following PC, and it was evident that the majority of gains in perfor-

mance were a result of increasing running velocity toward the end of the trial (25 - 30 minutes). During the early stages of exercise (0 - 5 minutes) HR was significantly lower, after which it was not different. However, the observation that HR was not different between conditions for the remainder of the run (10 - 30 min) indicates that subjects were able to run at a higher intensity with a similar HR, as evidenced by the higher average running speed and significantly greater distance achieved by the precooled subjects in the allotted 30 minutes. However, the end of exercise HR was similar for both conditions. It remains unclear why HR would be similar at the end of exercise given the reduced thermoregulatory strain, increased capacity for heat storage and higher work output for subjects following precooling. This result indicates that subjects performed to their maximum physiological capacity. It is also possible that subjects adopted a pacing strategy that was consistent with reduced cardiovascular and thermoregulatory demands. For instance, it has been suggested that during self-paced exercise, subjects are able to take advantage of pacing rather than performing to an imposed or fixed external workload which would allow the individual to complete the activity with an attenuated physiological strain and reduce the likelihood of premature fatigue.[13]

During prolonged exercise in the heat, haemoconcentration can result as a consequence of progressive dehydration[9] and can ultimately lead to a reduced BV compromising skin blood flow and reducing the amount of convective heat transfer from body core to skin. In the present study the %ΔBV was –3.4% for CON compared with –1.8% for PC, although these values were not significantly different. The non-significant ΔPV of –6.9% for CON compared with –3.0% for PC, indicates that precooling had very little effect on PV. As can be seen from Table I, the percentage changes in PV are mainly a result of the discrete change in Hct, particularly for CON where the post-exercise Hct was significantly elevated compared with PC. This small change in Hct was unable to augment significantly the %ΔPV for CON.

Fortney *et al.*[6] have shown that a 10% reduction in BV can substantially increase the heat storage and T_c during 30 minutes of cycling exercise at 60% VO_{2max} in T_a of 35°C. Our results show that BV was relatively unaffected as a result of exercise in the heat either with or without whole-body precooling. Hence, it is difficult to attribute the attenuated cardiovascular strain to either changes in BV or PV. There are several reasons why we do not find a significant attenuation in reductions of either BV or PV. For instance, during both CON and PC trials subjects ingested similar volumes of water which may have prevented a large and gradual dehydration. Also, while adjusting the change in post-exercise body mass for fluid intake, it was apparent that total body sweating was similar for both trials. However, what is not readily apparent is the greater absolute work rate of the subjects during PC compared with CON. Hence, although water ingestion was similar, the sweat rate during PC was relative to the increased intensity of exercise. This indicates that cardiovascular strain during exercise was attenuated mainly as a result of a significantly reduced $\bar{T}_{sk}$ possibly alleviating the need to increase cutaneous circulation for increased convective heat transfer. Other possible reasons for a reduced cardiovascular strain and increased performance following precooling might be an increased running economy. Although not measured in the present study, oxygen consumption was measured in two other similar studies.[2,12] In both these studies oxygen consumption was similar for control and precooled subjects, and given the increased exercise intensity either by higher running speeds[2] or cycling speeds[12] for a similar metabolic cost, it is quite possible that economy of running in the present study was increased following precooling.

Another difficulty relating to studies quantifying BV and PV changes during exercise is the possible effect of posture. It has long been recognised that posture has a confounding effect on BV.[5] For instance, pre-exercise blood samples taken while seated may indeed result in different BV calculations if a subsequent sample is taken while standing.[4] For this reason the blood samples were taken while subjects were seated during both pre and post-exercise.

Conclusion

In conclusion, the results of this study indicate that whole-body precooling does not induce haematological changes that could account in a significant way for an attenuated cardiovascular strain during prolonged running exercise in warm, humid conditions. Although the benefits of precooling can in part be attributed to attenuated thermoregulatory and cardiovascular strain, it seems that the attenuated cardiovascular strain is primarily a result of a reduced T_c and T_{sk} reducing the need for increased cutaneous circulation.

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ORIGINAL RESEARCH ARTICLE

Does heart rate adequately reflect exercise intensity during mini-trampoline exercise?

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Abstract

Objectives. Quantification of exercise intensity for exercise prescription on the mini-trampoline is difficult, as the relationship between heart rate (HR) and oxygen consumption (VO_2) during mini-trampoline exercise is not clear. The aims of this study were to elucidate the relationship between HR and VO_2 during mini-trampoline exercise, and to compare this with the equivalent relationship obtained during treadmill running over a comparable range of HRs.

Design. Fifteen male subjects aged 17 - 24 years jogged on a mini-trampoline at cadences of 100, 120, 160 and 200 steps/min with a 15 cm leg lift, and at a further workload of 120 steps/min with 90° hip flexion. After a 90-minute rest period, five submaximal treadmill workloads were selected for each subject to give a similar range of HRs to those achieved on the mini-trampoline. Following the fifth workload on the treadmill, subjects continued to exhaustion for determination of peak VO_2.

Main outcome measures. VO_2, HR, minute ventilation, tidal volume, and breathing frequency before and during exercise.

Results. VO_2 relative to HR was significantly lower during exercise on the mini-trampoline ($P < 0.001$). HRs obtained during mini-trampoline exercise overestimated VO_2 by up to 450 ml/min when compared with treadmill exercise at the same HR. The relationship between VO_2 (ml/min) and HR (beats/min) on the treadmill was linear: $VO_2 = 19.99 \times HR - 1\,046$ ($r^2 = 0.97$), while the relationship between VO_2 and HR for trampoline exercise showed a pronounced elevation in HR before any elevation in VO_2. The mean VO_2 while stepping at 120 steps/min with a leg lift of 90° hip flexion was significantly higher than with a leg lift of 15 cm (2.10 l/min v. 1.97 l/min, $P < 0.001$).

Conclusions. These results suggest that the use of HR as a simple monitor of exercise intensity and the use of step frequency as the method of changing exercise intensity during mini-trampoline jogging should be viewed with caution.

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Introduction

Running or bouncing on a mini-trampoline or 'rebounder' has been advocated as a simple means of achieving aerobic fitness and weight loss.[1,2] The mini-trampoline is relatively inexpensive, small, portable, and offers the benefit of a low-impact workout in a confined space. It is therefore suitable for home exercise. Despite the mini-trampoline having been developed in 1938 and patented in 1975, the physiological response to rebounding remains unclear and very little has been published to support the claims of improved cardiovascular fitness and weight loss.[9]

Exercise prescription for improvement of cardiovascular fitness requires quantification of the intensity of the exercise performed. Exercise energy costs on the mini-trampoline reported in five studies[2,4,6,9,10] show a variation of 279%. This indicates that a range of exercise intensities can be obtained on the mini-trampoline.[9] Methods of changing the intensity of mini-trampoline running or bouncing include changing the foot strike frequency,[5,9] the height of leg lift[12] and the addition of simultaneous 'pumping' of hand-held weights.[10] Heart rate (HR) has been proposed as a means of quantifying exercise intensity on the rebounder,[12] and has been used in training studies.[3,4] Target HRs of 70 - 85% of age-predicted maximal HR (HR_{max}) are said to be required to achieve aerobic training using the mini-trampoline.[12]

While the oxygen consumption (VO_2) to HR relationship (VO_2/HR) for treadmill running is well established, it is not well defined for exercise on the mini-trampoline. It is conceivable that exercise involving spring-assisted vertical movement may alter venous return and stroke volume and thereby affect the VO_2/HR relationship. Bhattacharya *et al.*[1] reported a linear VO_2/HR relationship with two-footed bounc-

ing on a 'regular size' trampoline at foot-lift heights of 18 - 100 cm, with no difference between trampoline and treadmill-derived VO_2/HR relationships.[1] At any given HR, the average VO_2 on the trampoline was, however, not significantly lower than the treadmill-derived VO_2.

Gerberich *et al.*[5] measured the relationship between VO_2 and HR during mini-trampoline and treadmill jogging in a group of occupationally sedentary women. A 17% increase in HR with a 14% increase in VO_2 was reported over a range of stepping frequencies from 105 steps/min to 205 steps/min on the mini-trampoline. At rebound jogging cadences of 105 - 165 steps/min, VO_2 (ml/min) was unchanged, while HR rose from 156 to 170 beats/min. This unusual observation was not elaborated upon. In addition, at any given HR the average VO_2 obtained on the trampoline was also lower than that obtained on the treadmill. These findings have not been confirmed, nor have the ventilatory responses to different stepping frequencies been reported.

Data on energy expenditure during mini-trampoline use, which would be useful for optimising exercise prescription for weight loss, are also limited. Reported VO_2 varies from 17 - 40 ml/kg/min.[2,6,10] This broad range is probably a reflection of the variation in mini-trampoline technique, particularly with regard to stepping frequency and leg-lift height.

A limited number of training studies using rebounding exercise have been completed investigating cardiovascular improvements and weight loss. Some report a significant improvement in VO_{2max},[4,11,13] while one showed no significant improvement.[3] Descriptions of mini-trampoline protocols used in these studies are scanty and certainly not standardised between studies with regard to step frequency and step height. Step frequency has varied considerably and leg-lift height is frequently unspecified. Some studies have involved 'rebound aerobics' rather than jogging.[11]

The aims of this study were to investigate the VO_2/HR relationship over a similar range of HRs on both the mini-trampoline and the treadmill and to investigate concurrently the effect of step frequency and leg-lift height on HR, VO_2 and ventilatory parameters while exercising on the mini-trampoline.

Methods

Subjects

Eighteen male subjects aged 17 - 24 years were recruited for this study. All were healthy and active, with a considerable range in the level of daily activity within the group. One subject experienced cramp of the hip flexors during the trial and was unable to complete the testing protocol. HR data were incomplete for two subjects and their results were excluded, leaving 15 subjects for analysis. Informed written consent was obtained from all subjects and the study was conducted with the approval of the Ethics and Research Committee of the Faculty of Medicine, University of Natal.

Procedures

All subjects were familiarised with both mini-trampoline and treadmill jogging before testing. Prior to the commencement of testing, the subjects' height and weight were measured, and a medical history obtained. A multi-stage submaximal exercise protocol was then completed on the mini-trampoline. This was followed by a multi-stage submaximal exercise protocol on the treadmill, which was then extended to elicit a maximal response. It was not possible to randomise the order of the two tests as the treadmill workloads for each subject were assigned according to the range of HRs achieved during the mini-trampoline protocol and because of the maximal nature of the treadmill protocol.

The subjects performed a five-stage submaximal protocol on the mini-trampoline at stepping frequencies from 100 to 200 steps/min (Table I). The duration of the first workload was 5 minutes, with subsequent workloads lasting 3 minutes. There was a 1-minute rest interval between each of the first four workloads and a 5-minute rest interval before the final workload. Step frequency was timed to a metronome. A step height of 15 cm was used for the first four stages and was closely monitored by a designated observer using measured vertical markers. Leg lift for the fifth stage required subjects to flex their hips to 90° during each stride. The step frequency of 120 steps/min chosen for comparison of leg-lift heights was based on Katch *et al.*'s[6] observation that a step frequency of approximately 120 steps/min was the most common naturally selected frequency when mini-trampoline jogging.

TABLE I. Five stage mini-trampoline protocol

Stepping frequency (steps/min)*	Step height (cm)	Duration (min)
100	15	5
120	15	3
160	15	3
200	15	3
120	90° to vertical†	3

* Each foot strike equals one step.
† The subject's hip is flexed to 90°.

Subjects then rested for 90 minutes before undertaking the treadmill protocol (Powerjog EG10). During this time subjects were allowed to drink water. Mean resting HR after 90 minutes of rest was no different to that before the mini-trampoline protocol. Workloads were of the same duration and with the same rest intervals as during the mini-trampoline exercise. Workloads were individually assigned according to the range of HRs achieved by each subject during the mini-trampoline exercise protocol, with knowledge of their treadmill HR response from the familiarisation session. The first two workloads were at walking speeds, with the remaining three at running pace with increases in speed and slope between workloads. After the fifth workload, slope and speed were increased every minute until the subject could no longer continue. In all instances, the HR at the point of exhaustion was more than 90% of the predicted HR_{max} for age and in all but two cases, the respiratory exchange ratio (RER) was greater than 1.05.

The gas exchange analysis was performed using open circuit spirometry (Oxycon Gamma, Mijnardt) calibrated for gas concentrations and volume before every testing session.

