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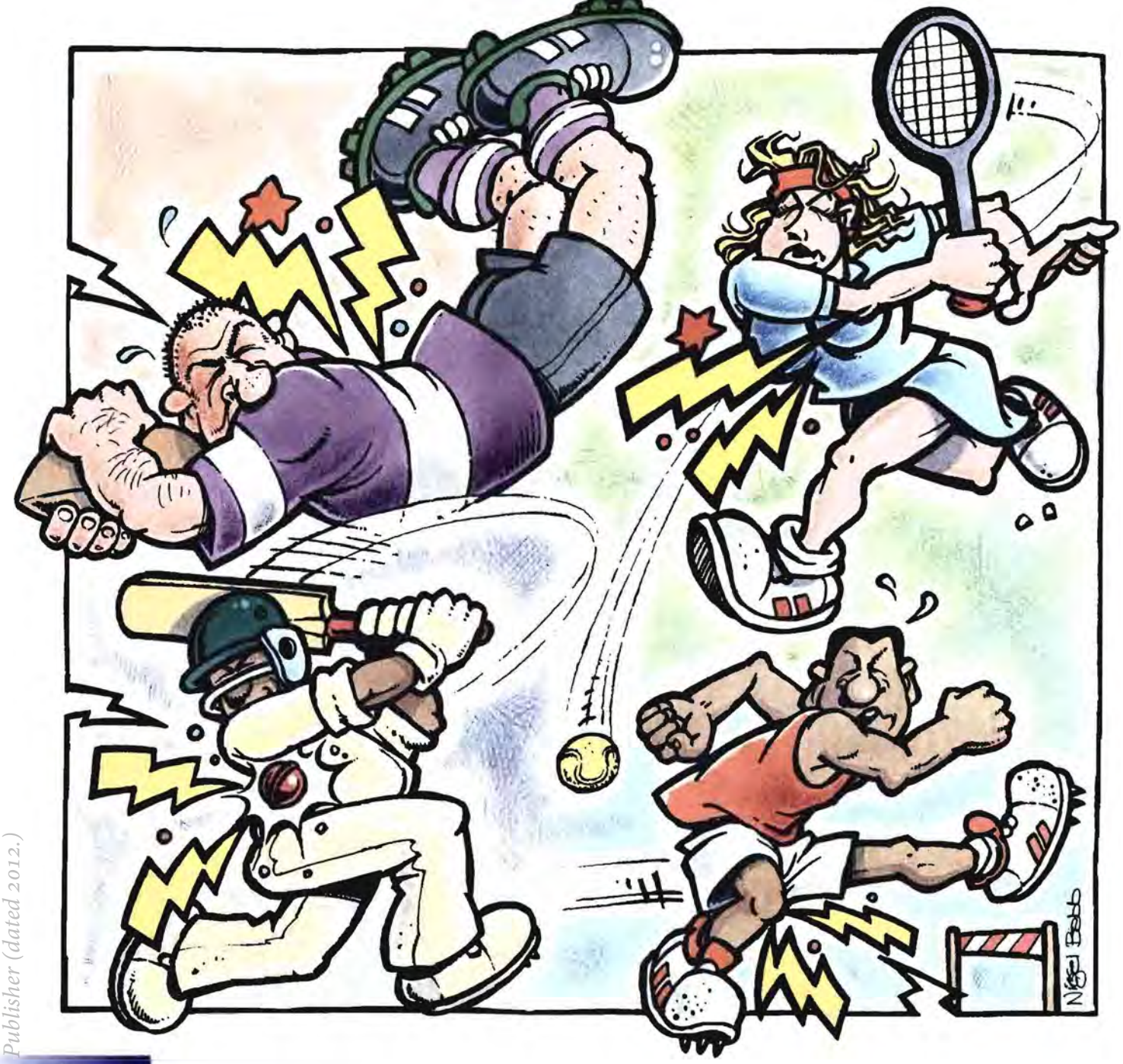

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EDITORIAL

The content of the second issue of the Journal in 1998 is representative of the diversity of scientific interests among members of the South African Sports Medicine Association (SASMA). I believe that the strength of the Association lies in this diversity, and I remain convinced that Sports Medicine is best practiced and researched in a multi-disciplinary setting.

This issue has a number of unique contributions. Firstly, Dr Myburgh contributed the invited Review Article for this Edition. The importance of achieving an optimum peak bone mass must be emphasized. In particular, the positive role of physical activity to achieve peak bone mass must be encouraged in children and adolescents. As pointed out by Dr Myburgh, it is important to remember that the young female athlete with menstrual dysfunction can develop a low bone density, and clinicians must be aware of this potential danger. The Editorial Board of this Journal will continue to publish high quality Invited Review Articles of similar caliber to the one written by Dr Myburgh.

Secondly, the original research articles in this edition are once again of a high standard, and represent different areas of Sport Medicine. Dr Du Toit and his colleagues designed a unique study to examine the force absorption and rebound characteristics of cricket batting pads. Their results are likely to influence the design and choice of pads used by players. Ms Fuller and her colleague investigated the potential use of an infrared auditory canal thermometer to measure the core temperature in athletes - a clinical measurement that is of extreme importance in diagnosing heat illness. They conclude that infrared auditory canal thermometry is too variable and does not provide the clinician with a "quick and easy" measure of core temperature.