Throughout both mini-trampoline and treadmill exercise, minute ventilation (V_E), VO_2 and RER were measured continuously. HR was measured every 5 seconds throughout exercise using telemetry (Polar Sport Tester HR monitor). All values reported are those averaged over the last 30 seconds of the workload. Energy expenditure, based on the thermal equivalent of oxygen adjusted for the RER, was calculated at each workload.[7]

Additional investigations in five subjects confirmed that submaximal mini-trampoline exercise followed by 90 minutes of rest did not influence VO_2 or HR response during the treadmill protocol.

Statistical analysis

Data are presented as mean and one standard deviation (SD). The effect of workload and mode of exercise (mini-trampoline v. treadmill) on VO_2 with regard to HR was determined by two-way analysis of variance with repeated measures. The effect of mini-trampoline stepping cadence on respiratory responses was determined using a one-way analysis of variance (ANOVA). Comparison of leg-lift height was carried out using the Student's paired *t*-test. Correlations between variables utilised Pearson's correlation coefficient. Significance was set at $P < 0.05$.

Results

The subjects' age, mass, height and VO_{2max} are shown in Table II.

Mean HR and VO_2 for each submaximal workload during mini-trampoline and treadmill exercise and the maximal data from the treadmill exercise test to exhaustion are presented in Table III. There was an increase in both VO_2 and HR with increased stepping frequency (100 - 200 steps/min) on the mini-trampoline and with increased workload on the treadmill.

TABLE II. Subjects' characteristics ($N = 15$)

Characteristic	Mean	Range
Age (yrs)	20.9 (1.8)	17 - 24
Mass (kg)	63.6 (4.7)	58.2 - 73.8
Height (cm)	170.9 (3.7)	162.5 - 176.7
VO_{2max} (ml/kg/min)	52.3 (5.9)	40.9 - 59.6
VO_{2max} (l/min)	3.32 (0.42)	2.55 - 3.83

In order to compare VO_2 relative to HR during the two modes of exercise, the oxygen pulse (VO_2 per heart beat) was examined. For ease of comparison, treadmill VO_2 values were modelled for the exact HRs obtained during mini-trampoline exercise, using the treadmill VO_2/HR relationship derived from the treadmill linear regression equation in Fig. 1. The differences in VO_2 and oxygen pulse between trampoline and treadmill exercise at any given HR are shown in Table IV.

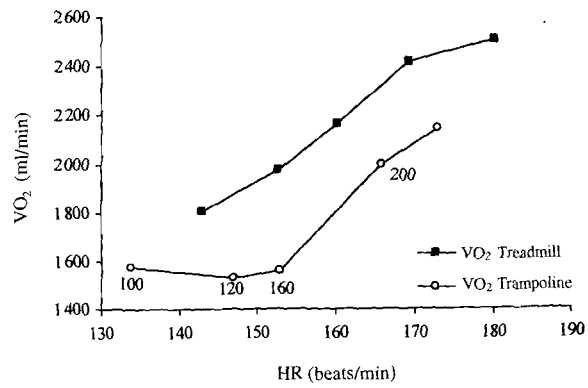

Fig. 1. Relationship between HR and oxygen consumption on treadmill and on the mini-trampoline at stepping frequencies of 100, 120, 160 and 200 steps/min and 200 steps/min with 90° hip flexion. The regression equation for treadmill data is VO_2 (ml/min) = 19.99 x HR – 1 046.

Two-way ANOVA indicated a significant effect of mode of exercise on the oxygen pulse, with a lower result during trampoline exercise ($P < 0.001$). Thus at any given HR, VO_2 was higher on the treadmill than on the mini-trampoline. The magnitude of the difference is shown in Table IV.

Although there was no significant interaction effect of mode of exercise and increasing exercise workload on oxygen pulse ($P = 0.08$), the data displayed in Fig. 1 reflect the trend toward a different slope of the VO_2 v. HR relationship. During treadmill exercise, HR and VO_2 both increased from workload 1 through to maximum workload, resulting in a linear relationship (VO_2 (ml/min) = 19.99 x HR (beats/min) – 1 046, $r^2 = 0.97$). During mini-trampoline exercise, HR was increased with each increase in stepping frequency, while VO_2 only increased significantly when the step frequency was raised to 200 steps/min ($P < 0.01$). This was an unex-

TABLE III. Heart rate and oxygen consumption during mini-trampoline and treadmill exercise, expressed as mean and 1 SD

	Mini-trampoline			Treadmill	
Steps/min	Heart rate (beats/minutes)	VO_2 (l/min)	Stage	Heart rate (beats/min)	VO_2 (l/min)
100	134 (19)	1.57 (0.36)	1	143 (14)	1.81 (0.16)
120	147 (17)	1.53 (0.32)	2	153 (16)	1.97 (0.18)
160	153 (19)	1.56 (0.25)	3	160 (17)	2.17 (0.23)
200	166 (19)	2.00 (0.29)	4	169 (17)	2.41 (0.19)
120/90°	173 (15)	2.14 (0.27)	5	180 (15)	2.50 (0.18)
			Max	201 (6)	3.32 (0.42)

TABLE IV. Comparison of mean oxygen consumption and oxygen pulse at HRs obtained during trampoline exercise. The mean treadmill oxygen consumption is calculated from the regression equation derived in Fig. 1, and the mean treadmill oxygen pulse is calculated from the modelled treadmill oxygen consumption

HR trampoline (beats/min)	VO_2 trampoline (ml/min)	VO_2 treadmill (ml/min)	Difference (ml/min)	O_2 pulse trampoline (ml/beat)	O_2 pulse treadmil (ml/beat)	Difference (ml/beat)
134	1 570	1 634	64	11.72	12.19	0.48
147	1 530	1 894	364	10.41	12.88	2.47
153	1 560	2 014	454	10.20	13.16	2.97
166	2 000	2 274	274	12.05	13.70	1.65
173	2 140	2 414	274	12.37	13.95	1.58

pected finding and more intermediate stepping frequencies are required in order to describe the curve with precision. The mean HR and VO_2 obtained at a step frequency of 120 steps/min with the hips flexed to 90° on the mini-trampoline was significantly greater than with a foot lift of 15 cm ($P < 0.001$) (Table III). The mean energy expenditures at the different step frequencies are shown in Fig. 2.

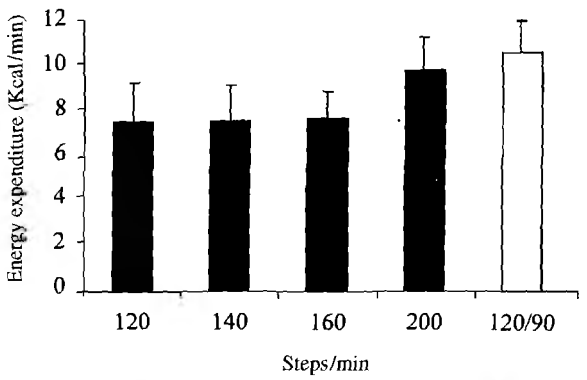

Fig. 2. Energy expenditure at the different stepping frequencies expressed as mean and one standard deviation.

The ventilatory changes during mini-trampoline jogging are shown in Table V. With increasing step frequency on the mini-trampoline, V_E and respiratory rate increased significantly ($P < 0.001$), in a fashion best described by a third-order polynomial, although more points are required to describe this relationship with precision. In contrast, tidal volume was not significantly increased over the range of step frequencies used in this study despite the increase in V_E, which must therefore be accounted for primarily by the increases in breathing frequency ($P < 0.001$). At 120 steps/min with higher leg lift, the respiratory rate was less than at 200 steps/min, despite the 120 steps/min with 90° hip flexion workload provoking a higher V_E and VO_2.

The relationship between $\%HR_{max}$ and $\%VO_{2max}$ differs between the two exercise modalities. When exercising on the trampoline, a higher percentage of HR_{max} is required to achieve a $\%VO_{2max}$ comparable with that achieved on the treadmill. For example, 75% HR_{max} represents 60% VO_{2max} during treadmill exercise, while during mini-trampoline exercise, 75% HR_{max} equates to only 47% VO_{2max} (Fig. 3). The percentage of HR_{max} utilised by individuals when exercising on the mini-trampoline at step frequencies of 120 steps/min and 160 steps/min shows a weak relationship with individuals' VO_{2max} (Table VI).

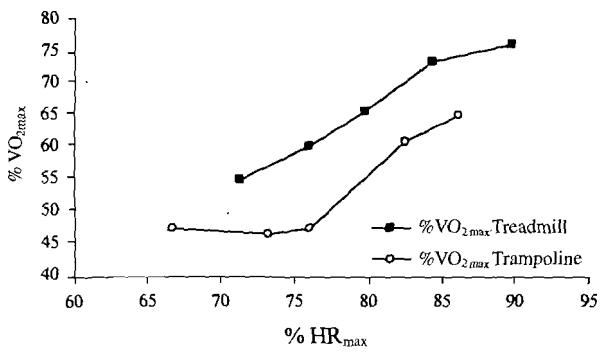

Fig. 3. The relationship between $\%HR_{max}$ and $\%VO_{2max}$ on treadmill and on the mini-trampoline.

TABLE V. Ventilatory results during mini-trampoline exercise expressed as mean and 1 SD

Steps/min	V_E (l/min)	V_T (l)	f (breaths/min)	Steps/breath	V_E/VO_2
100	32.6 (9.3)	0.97 (0.30)	33.1 (10.4)	3.0	20.8
120	38.4 (11.1)	0.99 (0.27)	38.0 (12.7)	3.2	25.1
160	40.0 (8.6)	1.00 (0.27)	40.0 (12.4)	4.0	25.6
200	52.7 (12.7)	1.17 (0.33)	45.6 (16.8)	4.4	26.4
120/90°	54.4 (9.4)	1.32 (0.30)	42.1 (13.2)	2.9	25.2

V_E = minute ventilatory volume; V_T = tidal volume; f = respiratory rate; V_E/VO_2 = oxygen equiavalent.

TABLE VI. Correlation of the percentage of HR_{max} with the perecntage VO_{2max} achieved on the mini-trampoline

$\%HR_{max}$ at 100 steps/min	$r = -0.42$, NS
$\%HR_{max}$ at 120 steps/min	$r = -0.58$, $P < 0.05$
$\%HR_{max}$ at 160 steps/min	$r = -0.55$, $P < 0.05$
$\%HR_{max}$ at 200 steps/min	$r = -0.31$, NS
$\%HR_{max}$ at 120/90° steps/min	$r = -0.10$, NS

NS = not significant.

Discussion

In a review of rebounding exercise and cardiorespiratory fitness, Smith and Bishop made the following assertion, 'Obviously the rate at which a subject jogs on a rebounder will influence the energy cost'.[9] In the present study, at a foot lift of 15 cm, an increase in the jogging rate did not, however, provoke an increase in VO_2 or energy expenditure except at the highest step frequency of 200 steps/min.

As the mini-trampoline is a popular exercise modality in the home, where the simplest objective measure of exercise intensity is HR, a knowledge of the VO_2/HR relationship is important. VO_2 was consistently lower during exercise on the mini-trampoline when compared with treadmill exercise at a comparable HR. In addition, the VO_2/HR relationship observed while jogging on the mini-trampoline was non-linear, with the HR relative to the measured VO_2 being disproportionately high at stepping frequencies of 100, 120 and 160 steps/min. As no measurements were taken between 160 and 200 steps/min, it is not possible to clarify the point or nature of the inflection in the VO_2/HR curve. This finding partially supports the findings of Gerberich *et al.*'s study[5] of untrained women, which did not illustrate a significant increase in VO_2 even at the higher workloads.

The cause of the increasing HR with an unchanging VO_2 is unclear. If the constant VO_2 reflects a constant cardiac output, then the increase in HR may be explained by a reduction in stroke volume secondary to possible changes in venous return. Venous return may be altered by vertical movement and reduced calf muscle pump activity or breathing pattern relative to vertical movement. This response requires further investigation.

The VO_2 and associated energy expenditure observed in the current study fell within the range of values reported in previous studies.[5,10] At the lower three stepping frequencies, VO_2 was below that recommended for improvement in cardiovascular fitness.[8] Even at a stepping frequency as high as 160 steps/min, the mean VO_2 of 1.56 l/min (24.5 ml/kg/min), represented 47% of VO_{2max} with a range of 27% VO_{2max} - 68% VO_{2max}. This exercise intensity is unlikely to result in significant improvements in cardiovascular fitness, nor is it likely to be associated with substantial weight loss.

Use of HR target zones for training has become popular, and 70% HR_{max} is often quoted as the threshold level above which aerobic training effects will occur. Extrapolation of data from the mini-trampoline and treadmill $\%HR_{max}/\%VO_{2max}$ curves indicate that a level of VO_2 comparable with that achieved at 70% HR_{max} on the treadmill is achieved at approximately 80% HR_{max} on the mini-trampoline. HRs in excess of 80% HR_{max} were only achieved at a step frequency of 200 steps/min with a 15 cm lift, and at 120 steps/min with 90° hip flexion.

The height of leg lift significantly affects VO_2, and at 120 steps/min an increase in the leg lift from 15 cm foot lift to 90° of hip flexion resulted in an increase in the VO_2 of 41% ($P < 0.001$). This is in contrast to an increase in VO_2 of only 3% when stepping frequency was increased from 120 to 160 steps/min. A significant 31% increase in VO_2 was obtained when stepping frequency was increased to 200 steps/min ($P < 0.001$), but this stepping frequency requires considerable co-ordination and motivation from the subjects. Studies investigating the use of hand-held weights while mini-trampolining have reported an increase in energy expenditure of a similar degree, dependent upon the weight and the 'pumping' height. Increasing the height of leg lift may be a suitable and simpler alternative to the addition of hand-held weights.[10]

Energy utilisation at HR zones commonly associated with training and aerobic weight reduction programmes is lower in trampoline jogging than in treadmill running and may be insufficient to achieve the desired effect. The use of HR as a simple monitor of exercise intensity and the use of step frequency as the method of changing exercise intensity during mini-trampoline jogging should be viewed with caution. Further studies of ways to increase exercise intensity and VO_2 using the mini-trampoline are required, and the cause of the reduction in VO_2 relative to HR in trampoline jogging warrants further investigation. Traditional target HR zones need to be reassessed for use with mini-trampoline jogging.

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ORIGINAL RESEARCH ARTICLE

Clustering of athletic ability in male Kalenjin scholars

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Abstract

Aim. The present study aimed to establish any biological or socio-cultural differences between a group of runners and non-runners attending high school in the heart of Kenya's Kalenjin-speaking region.

Methods. A case-controlled demographic and anthropometric study was performed on schoolchildren at St Patricks School, Kenya. Two sample groups were used. The first group consisted of pupils who participated in athletics, and the second group consisted of pupils who did not.

Results. Eighteen of the runners had at least one first-degree relative involved in competitive running, while of the non-runners, two had a first-degree relative running competitively. Runners tended to be heavier and taller than non-runners and jumped significantly further in the standing long jump test.

Conclusions. These data could support the hypothesis that among the Kalenjin Kenyan tribe, there exists on a hereditary basis specific lineages with superior running ability. Alternatively, the social-cultural hypothesis that a proven family history of running ability encourages potential adolescent runners to follow the familial example may also be supported by these findings.

Introduction

There is substantial speculation that specific biologically defined populations have genotypes that enable them to succeed in given athletic events at a rate disproportional to those without the speculated genotype.[4] Areas of focus include the competitive successes of West African sprinters and East African long distance runners,[2,8,11] and elite black male South African long distance runners.[3,13,14] A previous study[3] showed that elite black male South African long distance runners demonstrated 'superior fatigue resistance' to their non-black counterparts. This finding could explain why 90% of the top positions in South African road races from 5 to 56 km are filled by black athletes,[3] who compromise less than 20% of the South African running population. As early as 1944, the South African scientist Ernst Jokl[5] speculated that: 'Serious consideration should be given to the hypothesis that the Negro muscle — in contrast to the muscles of whites — is a superior machine, producing from a given amount of fuel more energy and less heat'.

In recent years Kenyan runners have dominated long and middle distance running events.[6,9,12] Kenya's more than 40 indigenous languages, which correspond closely with ethnic groups or tribes, are commonly classified as Bantu, Nilotic or Cushitic. Approximately three-quarters of Kenya's international runners come from a Nilotic group known as the Kalenjin, who comprise about 11% of the Kenyan population.[7,15]

Manners[6] has suggested that the conventional explanations for the extraordinary competitive success of Kenyan runners — living at 2 000 m altitudes, enjoying the ideal climate of the Kenyan highlands, subsisting on a high carbohydrate diet, using walking or running as a principal mode of transportation and strongly motivated by the material rewards available to successful runners — do not adequately explain the concentration of running success among the Kalenjin. He invokes the possibility of genetic predisposition to running ability based on a collection of case studies and the hypothesis that customs peculiar to the Kalenjin people may have acted as selective pressures towards such genetic ability. These include cattle raiding, which called for long treks where speed and endurance were essential, and which was rewarded by increased ability to pay for brides. If this hypothesis is correct and the relevant customs have been practised for many centuries, one would expect running ability to be distributed throughout the Kalenjin population, as indeed Manners[6] and his informants believe. However, based on the high degree of biological relations among internationally successful Kalenjin runners, there is also the suggestion that even within the Kalenjin and perhaps other Kenyan populations, specific lineages with superior running ability may exist.

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Eldoret is the second largest town in the Rift Valley province. Near Eldoret lies Iten, home of St Patrick's school, which has, over the years, produced a disproportionate number of elite Kenyan runners.[12] St Patrick's has developed a limited policy of preferentially admitting athletically talented pupils whose academic standard may otherwise have precluded their entrance into the school. The present study aimed to establish whether there were any biological or socio-cultural differences between a group of runners and non-runners, all of whom attend St Patrick's school in the heart of Kenya's Rift Valley province.

Material and methods

The case-control study was conducted at St Patrick's School, Iten, Kenya. The former headmaster and running coach at the school and coach to a number of other elite Kenyan athletes (CO'C), administered a standard questionnaire to 50 male students at the school between the ages of 15 and 21 years. Two sample groups were used. The first (N = 25) was composed of pupils who participated voluntarily in athletics as an extra-curricular activity. The second sample (N = 25) consisted of pupils who did not participate in athletics. All these boys were born and grew up in Kenya's Rift Valley. The questionnaire detailed: (*i*) demography, including questions regarding parents' highest level of education; (*ii*) whether there were any first-degree relatives who participated in competitive running and at what level; (*iii*) tribal background dating back to maternal and paternal grandparents; and (*iv*) what students planned to do after leaving school. A standing long jump test was also administered to all participants. The test required both feet to be flat on the ground, shoulder width apart, toes just behind the line, knees at 120°, and arms to the sides. A counter movement of the arms was allowed immediately before the jump. The jump was measured to where the heels landed. Three opportunities were given, and the best result was recorded. Additionally, pupils were weighed to the nearest kilogram, and height was measured to the nearest centimetre. This information was used to calculate the body mass index (BMI) (kg/m^2).

A standard chi-square analysis was performed to assess the significance of differences between variables in the two groups for each of the following: related running relatives, tribal background, level of parental education, and intention to pursue further education. A Student's t-test was performed to assess the significance of differences between BMIs of runners and non-runners, long jump differences, height and weight differences, age differences, and differences between centimetres jumped per kilogram of body weight.

Results

Of the 25 runners, 23 could confidently trace their origins to Kalenjin or one of the Kalenjin sub-tribes, bilaterally for two generations. Twenty-one of the non-runners could do likewise. There was no significant difference between the tribal background of runners and non-runners. Of the runners, 16 had at least one parent with post-primary school education, while 19 of the non-runners had at least one parent with post-primary school education. This difference was not significant (Table I).

Eighteen of the runners had at least one first-degree relative involved in competitive running. These 18 included 4 international, 4 national, 2 provincial, 5 district and 1 zonal runner(s). Of the non-runners, 2 had at least one first-degree relative running competitively, 1 at provincial level and 1 at district level. This difference was significant (P < 0.001, Table I).

Ten runners intended to seek post-secondary school education, while 24 non-runners had a similar intention. This difference was significant (P < 0.001, Table I). Of the 15 runners who did not intend to seek such education, 6 hoped to become professional athletes, and 9 hoped to seek employment. When the 6 intending to become professional athletes were removed from the group, leaving 10 of 19 runners intending to pursue post-secondary education, the number was still significantly (P < 0.001, Table I) less than the 24 of 25 non-runners intending to pursue such education.

When the runner sample was broken down into runners competing at provincial level and above and those competing below this level, 4 of the 11 runners competing at the higher level intended to pursue a career in athletics compared with 2 of the 12 runners competing at the lower level. This difference was not significant.

TABLE I. Socio-cultural comparison between runners and non-runners attending St Patrick's School, Iten, Kenya

Category value)	Runners (N)	Non-runners (N)	Significance (P-
Exclusively Kalenjin background over two generations	23/25	21/25	NS
One or more first-degree relatives running competitively	18/25	2/25	< 0.001
One or more parents with post-primary school education	16/25	19/25	NS
Intention to seek post-secondary education	10/25	24/25	< 0.001
Intention to seek post-secondary education, excluding subjects intending to become professional athletes	10/19	24/25	< 0.001

TABLE II. Anthropometrical data (mean (SD)) for runners and non-runners

Measurement	Runners	Non-runners	Significance (*P*-value)
Age (years)	17.6 (1.114)	16.8 (1.218)	< 0.05
Height (m)	1.74 (0.06)	1.65 (0.08)	< 0.001
Weight (kg)	57.9 (5.29)	52.5 (8.27)	< 0.05
Body mass index (kg/m^2)	19.06 (1.28)	19.09 (2.21)	NS
Best standing long jump (m)	2.29 (0.21)	1.90 (0.21)	< 0.001
Long jump/weight (cm/kg)	3.97 (0.39)	3.71 (0.78)	NS

Table II lists the anthropometrical data of runners and non-runners and their standing long jump performance. The two groups were not ideally matched as runners were significantly older, heavier and taller, but BMIs were not different between the two groups. Runners also demonstrated less variation in body mass than the non-runners. Runners jumped significantly further in the standing long jump, but this difference disappeared when results were corrected for differences in body mass.

Discussion

The finding that the tribal backgrounds of runners and non-runners were not significantly different suggests that both samples were indeed from one population. Accordingly, the most notable finding of this study was that the schoolboy runners had an overwhelming preponderance of first-degree relatives also involved in competitive running. In contrast, only 2 (8%) of the schoolboy non-runners had a close relative participating in competitive running.

St Patrick's longstanding reputation for producing outstanding runners, many of whom have achieved international success, has made it *the* institution of choice in the Rift Valley province for boys who have serious running ambitions. In view of this, the marked difference in the figures for relatives involved in competitive running suggests one of two possible interpretations. It can be argued[6] that the difference results from family differences in role models and encouragement — boys encouraged to run by other runners in the immediate family are more likely to seek education at St Patrick's and to participate in athletics after joining the school. Or, alternatively, perhaps instead of or in addition to these social effects, the data offer strong support for the initial hypothesis that among the Kalenjin there exists on an hereditary basis specific lineages with superior running ability. It should be noted that athletics at St Patrick's is open to all scholars, and the rewards earned by successful runners, namely opportunities to travel and possible scholarships to American Colleges or professional running careers, are familiar to every boy in the school. Yet the school's runners come predominantly from families whose members include other competitive athletes. However, there are important limitations to this study that need to be recognised.

First, neither of the two sample populations was randomised, nor were the two samples well matched for age. Second, the reliability of the measuring instruments is not known. Finally, we failed to establish what percentage of the runner sample was attending the school as a result of St Patrick's preferential admissions policy.

In view of these considerations, these results should be considered provisional pending a similar study conducted by researchers using larger randomised age-matched samples and certified measuring equipment, taking into account the number of preferentially admitted athletes. If the present results are confirmed, a further study would be needed to test the hypothesis that lineages with superior running ability exist on a biological basis. This study would need to perform physiological tests on the younger siblings of families with and without histories of athletic excellence before the siblings had started running, and therefore before they had experienced a training effect.

Surprisingly, the BMIs of the two groups were essentially identical. As the teenage years are the most metabolically expensive of an individual's life, and as running would add a further metabolic stress, one would expect the runners to have lower BMIs. This is especially true for Kenyans in Iten who must subsist, for economic reasons, on a high-bulk, low-caloric rural diet.[12] It may be that the runners simply ate more than the non-runners, as food at St Patrick's is not strictly rationed and the kilojoule intake of the populations was not monitored. Alternatively, the runners' metabolic rates may have adapted to higher energy demands and a relative caloric deficiency.

Runners were taller and heavier than non-runners. This could be because runners were older, although growth velocity generally declines in the late teens.[10] Alternatively, runners might comprise a separate population within the Kalenjin-speaking people or they may indeed have enjoyed better than average nutritional circumstances.

The significantly better performance of the runners in the long jump may be explained by their greater height and presumed greater stride length. An alternate explanation would be that the runners were more powerful, again either on a genetic basis or as a result of their training.

The finding that a disproportionate number of athletes competing at provincial level and above intended to pursue a career in athletics suggests that running performance may influence career choice. The knowledge that close relatives pursued a running career would probably act as the spur to start running. However, new runners must also be aware of the large number of Kalenjin runners who do not achieve international success or for whom international success does not guarantee long-term financial security.[1,6,12]

This explanation, however, does not tell us why runners not planning to pursue a career in athletics do not intend to pursue post-secondary school education. This is especially surprising since the educational levels of the parents of the runners and non-runners were similar, and the non-runners

overwhelmingly intended to pursue such education. This finding may be an artefact of St Patrick's preferential admissions policy, in that a disproportionate number of the running subjects may have demonstrated lower academic capacity from the start. Alternatively, running performance could be a better predictor of career choice in Iten than the level of parental education.

In summary, if the localisation of athletic talent in circumscribed areas of the Rift Valley of Kenya[1] is due to the biological pressure of a high altitude rural lifestyle, then one would expect running ability to be distributed throughout the population resident in that area. But this study seems to identify a group of runners from the same population as their non-running counterparts with similar opportunity to participate in running at school, but with a marked difference in the number of biological first-degree relatives who are competitive athletes, with similar BMIs despite presumed increased energy expenditure from participating in running, and with similar role models in terms of level of parental education, yet dissimilar goals in terms of education.

The argument for a biological basis for this finding is suggested by the strong familial links, presumed more efficient metabolism, superior relative absolute long jump ability, and similar parental role models in a population that is otherwise identical.

Alternatively, an equally convincing socio-cultural explanation can be made by arguing that a proven family history of running ability encourages potential adolescent runners to follow the familial example, especially as the prospect of financial success for children born in a poor rural community will be a profound motivator. Further, it is possible that lineages with superior running ability, if they do indeed exist, may have been formed through a culture of running. Until very recently, social pressures governing mate selection cannot have related directly to competitive running which has assumed social significance in Kenya in the last 50 years. But success in the possibly related enterprise of cattle raiding has long been a significant factor in mate selection[6] and could have resulted in the observed concentrations of ability.

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ORIGINAL RESEARCH ARTICLE

Popliteal vascular entrapment syndrome — a cause of leg pain to be considered in young athletes

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Abstract

Objective. To provide an explanation for the symptoms experienced by, and clinical approach to patients presenting with foot and calf pain or paraesthesiae brought on by exercise when such symptoms are due to popliteal vascular entrapment.

Design. In this study the clinical features of 93 instances of popliteal vascular entrapment occurring in 51 patients over an 11-year period are presented. In addition, the embryology of the popliteal artery is reviewed and its relevance to the development of popliteal entrapment explained.

Setting. The study was conducted in a single major vascular surgical centre in the Johannesburg area, and the cases were drawn from all the vascular surgeons practising at that centre.

Interventions. In patients suspected of suffering from popliteal vascular entrapment, the diagnosis was confirmed angiographically. Patients were subjected to release of the entrapment mechanism if the underlying artery had not yet undergone occlusion, or to replacement of the popliteal artery if the artery was occluded.

Main outcome measures. The diagnosis was confirmed by the operative findings, and the adequacy of treatment was determined by the extent of relief of symptoms and return to sporting activities postoperatively.

Results. Bilateral popliteal entrapment was found in 42 of 51 patients. The mean age at the time of presentation was 34.9 years (SD 11.6 years). Claudication was the most frequent presenting symptom (75 of 88 limbs). Types I, II, III and IV popliteal entrapment were found in 61 limbs (15 arteries occluded), while 32 limbs (3 occlusions) presented with a 'functional' popliteal artery entrapment (apparent absence of a developmental anatomical abnormality). Of 18 limbs with severe ischaemia and associated occlusion of the popliteal artery, 15 underwent bypass grafting with reversed saphenous vein grafts. All replacement vein grafts remained patent during follow-up (median 4.2 years, range 1 - 11 years). One patient was treated with vein patch angioplasty which re-occluded within 6 months and required vein graft replacement.

Conclusions. The popliteal entrapment syndrome is much more prevalent than has formerly been appreciated. Failure to appreciate the diagnosis when the patient presents with early symptoms usually results in progression of the pathology of the entrapment to the point where degenerative changes in the entrapped vessel culminate in occlusion and thrombosis. Correct diagnosis and surgical intervention before the development of thrombosis at the site of the entrapment results in prompt and lasting relief of symptoms, and has the additional major benefit of preventing further degeneration of the involved artery.

On the basis of observations made in this series and in the surgical literature, surgical correction is advised in all cases of types I, II, III, and IV entrapment at time of diagnosis to avoid occlusion as a result of continued arterial wall degeneration. On the other hand, in those patients presenting with symptomatic 'functional' entrapment, surgery is only advised if the symptoms are typical and severe, since up to 50% of the normal population demonstrate transient popliteal artery compression with extremes of plantar- or dorsiflexion.

On the basis of the severe histological changes found in those popliteal arteries that have undergone occlusion at the time of presentation, it is advised that the popliteal artery should be completely replaced, ideally by a vein graft, when significant degeneration or occlusion of the popliteal artery is noted at the time of operation.

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Introduction

The young athletic individual who presents with symptoms of leg and foot pain on exercise presents a problem in diagnosis and management for the clinician. Full and correct evaluation and investigation of these patients usually permits an accurate diagnosis to be made,[15] resulting in correct management and consequent alleviation of symptoms. In the young athlete presenting with typical claudication-like symptoms of the calf and foot, popliteal vascular entrapment

is an important and reversible cause of leg pain and paraesthesiae, and may be the underlying cause of the claudication symptoms in up to 60% of cases[51,60] in some series. Unlike most other common causes of leg pain, popliteal vascular entrapment, if not correctly diagnosed and managed, may well result in occlusion of the popliteal artery with the development of an ischaemic threat to the limb.[51,64] It is therefore important for all health professionals who deal with patients presenting with leg pain on exercise, to have a good appreciation of the manifestations and natural history of the popliteal vascular entrapment syndrome. Failure to make the diagnosis when the condition is the cause of the presenting symptoms may result in the condition progressing to popliteal artery occlusion and even to the development of critical ischaemia of the leg. On the other hand, correctly diagnosed and treated, the long-term prognosis is good, with the majority of individuals returning to their normal sporting activities.[51]

First described by a medical student[73] in 1879 following dissection of an amputated leg, popliteal artery entrapment syndrome was considered by early authors to be a rare phenomenon. After Hamming[41] described the first clinical case in 1958, various isolated case reports were published.[1,3,12,13,20,26,28,33-35,42-44,52,63,75] In the mid-1960s the term 'popliteal artery entrapment syndrome' was introduced[8,11] to describe the condition. Servello[71] first drew attention to the reduction in palpable distal pulses frequently observed when patients with this condition performed forced plantar- or dorsiflexion. Biemans and Van Bockel,[7] in an extensive review of the literature in 1977, focused attention on the clinical syndrome of popliteal vascular entrapment.

Early authors believed the popliteal artery entrapment syndrome to be rare,[7,12,17,19,22,36,37,47,52,64,72,74] but it has become apparent that the condition is considerably more common than previously appreciated.[8,17-19,22,29,31,35,45,47,56,61,62,67,68] While the incidence of the condition in the general population is not known, Bouhoutsos and Daskalakis,[8] in the first large series of cases described, reported an incidence of 0.165% in young males entering the Greek military service. In a post-mortem study Gibson et al.[37] found a prevalence of 3.5%. Unfortunately, a substantial proportion of normal individuals will compress or occlude their popliteal artery with forced plantarflexion or dorsiflexion and will have no symptoms, a phenomenon that has precluded the use of non-invasive tests or duplex Doppler being used as a potential screening tool for accurately evaluating the true occurrence of popliteal artery entrapment syndrome in the asymptomatic general population.[2,14,21,27,65,77]

The entrapment mechanisms has been documented to involve the popliteal vein in up to one-third of cases.[7,19,36,37,42,63,75] The bilateral occurrence of the condition was at first assumed to be rare, but recent literature indicates a high prevalence of bilateral disease.[12,17,22,28,37,64] Popliteal artery entrapment syndrome has been reported to occur in more than one individual in a family.[72]

Classification and embryology

Early attempts to classify the various types of popliteal artery entrapment syndrome were based on the anatomy observed at operation.[8,47] Better appreciation of the embryology of the leg arterial supply, and how the development may vary resulting in different types of vascular entrapment, led to a more rational classification based on the developmental anatomy, with five types of popliteal artery entrapment syndrome[61,64] currently described.

Embryology

During development in the human, with limb bud rotation medially and extension of the knee, the medial head of the gastrocnemius muscle migrates from its original lateral position[6,16] and moves across the popliteal fossa. With further development the definitive attachment of the medial head of the gastrocnemius muscle is to the posterior surface of the medial femoral condyle.

The embryological popliteal artery in the developing limb bud is the continuation of the primitive axial or ischiadic artery.[69,70] The proximal portion of the adult popliteal artery develops from the femoral artery[16] by fusion of the developing femoral arterial plexus and the axial popliteal artery. The mid-portion of the definitive popliteal artery is directly derived from the remnant of the axial artery. The primitive distal popliteal axial artery, lying deep to the forming popliteus muscle, disappears at about the 20 - 22 mm stage of the embryo, while the definitive distal popliteal artery forms superficial to the popliteus muscle by the fusion of two new vessels (the newly anterior and posterior tibial vessels) after the medial head of the gastronemius has migrated medially across the popliteal fossa. During the development of the popliteal fossa, the medial head of the gastrocnemius therefore migrates through the popliteal fossa at about the same time as this rearrangement of the arterial structures.[16]

Classification of popliteal vascular entrapment syndrome

If the definitive distal popliteal artery forms before the migration of the medial head, the newly formed popliteal artery may be swept medially with the definitive artery now lying medial to the normally placed medial head of the gastrocnemius muscle. This results in the type I popliteal entrapment with a marked medial deviation of the popliteal artery in the popliteal fossa, both anatomically and on angiography, as depicted in Fig. 1 (type I). Compression of the artery then results from pressure by the gastrocnemius tendon.

Alternatively, a prematurely formed definitive distal popliteal artery may arrest the medial migration of the medial head, resulting in a type II entrapment with the medial head of the gastrocnemius now more laterally placed than normal. In the type II entrapment, the popliteal artery is medially displaced to a lesser degree, and lies deep and medial to the medial head of the gastrocnemius muscle, which attaches more laterally on the medial femoral condyle or intercondylar area. The artery therefore lies on the medial aspect of, and is entrapped by, the abnormally placed medial head of the gastrocnemius as demonstrated in Fig. 1 (type II).

Should mesodermal remnants of the migrating medial head persist posterior to the popliteal artery, or should the

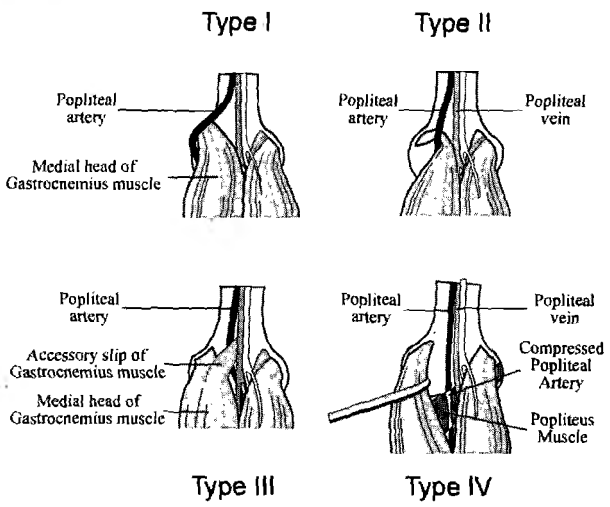


Fig. 1. Classification of types of popliteal artery entrapment syndrome.

artery develop within the migrating muscle mass, a type III popliteal entrapment may result. Here the entrapment mechanism is formed either by fibrous and tendinous bands derived from the remnants of the migrating medial head, or more commonly by an abnormal slip of mature skeletal muscle. These abnormal additional slips of muscle tissue may arise from either the medial or lateral femoral condyles (Fig. 1 (type III)). The definitive popliteal artery may even pass between a double origin of the medial head of the gastrocnemius.

If the axial artery persists as the definitive distal popliteal artery, it will lie in the primitive position, deep to the popliteus muscle or fibrous bands,[40] resulting in a type IV entrapment (Fig. 1 (type IV)).

When any type of entrapment mechanism includes or surrounds the popliteal vein as well as the artery, Rich *et al.*[63,64] have termed this a type V entrapment.[7,36,42,50] Any of the types of entrapment (with the possible exception of type I), may include the tibial nerves resulting in neurological paraesthesiae in addition to claudication as the presenting symptom.[15]

A type of popliteal artery entrapment occurs in the apparent absence of an anatomical abnormality, termed 'functional' entrapment.[27,65,77] The exact nature of the entrapment mechanism remains uncertain, but it has been postulated that a hypertrophic medial head of the gastrocnemius impinges on the medial and posterior aspect of the popliteal artery.[14,65] Up to half of apparently normal asymptomatic individuals may display the phenomenon of reduced or abolished popliteal artery blood flow with extremes of plantarflexion or dorsiflexion against resistance.[2,14,65,77] Such compression of the popliteal artery in the absence of symptoms should not be regarded as pathological, but when classical symptoms are associated with the clinical demonstration of functional popliteal vascular entrapment syndrome, the condition probably requires treatment. It has been proposed that this 'functional' type of popliteal entrapment be termed type VI.[51] Occasionally popliteal artery entrapment syndrome may be acquired following vascular surgical reconstruction for femoropopliteal arterial disease.[6,11]

Clinical picture

The clinical diagnosis of popliteal artery entrapment relies on recognition of the clinical picture of calf or foot claudication occurring with exercise in the young and often athletic individual, sometimes accompanied by paraesthesiae of the foot.[2,5,12,15,18,63] The syndrome was previously thought to be more common in males. Often the initial symptoms are precipitated by an episode of intense physical activity of the lower limbs, e.g. running a marathon. Symptoms may include cramping in the calf and foot, coldness, blanching, paraesthesiae and numbness. Some patients may present with an aneurysm of the popliteal artery. Any popliteal artery aneurysm in a young patient without a history of risk factors should suggest the presence of popliteal vascular entrapment syndrome.

The ankle pulses are normal at rest if occlusion has not taken place. Untreated, the compression mechanism frequently results in deterioration of the popliteal artery with the passage of time resulting in eventual occlusion[13,23] and the absence of normal ankle pulses. Sudden onset of severe disabling claudication and absent ankle pulses, usually in the absence of risk factors predisposing the individual to atheroma, characterise those patients in whom occlusion of the popliteal artery has taken place due to popliteal entrapment. These patients may present with rest pain or ischaemic ulcers, although the development of critical ischaemia with occlusion of the popliteal artery is rare.[51] Distal emboli[19,30,37,68] may result consequent on focal thrombus formation[2,33] at the site of entrapment or from popliteal aneurysm formation[6,19,39,51,64] secondary to the entrapment.

In patients who present with classical symptoms as described above, the presence of normal pulses at rest which diminish or disappear with forced plantarflexion or dorsiflexion, is diagnostic. Absent pulses in a young athletic individual who presents with claudication should always be considered to be due to popliteal vascular entrapment syndrome unless other pathology is demonstrated.

Diagnostic modalities

The diagnosis of popliteal artery entrapment syndrome may be confirmed by Doppler ankle pressures,[8,22,49,58,64] pulse volume recordings,[19] duplex Doppler,[24,22,23,54] computerised axial scanning,[59,66,79] magnetic resonance (MR) imaging,[14,32,57,77] and MR angiography. All of these modalities rely on the demonstration of popliteal artery compression with reduced or abolished popliteal artery blood flow occurring with forced active plantarflexion or dosiflexion of the foot against resistance. However, the most widely used diagnostic modality continues to be contrast angiography (Fig. 2a-d), particularly in order to plan surgery when degeneration, aneurysm or occlusion of the popliteal artery is suspected.[31,38,61] The clinical evaluation, non-invasive tests, and angiography all require forced active plantarflexion or dorsiflexion of the foot against resistance, with the knee fully extended, in order to demonstrate the abnormality if the artery has not yet undergone degenerative changes.[27,77]

Patients and methods

This study presents 51 patients (93 limbs) treated for popliteal artery entrapment syndrome from January 1988 to December 1998. All these patients presented with claudication-like symptoms of the legs causing severe and debilitating symptoms. All patients were subjected to clinical evaluation followed by Doppler ankle/brachial pressure index measurement and duplex Doppler of the popliteal artery at rest. Patients with normal distal pulses were screened using both popliteal artery duplex Doppler and ankle Doppler recording during active plantar- and dorsiflexion against resistance. Where these tests were found to be positive, with reduced or abolished popliteal or distal flow with this manoeuvre, and in all patients with absent ankle pulses or abnormal Doppler findings at rest, the patients were subjected to conventional contrast arteriography, both in the resting and in the forced plantarflexion and dorsiflexion positions, for confirmation of the diagnosis.

Only patients with unequivocal evidence of popliteal artery entrapment, either on angiography or at operation, were included in this study. Patients were not included if their symptoms were not typical of popliteal artery entrapment syndrome, and no patient was included in the study on the basis of positive non-invasive tests alone in the absence of severe symptoms that interfered with normal sporting activities.

Results

Ninety-three instances of popliteal vascular entrapment in 51 individual patients were included in this study over an 11-year period dating from January 1988 to December 1998. The type of entrapment, presenting features and treatment are summarised in Tables I - IV.

In 42 patients the symptoms and entrapment were present bilaterally, and in 9 patients the condition was either totally asymptomatic or not present in the contralateral leg. The mean age of all the patients was 34.9 (range 16 - 55) years. There were 58 affected limbs in male patients (mean age 36.4, range 16 - 55 years), and 35 affected limbs in females (mean age 32.0, range 16 - 52 years).

In 75 limbs angiographical examination confirmed the presence of popliteal artery entrapment syndrome and demonstrated a healthy and patent popliteal artery. In all 75 limbs, the entrapment mechanism was released at operation, usually by myotomy of the medial head of the gastrocnemius muscle or abnormal muscle slips or tendinous bands responsible for the entrapment mechanism, and a healthy popliteal artery was confirmed on examination at surgery. All patients treated in this manner have retained healthy and patent popliteal arteries on follow-up (median follow-up 4.7 years, range 1 - 11 years). Almost without exception, those patients who had previously been compelled to stop their sporting activities because of the symptoms of popliteal artery entrapment, were able to resume normal sporting activities following their postoperative recovery. In 2 patients angiography suggested moderate 'functional' popliteal artery entrapment syndrome bilaterally with a long, diffuse narrowing of the popliteal artery on plantarflexion, but an otherwise angiograhically normal artery at rest. Both patients experienced resolution of their symptoms when they elected to discontinue their extreme physical activity, and they remain well and asymptomatic with normal popliteal arteries on duplex Doppler scan after 2 and 3 years' follow-up respectively.

Eighteen limbs demonstrated occlusion of the popliteal artery or distal embolisation due to aneurysmal change at the entrapment site (13 in males, mean age 33.9 years, SD 11.6 years; and 5 in females, mean age 35.4 years, SD 14.5

TABLE I. Presenting features of 93 limbs with popliteal artery entrapment syndrome

Type	I	II	III	IV	Functional or type VI	Total
Total in series	5	12	36	8	32	93
Occlusion with severe ischaemia	4	5	3	3	3	18
Entrapment causing typical claudication symptoms	1	7	33	5	29	75
Venous entrapment	1	3	6	0	0	10

TABLE II. Analysis of treatment of different types of popliteal entrapment syndrome

Type	I	II	III	IV	Functional or type VI	Total
Total	5	12	36	8	32	93
Occlusion	4	5	3	3	3	18
Myotomy and vein graft	3	4	3	3	3	16
Myotomy only	1	7	33	5	25	71
No operation	1	1	0	0	4	6

TABLE III. Demographics of 51 patients (93 limbs) presenting with popliteal artery entrapment syndrome

95 limbs in 51 patients	9 unilateral	42 bilateral
Mean age of 51 patients 34.89 yrs (SD 11.62)		
	Males	**Females**
Total limbs	58	35
Age (yrs)	36.46	32.03
Standard deviation (yrs)	11.32	11.46
Range (yrs)	16 - 55	16 - 52

years). Fifteen limbs were treated by replacement of the occluded segment of the popliteal artery with reversed saphenous vein grafts. Eight instances of aneurysms change were noted in this group of patients, all associated with type I - IV entrapments. All 15 remain well and patent on follow-up (median follow-up 4.8 years, range 1 - 11 years), with 14 of the 15 returning to normal sporting activities. One patient required amputation for advanced ischaemic change at the time of original presentation, and 1 patient was treated conservatively because of extensive distal thrombosis of the infragenicular vessels. Both of these patients were found to have hypercoaguable states. No other patients in this series who underwent occlusion developed critical ischaemia.

Discussion

With a greater awareness of popliteal artery entrapment syndrome we are observing an increase in the frequency of this diagnosis in young adults presenting to the sports medicine specialist.[9,10,25,46,55] Better evaluation of the problem of the athlete with calf pain[15,48,53,76] by sports medicine specialists, and improved investigation and screening of these cases has improved the diagnostic yield in the young patient with unexplained calf pain. More than half the patients under the age of 50 years presenting with claudication symptoms of the lower limbs in this and other series were subsequently demonstrated to have popliteal artery entrapment syndrome as a cause of their symptoms.[42,51]

Most early reports of popliteal artery entrapment syndrome described patients who had progressed to total occlusion of the artery. The natural history of the popliteal artery with unrelieved compression was documented to be an aggressive one, and on this basis surgery was advised in all patients with a confirmed diagnosis.[31] The description of progressive fibrosis of the entrapped vessel wall leading to aneurysm formation and thrombosis[1,15,19,24,64] supports this recommendation. As the pathology progresses with time, progressive fibrosis and destruction of the arterial wall occurs,[51] first in the arterial adventitia (stage I), then in the media (stage II), and finally in the intima (stage III). The implication is that the degree of arterial degeneration observed when thrombosis has occurred is so severe that the arterial wall cannot be salvaged. This explains the poor medium-term patency results obtained after popliteal artery occlusion treated with a lesser procedure such as thrombolysis, angioplasty or thrombectomy with patching. On the other hand, excellent long-term patency is reported after aneurysm repair or occlusion treated by saphenous vein graft. This argues strongly in favour of complete replacement of the popliteal artery, preferably by saphenous vein, when significant degeneration of the artery or aneurysm formation is noted either on pre-operative angiography, or at the time of operation.

Although the data in this study demonstrate no significant difference between the age of those patients presenting with popliteal artery occlusion and those in whom a myotomy only was required, the youngest patients in the series were in most cases athletes with type I or II entrapments, or with tight localised tendinous bands of the type III and IV entrapments who had undergone popliteal artery occlusion. The patients presenting with occlusion at an older age invariably had muscular entrapment mechanisms of type III or type VI. This finding suggests that the rate of arterial wall degeneration in popliteal artery entrapment syndrome may depend on the degree of compression and the magnitude of the forces exerted on the popliteal artery by the compression mechanism.

TABLE IV. Limbs with severe ischaemic symptoms due to popliteal artery occlusion caused by popliteal artery entrapment syndrome

Presenting with severe ischaemia	Male	Female
Number of limbs	13	5
Age of patients (yrs)	33.87	35.40
Standard deviation (yrs)	11.60	14.49

Most cases of type I and II entrapment are easy to diagnose on angiography and other imaging modalities. In addition, the more localised types of entrapment seen with type III and IV, are in our experience frequently distinguishable from the more diffuse narrowing of the artery found at angiography with the 'functional' or type VI entrapment. On the basis of these observations, it is strongly advised that surgical correction be offered in all cases of type I - IV at the time of diagnosis,[31] without waiting until arterial degeneration has resulted.

The demonstration that the popliteal artery will undergo some transitory compression or even temporary occlusion with extremes of plantarflexion or dorsiflexion in up to half of the normal population, cannot be ignored. The simple demonstration of popliteal artery compression in such stress positions cannot justify operation in patients with otherwise normal anatomy and minor or no symptoms.[14,27,78] On the other hand, we have in the present series documented three popliteal arteries that have undergone occlusion in two patients with a 'functional' or type VI symptomatic entrap-

ment, and apparently normal anatomy. The demonstration that a functional popliteal artery entrapment syndrome may progress to occlusion with the histological picture of chronic compression,[51] with the degeneration not due to antheroma, justifies a more aggressive surgical approach to symptomatic patients who are demonstrated to have a functional or type VI entrapment. Until further research elucidates the clinical significance and natural history of degeneration of the popliteal artery in the functional type of entrapment in both the asymptomatic and symptomatic patient, the correct management of this condition must remain controversial.

The various manifestations and types of popliteal artery entrapment syndrome are much more prevalent than originally appreciated. This diagnosis should be considered in any patient under the age of 50 years presenting with typical calf and foot claudication symptoms on exercise, particularly if the symptoms occur in an athletic individual and particularly if the normal risk factors for atheroma are absent. The finding of an isolated popliteal artery aneurysm or isolated popliteal artery occlusion in the young physically active individual without evidence of systemic disease should be considered to be due to popliteal artery entrapment syndrome unless proven otherwise. The evidence suggests that all patients in whom the type I - IV entrapment is diagnosed before occlusion of the artery should receive surgical release of the entrapment mechanism prior to deterioration of the popliteal artery by repetitive compression. On the other hand, only patients with a significant and typical history of

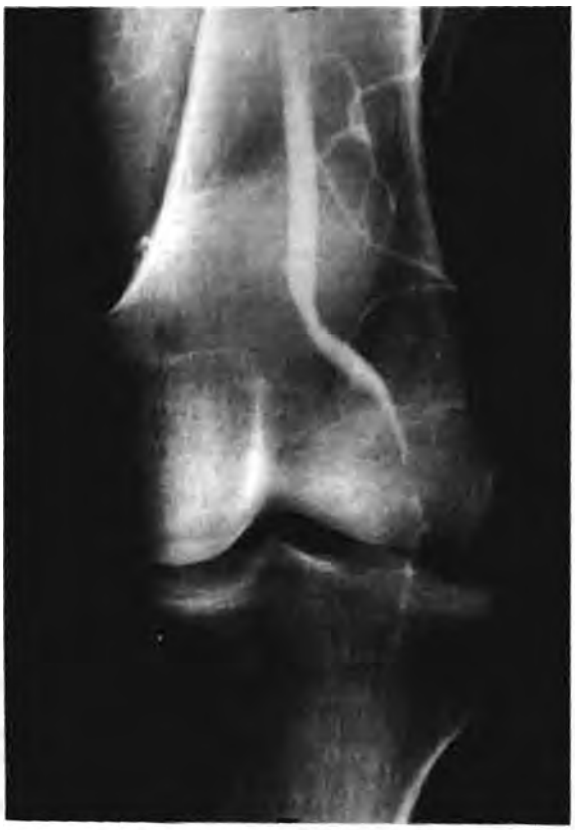
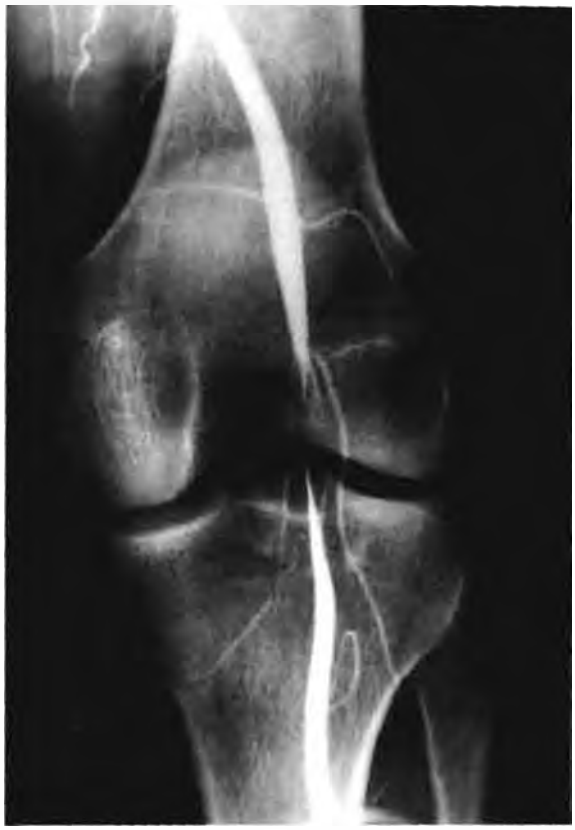
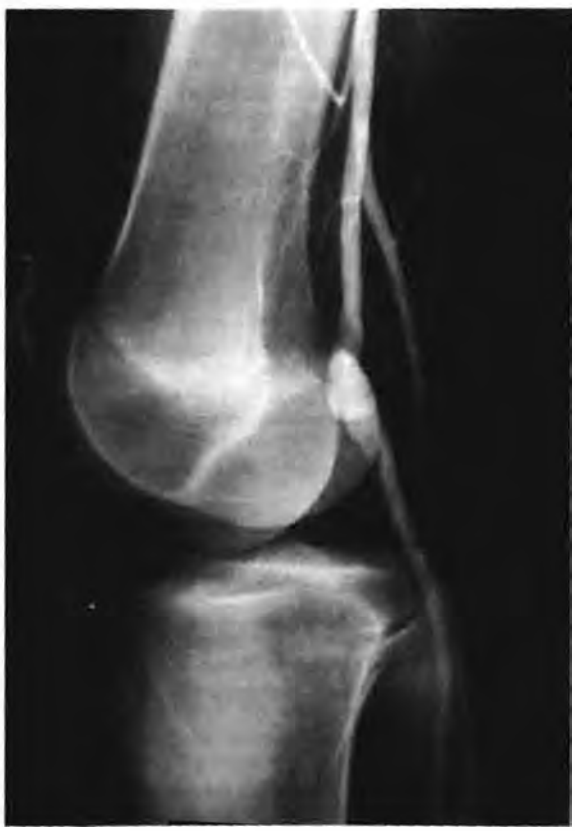


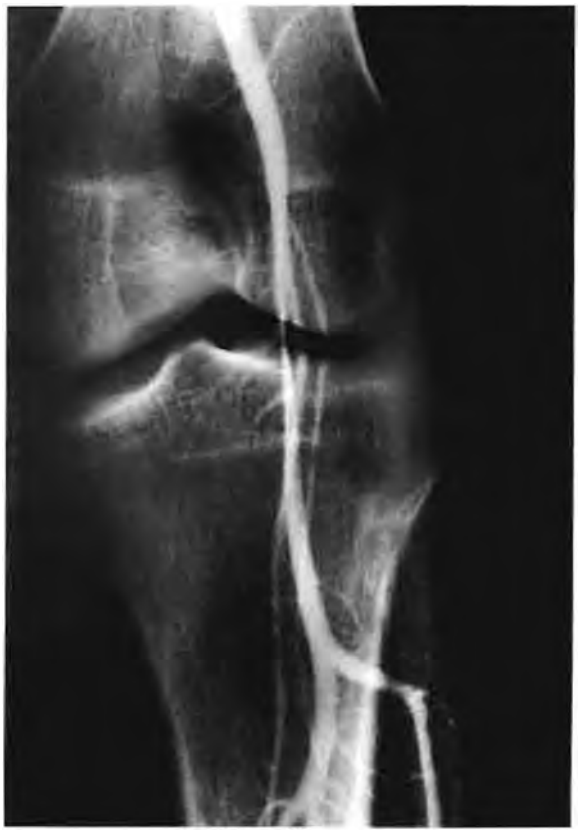
Fig. 2. Some examples of angiography of the popliteal artery entrapment syndrome. 2a. A medial deviation of the artery at rest due to a type III additional muscular head causing the entrapment.



2b. A localised entrapment of the artery with plantarflexion due to a localised muscular band causing a type III entrapment.



2c. Localised popliteal artery aneurysm formation due to popliteal artery entrapment syndrome.



2d. The longer smooth entrapment often seen with the 'functional' or type VI entrapment.

symptoms should be offered surgical treatment for the functional or type VI popliteal artery entrapment. Once the popliteal artery has undergone occlusion, the evidence suggests that the artery is beyond repair, and it is recommended that the artery be replaced, preferably by saphenous vein graft, to ensure optimum long-term popliteal artery patency in these often young and physically active individuals.

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ORIGINAL RESEARCH ARTICLE

Creatine supplementation and exercise performance in rugby players

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Abstract

Objective. To determine whether creatine supplementation improves exercise performance in rugby players.

Setting. Buffalo Park, East London.

Subjects. Twenty-five club rugby players, after completion of pre-season training.

Design. Field study. Seventeen rugby players volunteered for the creatine group and 8 rugby players volunteered for the control group. The creatine group ingested a creatine monohydrate supplement. The players ingested 20 g of creatine per day for 5 days as a loading dose, followed by a maintenance dose of 5 g of creatine per day for 79 days. The control group ingested no supplements. Subjects had baseline measurements taken before starting creatine supplementation. Players' body mass in kg, 50 m isolated sprint time, and the number of sit-ups and push-ups each in 30 seconds, were measured. The final measurements were taken 84 days after starting creatine supplementation. Only players who completed the study were analysed.

Results. Fourteen rugby players from the creatine group and 7 rugby players from the control group completed the study. The age of the players in the creatine and control groups was 25.2 ± 3.9 versus 21.3 ± 3.3 years respectively. Body mass did not change significantly in either group. The body mass changed from 88.8 ± 16.0 to 86.6 ± 14.0 kg in the creatine group and from 87.8 ± 9.9 kg to 87.0 ± 9.7 kg in the control group. Isolated sprint performance improved significantly in the creatine group from 8.3 ± 0.6 s to 7.9 ± 0.5 s ($P < 0.05$) Sprint performance did not change significantly in the control group: 8.4 ± 0.4 s to 8.5 ± 1.1 s. The number of push-ups and sit-ups in 30 s increased significantly in the creatine group from 34.7 ± 8.6 to 45.2 ± 6.3 ($P < 0.05$). and from 30.0 ± 5.9 to 35.0 ± 4.7 ($P < 0.05$) respectively. In the control group, the number of push-ups did not change significantly: 33.1 ± 9.9 to 33.4 ± 8.5. The number of sit-ups increased from 27.3 ± 3.2 to 29.2 ± 2.5. Forty-two per cent of the players in the creatine group experienced side-effects when ingesting creatine, compared with the control group which had no side-effects.

Conclusion. After 84 days of creatine supplementation, body mass did not change significantly, but isolated sprint performance and the number of push-ups and sit-ups performed by the rugby players in the creatine group increased significantly. A large proportion of the rugby players experienced side-effects when ingesting creatine monohydrate.

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Introduction

Creatine supplements are being ingested by athletes at all levels of sporting competition. The use of creatine is further popularised when anecdotal information of Olympic athletes reportedly using creatine as a supplement are reported in the press.[2] Athletes perceive that creatine may enhance their specific sports performance.

It is estimated that adenosine triphosphate (ATP) and phosphocreatine (Pcr) can sustain very high-intensity exercise for approximately 10 seconds.[3] Theoretically, creatine supplementation could increase intramuscular Pcr concentration and subsequent ATP formation, prolonging the duration of high-intensity physical activity and power output.[8,18] Overall, creatine supplementation could be of benefit to the athlete.

Of the many studies published on creatine, many, but far from all, show an improvement in performance.[9,11] The most convincing evidence for an ergogenic effect is seen in activities requiring isotonic strength and those that involve repetitive bouts of high-intensity exercise interspersed with short rest periods.[34] Most of these studies were performed under laboratory conditions. There are few studies on the effects of creatine supplementation on performance in the field and during competitive events.

One of the purported effects of creatine supplementation is an increase in body mass, particularly muscle mass.[31-33] This may occur in a number of ways. Creatine, being osmotically active, could cause an intracellular fluid shift, thereby

increasing intracellular water and body mass. It has been suggested that increased cellular hydration and/or increased Pcr may also stimulate protein synthesis and decrease protein degradation.[22,31-33] This effect, however, may not be directly due to increased intracellar PCr or hydration, but due to some other factor that creatine is affecting. Of the approximately 20 studies on the effect of long-term creatine supplementation on body mass, about 80% show body mass gains.[34] The best gains were seen in those athletes undergoing resistance training.[34]

According to available literature, isolated sprint running performance in athletes after creatine supplementation is either improved[26,30] or unaffected.[23,25,27,28] Therefore, the ability of creatine to improve isolated sprint running performance remains controversial.

Creatine supplementation may improve high-intensity, short-duration (≤ 30 s) exercise tasks, as demonstrated in another field study[6] where a continuous jump test for 45 s showed that subjects supplementing with creatine showed an increase in work output during the first 30 s of the task.

Accordingly, the aim of this study was to determine, in a field design, whether creatine supplementation over 84 days, increased body mass, improved isolated 50 m sprint performance and improved short-duration high-intensity exercise, as measured by the number of push-ups and sit-ups performed in 30 s each.

Methods

Research methods

Twenty-five rugby players volunteered for the study. Seventeen rugby players volunteered for exercise testing with creatine supplementation and formed the creatine group. These players were given 500 g of creatine monohydrate of the same brand. Eight players volunteered for exercise testing only and formed the control group. All players gave informed verbal consent to participate in the study and agreed to adhere to the conditions thereof. None of the players in the creatine group had ingested a creatine monohydrate supplement in the previous 3 months. This would be sufficient time for creatine levels to return to baseline should any player have previously ingested creatine monohydrate.[17,21] None of the players was vegetarian. This is relevant as vegetarians have virtually no dietary intake of creatine and rely on creatine synthesis in the liver, pancreas and kidneys.[11] Vegetarians have been shown to have lower plasma creatine levels.[10] This does not necessarily mean decreased tissue creatine content.[19] Theoretically, muscle Pcr levels could be lower in vegetarians. It has been shown that individuals with a lower muscle Pcr can have a greater increase in muscle Pcr with creatine supplementation,[14,19] raising the question of greater performance benefit.[12]

All 25 rugby players had completed the same organised pre-season training and were match fit and ready to play their first match. The training schedule for the duration of the study (and for the season) for all the players was as follows: practice sessions took place on Tuesdays and Thursdays, circuit training on Mondays in the gym, and matches on Saturdays. Exercise at practices involved running and skills training, and in the gym exercise involved using light weights for 15 - 20 repetitions.

Baseline testing was performed on all players directly before creatine supplementation. Exercise testing was conducted again after 84 days of creatine supplementation. Parameters to be tested were: body mass in kg, 50 m sprint time, the number of push-ups in 30s and the number of sit-ups in 30 s (as a measure of short-duration high-intensity exercise). Body fat percentage was not measured.

Environmental testing conditions were consistent. Data collection took place in the early evening before rugby practice. Dry, windless conditions prevailed and the same strip of dry grass was used on each occasion. The players were tested barefoot in order to standardise footwear. When performing push-ups, the tester placed a fist on the ground to ensure a full range of movement. Touching the tester's fist with the chest yielded a count. The player's feet were anchored during the sit-up test and a full range of movement was achieved by the player's back touching the tester's fist on the ground behind him. Three minutes elapsed between the push-up and the sit-up test. Creatine and control subjects were tested in the evening on the same day.

The creatine used in the study was creatine monohydrate with a percentage purity of 99.6% gravimetric. The dosing regimen for the creatine subjects consisted of a loading dose of 20 g/day divided into four daily doses and consumed over 5 days. This was followed by a maintenance dose of 5 g/day for the 79 days. Research has shown that creatine uptake into skeletal muscle is enhanced during creatine supplementation if the creatine is consumed together with a carbohydrate.[16,20] Players were instructed to consume the prescribed creatine amount mixed with 250 ml of grape juice, which amounts to 30 g of carbohydrate, to optimise creatine uptake into skeletal muscle.[3,13,16,20] Players were given 500 g of creatine for the study period accompanied by written instructions concerning dose and frequency of creatine ingestion. Creatine consumption started immediately after baseline testing and continued for an 84-day period. Players in the creatine group consumed no other nutritional supplements.

At the end of the study period of 84 days, 14 rugby players in the creatine group and 7 rugby players in the placebo group had all their measurements taken and successfully completed the study. Two players in the creatine group withdrew because of work commitments, while 1 player from each group withdrew because of injury. The 14 rugby players in the creatine group answered a questionnaire that evaluated the subjective effects of creatine supplementation.

Statistical methods

Only the data for those players who completed the study were analysed. The analysis of each group was done as follows: measurements recorded before starting creatine supplementation were considered as 'pre' creatine effect, and the measurements recorded after 84 days of creatine supplementation as 'post' creatine effect. This design allowed for the use of the dependent *t*-test or its non-parametric equivalent, the Wilcoxon matched pairs test, to analyse the relationship between the pre- and post-creatine results in each group separately. As none of the variables or the dif-

ference of the relevant variables were normally distributed, the Wilcoxon matched pairs procedure was used. Due to the non-normality of the data, the Mann-Whitney *U*-test was used to examine any relationship and compare the creatine and control groups. Results are expressed as the mean ± standard deviation (SD). The statistical significance was accepted when $P < 0.05$.

Results

Body mass and physical performance

In the creatine group, the average body mass decreased from 88.8 ± 16.0 kg to 86.6 ± 14.1 kg and was not significant. The 50 m isolated sprint speed decreased from 8.3 ± 0.6 s to 7.9 ± 0.5 s ($P < 0.05$). The number of push-ups per 30 s increased from 34.7 ± 8.6 to 45.2 ± 6.3 ($P < 0.05$) and the number of sit-ups increased from 30.0 ± 5.9 to 35.0 ± 4.7 ($P < 0.05$) (Table I).

Comparison of the creatine and control groups

The 14 rugby players in the creatine group were significantly older than the 7 players in the control group (25.2 ± 3.9 v. 21.3 ± 3.3 years, $P < 0.05$). The players in the creatine group completed more push-ups per 30 s at the final test compared with the players in the control group (45.2 ± 6.3 v. 33.4 ± 8.5, $P < 0.05$). The players in the creatine group completed more sit-ups per 30 s at the final test compared with the players in the control group (35.0 ± 4.7 v. 29.2 ± 2.5, $P < 0.05$). There were no other significant differences between the creatine and control groups.

Results of the questionnaire

Of the 14 rugby players supplementing with creatine, 42% reported side-effects while ingesting creatine (Table II). There were no reported symptoms in the control group. These reported side-effects involved the gastro-intestinal tract (4 players), the musculoskeletal system (1 player) and the cardiovascular system (1 player). These side-effects occurred in different players. The reported gastro-intestinal side-effects were experienced during the 5-day loading phase of creatine supplementation. The side-effects were diarrhoea, with 1 player reporting colic. One player reported cramping of the muscles in his thighs. One player described increased thirst, a dry mouth and having to pass urine less frequently, indicating possible dehydration. This effect was reported sporadically during the creatine supplementation period.

All 14 rugby players reported that they felt they had an increase in strength, stamina, recovery time and performance, with about half of the subjects experiencing these effects as early as 14 days after starting creatine supplementation.

All 14 rugby players considered creatine to be a safe product and would purchase it as an ergogenic aid. The players who decided not to supplement with creatine did so because they believed that it would not improve their performance.

Discussion

Isolated sprint performance over 50 m showed a significant improvement in the creatine group of rugby players. The increased Pcr stores in the creatine group means that there would be more PCr to break down before fatigue starts to set in.[8,15] More ATP could be available to enhance muscle contraction, and possibly account for the improvement in isolated sprint performance in the creatine group. These findings are consistent with studies in the literature that used similar sprint distances to test the athletes.[26,30] The improvements in performance were significant in these studies and ranged from 1 to 2%. Sprint distances in the studies that did not show an improvement in isolated sprint performance

TABLE I. Comparison of the pre- and post-test values (mean ± SD) in the creatine and control groups

	Creatine ($N = 14$)		Control ($N = 7$)	
Measurements	Pre	Post	Pre	Post
Body mass (kg)	88.8 ± 16	86.6 ± 14.1	87.8 ± 9.9	87.0 ± 9.7
50 m sprint(s)	8.3 ± 0.6	7.9 ± 0.5*	8.4 ± 0.4	8.5 ± 1.1
Push-ups in 30s	34.7 ± 8.6	45.2 ± 6.3*	33.1 ± 9.9	33.4 ± 8.5
Sit-ups in 30s	30.0 ± 5.9	35.0 ± 4.7*	27.3 ± 3.2	29.2 ± 2.5

TABLE II. Side-effects among rugby players ($N = 14$) ingesting creatine

System	Side-effects	Number	Percentage
Gastro-intestinal	Diarrhoea	3	21
	Colic	1	7
Musculoskeletal	Cramping	1	7
Cardiovascular	Dehydration	1	7
		6	42

were longer and ranged from 60 m to 150 m.[28,30]

The major finding in this study was a significant improvement in the number of sit-ups and push-ups among players in the creatine group compared with the control group. Supplementing with creatine assumedly increased muscle Pcr stores in most subjects in the creatine group.[8,15] In this study, players in the creatine group were able to increase their power output during the 30 s duration of the push-up and sit-up test and delay the onset of fatigue. However, the study was non-blinded, and the possibility of the placebo effect does exist in order to account for these significant findings.

Most studies show that total body mass increases with creatine supplementation.[4,5,7,13,24] This occurs within 5 days during the loading phase and continues during the maintenance phase.[24] Initially this is due to water retention, and later, with continued creatine supplementation, possibly to increased myofibrillar protein synthesis.[21] This study revealed that the body mass of players did not change significantly with creatine supplementation. This is not consistent with the data from the questionnaire, which revealed that 65% of players supplementing with creatine felt that their body mass had increased and that there was a change in their physical profile. There may have been a change in their ratio of fat-free mass to body mass. Dietary factors and calorie intake could also have affected the body mass measurement. One of the 'expected' outcomes of this study was that creatine supplementation would increase body mass. However, this did not happen and the reasons for this ergogenic effect without an increase in body mass need to be examined in further studies.

The only documented side-effect from clinical research studies is that of weight gain.[31-33] Undocumented side-effects of creatine supplementation have appeared in lay publications and in the media.[1] These include gastro-intestinal distress, muscle cramping/muscle injury and dehydration.

The speculated mechanism of stomach upset is that the maximum absorption rate of creatine in the intestine may be exceeded. Creatine in the intestine draws water into the intestine and could cause loose stools and diarrhoea.[34] One could hypothesise that supplementing with large doses of creatine (> 35 g/day) may cause gastro-intestinal symptoms. In this study, it was interesting that players in the creatine group who developed stomach upset did so during the 5-day loading phase.

The most commonly reported anecdotal side-effect is that of muscle cramping.[1,29] Only 1 player out of 14 reported this effect in this study. It is thought that this muscle dysfunction may be related to electrolyte imbalances in the muscle cell.[29] These anecdotal side-effects have been refuted in certain scientific studies.[24]

Despite the reports on side-effects, all subjects supplementing with creatine felt that creatine was a safe product and would purchase creatine as an ergogenic aid.

In summary, this study showed that creatine supplementation significantly improved isolated sprint performance and the number of sit-ups and push-ups rugby players could perform. Contrary to results from other studies, body mass did not change significantly. A large proportion of the players reported experiencing side-effects when ingesting creatine. However, the rugby players did not interpret this as a cause for concern and would purchase creatine monohydrate as an ergogenic aid.

Thanks to Mr J Baxter, Department of Statistics, Rhodes University, Grahamstown, for statistical analysis of the data.

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Drug-free sport

To the Editor: The South African Institute for Drug-Free Sport has noted with surprise and concern the conclusions of the research article in the November edition of the *South African Journal of Sports Medicine* entitled 'Substance abuse and knowledge thereof among elite South African athletes'.[1]

The use of drugs to enhance athletic performance is against the rules laid down by the governing bodies of most recognised sports worldwide, and elite athletes, particularly those competing at international level, have an obligation to familiarise themselves and comply with the policies and regulations of their governing bodies regarding drugs and sport.

Relevant information on prohibited and permitted drugs in sport is available from a variety of sources. Most sports federations are updated annually with the IOC list of permitted and banned substances in sport, NOCSA produces an excellent booklet which it distributes free of charge comprising an alphabetical and therapeutical list of prohibited, restricted and permitted drugs in sport, and information can be accessed from the Internet, the SA Institute for Drug-Free Sport Hotline (021 - 448 3888, 9.00 a.m. - 1.00 p.m. weekdays) and the Institute's website, **www.drugfreesport.org.za**

Ignorance of the issues surrounding doping can also no longer be used as an excuse by doctors and pharmacists. It is important that they equip themselves with salient information in order to avoid the possibility of recommending or prescribing the use of prohibited substances to patients involved in competitive sport.

Doping substances and methods are prohibited in sport for various reasons, most notably: *(i)* their performance-enhancing effects which contravene the ethics of sport and undermine the principles of fair participation; *(ii)* the harm which they may cause to a competitor's health; and *(iii)* the legal implications of using certain substances, such as anabolic androgenic steroids, a Schedule V drug.

The Institute conducts a comprehensive national drug testing programme in accordance with International Standards for Doping Control (last year 1 600 sportsmen and women from 41 sporting disciplines were tested both in and out of competition, and this will be increased to 1 700 tests across 49 sporting disciplines during the current year). The Institute has over 50 independent Doping Control Officers nationally, who undergo stringent annual training and refresher courses, and in line with international practice, out of competition testing will be increased substantially in future.

Education and the provision of information are also key elements in any national anti-doping strategy. Athletes and coaches need to be informed of their obligations under the drug-testing policy of their sport. School programmes are needed to raise awareness of the issues surrounding doping and drugs in sport and to influence attitudes towards healthier behaviour. The medical and pharmaceutical professions must be kept informed and updated on prohibited and permitted substances and the risks associated with the use of those substances both on and off the playing field.

The Institute tries to reach all these target markets through a variety of education and awareness campaigns, seminars, lectures, workshops and the distribution of promotional and educational material. This year the education programme has been extended to target schoolchildren, as research has produced some disturbing statistics regarding steroid abuse.

South Africa enjoys considerable status internationally in the field of anti-doping, and is at the forefront of the latest anti- doping strategies and testing methods. The Institute is among a handful of international anti-doping agencies preparing for ISO 9002 accreditation this year, and South Africa has one of only 27 IOC-accredited laboratories worldwide. The Institute is represented at meetings of the Monitoring Group on Anti Doping at the Council of Europe; Minister N Balfour, the Minister of Sport, serves on the Executive Board of the World Anti Doping Agency (WADA); and the Chairman of the Board of SAIDS, Dr Ismail Jakoet, was selected as a WADA anti-doping monitor at the Sydney Olympics.

Sport is an important part of the South African way of life, and our sporting achievements are a source of great national pride. Doping violates the integrity of sport, carries serious health risks for individuals, and promotes the notion that dishonesty can be rewarded.

As custodian of South Africa's anti-doping programme, the South African Institute for Drug-Free Sport, created by an Act of Parliament in 1977 as an initiative of Sport and Recreation South Africa, is committed to promoting drug-free sport and ethical sporting practices in this country.

Enquiries: Tel. 021 - 683 7129 / Fax 021 - 683 7274 / Email: **drugfree@iafrica.com**

Daphne Bradbury

General Manager
South African Institute for Drug-Free Sport

1. Coopoo Y, Jakoet J. Substance abuse and knowledge thereof among elite South African athletes. *South African Journal of Sports Medicine* 2000; **7:** 10-13.

Early postural correction

To the Editor: May I draw your attention to the opinion of renowned doctors in the field of sports medicine, namely that the aetiology of the majority of sports injuries concerns widespread postural faults in weight-bearing joints. What is little known, however, is the discovery by Neumann-Neurode at the turn of the last century that babies like to practise effective remedial exercises in adult hands, with better and quicker results than are obtained with older children of any age.

Since postural faults are frequently inherited, it is not surprising that they are usually noticeable in infancy. The medical value of early remedial exercise is backed up by the positive research results of Professor J Trueta[1] at the Nuffield Centre of Orthopaedic Surgery in Oxford. He proved that infantile partially ossified bones respond more strongly to the stimulus of exercise and become thicker, longer and stronger in less time than older ossified bones.

That infants have greater capacity for growth, regeneration and adaptation is already common medical knowledge and points to the advantage and need to recognise and correct postural faults in babyhood (club feet and dislocated hips are well-known examples). Such corrections are one important form of preventing common postural injuries in later years.

At present it is impossible to measure a baby's postural changes accurately enough for research. At the same time the need for timely correction is so great and the technique of baby exercise is well-enough documented that the subject of orthopaedic musculoskeletal correction has now become a postgraduate physiotherapy subject in ongoing university courses. I am not alone in thinking that early postural correction will become generally accepted as one means of preventing common sports injuries.

For more information contact: Agnes Wenham (MCSP), tel: (011) 788-5028; or Colleen Westgate (BSc Physio), tel: (011) 787-7293.

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South African Journal of Sports Medicine

Scope. The *South African Journal of Sports Medicine* is an international, refereed journal published for professionals with a primary interest in sports medicine and exercise science practice. The journal publishes original research and reviews covering diagnostics, therapeutics and rehabilitation in healthy and physically challenged individuals of all ages and levels of sport and exercise participation. Original manuscripts, i.e. those that have not been published elsewhere except in abstract form, will be accepted from all countries and subject to peer review by the Editors and Editorial Board. *The South African Journal of Sports Medicine* invites articles for submission from the areas of: (1) diagnosis, treatment, and rehabilitation of sport- and exercise-related injuries, (2) medical illnesses induced by or exacerbated by exercise, (3) the relationship between exercise and health, including exercise physiology, (4) the medical care of physically active individuals, (5) sports psychology, (6) sports nutrition, and (7) biomechanics related to sport. Articles are invited from within the following categories:

ORIGINAL RESEARCH: Clinical research and basic science articles that are clinically relevant.

BRIEF REPORTS: Clinical studies that are limited in depth or scope but with important findings to report.

CASE REPORTS: Reports of clinical observations that have been carefully documented are particularly instructive.

Additional manuscripts may be submitted, after consulting with the Editor-in-Chief, in the following categories:

LETTERS TO THE EDITOR:

LEAD EDITORIALS: These are short syntheses of data and current thought on topical issues in the field of sports medicine.

REVIEW ARTICLES: These should be concise, in-depth, and well referenced; they should use the principles of critical appraisal (evidence-based medicine).

POSITION STATEMENTS: These succinct but comprehensive documents are typically prepared by a recognised society for the purpose of providing clinical guidelines in important areas of sports medicine.

Form of manuscript. Send manuscripts to Professor Mike Lambert, Sports Science Institute of South Africa, P O Box 115, Newlands, Cape Town, 8000, Tel: (021) 650 4558, Fax (021) 686 7530. Three copies of each manuscript must be submitted, in English in triple-spaced, typewritten form with a 5 cm (2 inch) left margin. Pages should be numbered from the title page. The text of the manuscript should be in the following sequence: Structured abstract (including key words), Introduction, Methods, Results, Discussion, Conclusions, Acknowledgements, References, tables, and figure legends. For clarity, subheadings are recommended wherever appropriate. In the case of research articles, a short section in the Discussion or Conclusion should summarise the clinical relevance of the research. The author should retain a copy for reference, as manuscripts are not routinely returned.

The title page of each manuscript should include only the article title, the author's full names (first name, middle initial, last name), academic degrees and affiliations, the name, address, telephone and E-mail numbers of the person to whom proofs and reprint requests should be addressed, necessary footnotes to these items, and a running title not exceeding 45 letters and spaces. Indicate specific institutional affiliations of each author. Please list degrees or their equivalents. Information concerning sources of financial support should be placed in the Acknowledgement section.

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The format of a case report is different from other submitted manuscripts. The differences are as follows;

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2) The report will be published without an abstract.
3) A maximum of 10 references will be accepted.
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OBJECTIVE. State the main question or objective of the study and the major hypothesis tested, if any.

DESIGN. Describe the design of the study indicating, as appropriate, use of randomisation, blinding, criterion standards for diagnostic tests, temporal direction (retrospective or prospective), and so on.

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INTERVENTIONS. Describe essential features of any interventions, including their method and duration of administration.

MAIN OUTCOME MEASURE(S). The primary study outcome measures should be indicated as planned before data collection began. If the hypothesis being reported was formulated during or after data collection, this fact should be clearly stated.

RESULTS. Describe measurements that are not evident from the nature of the main results and indicate any blinding. If possible, the results should be accompanied by confidence intervals (most often 95% interval) and the exact level of statistical significance. For comparative studies confidence intervals should relate to the differences between groups. Absolute values should be indicated when risk changes or effect sizes are given.

CONCLUSIONS. State only those conclusions of the study that are directly supported by data, along with their clinical application (avoiding over-generalisation) or whether additional study is required before the information should be used in usual clinical settings. Equal emphasis must be given to positive and negative findings of equal scientific merit.

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DATA EXTRACTION. Summarise guidelines used for abstracting data and how they were applied.

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1. Stratford PW, Miserfi D, Ogilvie R, Binkley J, Wuori J. Assessing the responsiveness of Five KT1000 knee arthrometer measures used to evaluate anterior laxity at the knee joint. *Clin J Sport Med* 1991; 1L: 225-8.

Book

2. Antonaccio MJ. *Cardiovascular Pharmacology.* New York: Raven Press, 1990.

Chapter in a book

3. McGinty JB. Ligament, bone, and nerve complications. In: Sprague NF, III, ed. *Complications in Arthroscopy.* New York: Raven Press, 1989: 87-106.

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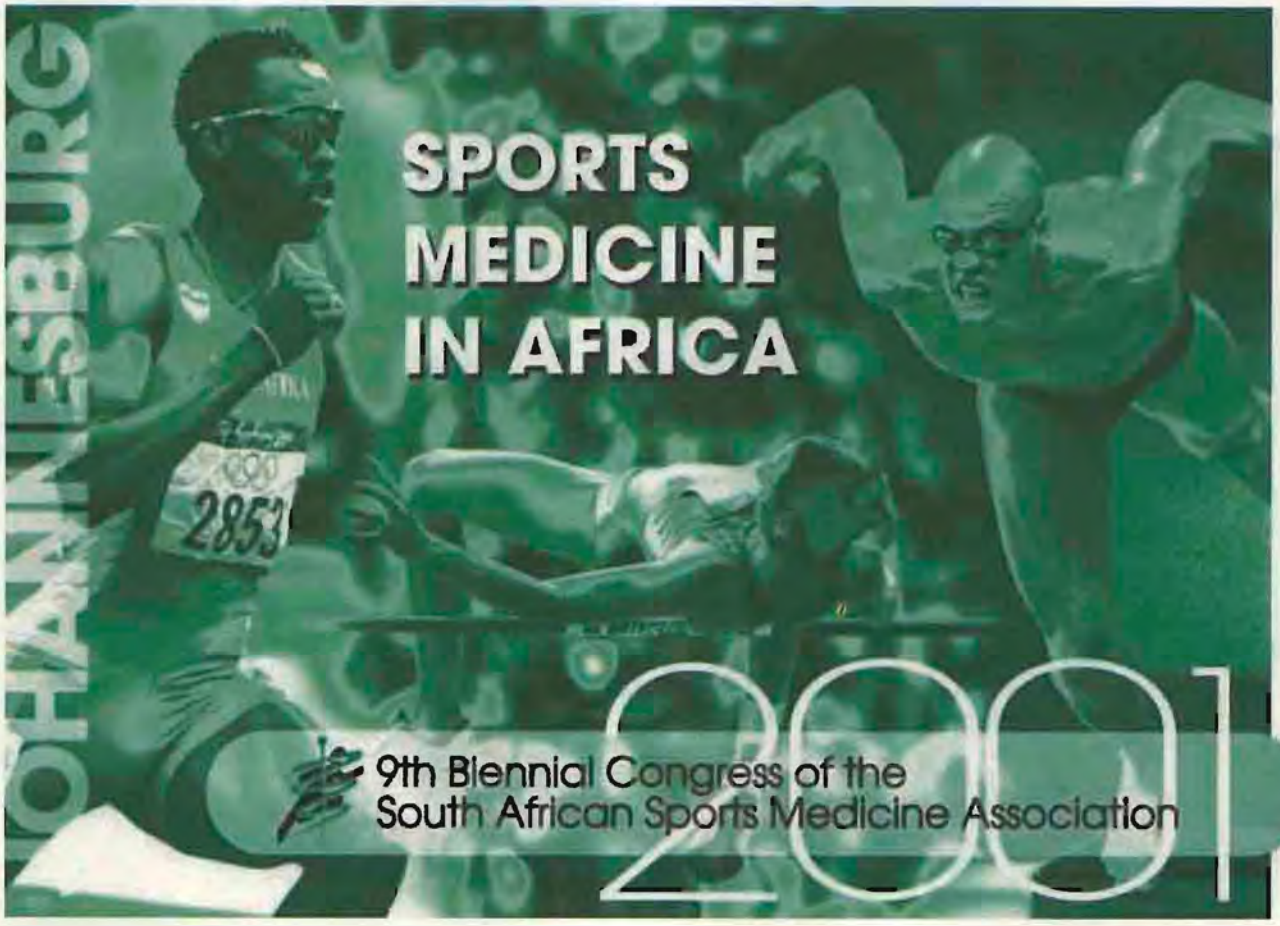

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