Thirdly, we are happy to introduce a new form of presenting research data in the form of a brief report. This format allows researchers to publish data that is of interest, but where the content, depth or limitations preclude it from being published as a full research article. Dr Marino reports on the urinary catecholamine excretion during outdoor sports rock climbing. He concludes that urinary catecholamine excretion does not increase after a strenuous climb of 7-8 minutes.

Finally, we are proud to publish the Position Statement on "Ethics in Sports Medicine", which has been reproduced, with permission, from the International Sports Medicine Federation (FIMS). We will, in future, continue to publish Position Statements, as these are a very useful yardstick by which we can evaluate our approach to problems (clinical and other) in Sports Medicine. I would like to take this opportunity to thank the International Sports Medicine Federation (FIMS) for allowing us to publish their material.

Now it just remains for me to wish you all a happy festive season, a restful holiday, and great sporting and personal success in 1999.

Prof MP Schwellnus, MBBCh, MSc (Med), MD, FACSM
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Exercise and peak bone mass: An update

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INTRODUCTION

Exercise has been widely recommended as a means of preventing osteoporosis. The rationale for this recommendation was based largely on the inference that immobilisation and weightlessness lead to bone loss and therefore exercise should lead to bone gain. Since peak bone mass is a major determinant of bone mass and fracture risk later in life, any factors that could enhance peak bone mass can be considered to be beneficial in preventing osteoporosis in the long-term. Exercise is a lifestyle factor that can be modified and if it does indeed increase peak bone mass, it may offer protection against bone fragility in old age. New evidence suggests that exercise during growth can lead to large increases in bone mass.[2,19] If these increases are maintained into early adulthood, they will result in increased peak bone mass. In contrast, during adulthood exercise results in relatively small gains in bone mass and appears to play a more important role in maintenance of bone density and possibly prevention of early bone loss. Exercise may not always be beneficial however, since athletic primary and secondary amenorrhea may lead to either a failure to gain bone or bone loss.

Current nomenclature: In vitro measurement of bone breaking strength has determined that bone density can account for up to 80 percent of the variance.[37] Therefore bone density is used as a surrogate measure of the breaking strength of bone. In vivo measurement of the mineral content of the skeleton can be done using dual energy X-ray absorptiometry (DXA) or quantitative computed tomography (QCT). Both determine an estimate of bone mineral content, which can be expressed as either a mass or a density. Bone mass is the amount of mineral in grams (g) unadjusted for size. Bone mass divided by the area of the region analysed (g/cm^2) is termed areal bone density. Although areal bone density only adjusts for the width and length of a bone (not depth), it is the unit most commonly reported in the literature. Volumetric bone density is the bone mass divided by the volume of the region measured (g/cm^3) and can be calculated from DXA scans by using equations that estimate the size of the vertebra and femoral neck.[5] This estimate is termed bone mineral apparent density (BMAD).

The term 'density' without a clarifying prefix can be misleading because it does not distinguish between areal, volumetric or apparent bone density. Analysis of samples of bone has shown that true bone density does not increase with age or size. In other words, the chemical composition of the skeleton remains constant during growth and adulthood.[49,56] It is important to acknowledge that an increase or decrease in areal or volumetric bone density is not a change in the chemical composition of bone but rather a change in total bone length, bone size, cortical thickness, medullary width and/or the biomechanical organisation of the trabecular structure.[49]

When is peak bone mass achieved?

Peak bone mass is the term used to describe the maximal lifetime amount of bone tissue aquired in individual bones and the whole skeleton. It is the consequence of the nett accrual of bone mass due to growth during the childhood years and the balance between accrual and resorption rates in the adult premenopausal period. Approximately 80 to 85 percent of peak bone mass has been accrued by the age of menarche. About half of this is achieved during pre-pubertal growth (~10 to 12 years) and the other half is achieved very rapidly in the 2 to 4 years of pubertal growth.[14] Bone mass accrual continues slowly after puberty and contributes a further 15 to 20 percent to the peak bone mass. While it is well accepted that bone mass continues to be accrued after linear growth has ceased (at about 16 years in females) there is still debate about when peak bone mass is achieved. There have been reports that peak bone mass may be achieved from as early as 17 to 18 years to as late as 35 years.[17,33,54]

Theintz et al. (1992) showed no increase in bone density at the femoral neck or the lumbar spine after 17 to 19 years of age.[54] In contrast Mazess and Barden (1991) report that peak bone mass may be achieved later in adulthood. They reported small differences in bone density between women aged 20-24 and women aged 30-34: lumbar spine was 3% higher in the older cohort, but no differences were noted in the femur.[33] There are several reasons for these conflicting results: a) measurement of bone mass at one site may not be representative of the entire skeleton or other sites within the skeleton; b) expression of results as bone mass may differ from expression of results as bone density due to changes in bone shape and c) the results of cross-sectional studies can be influenced by

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the different cohorts.[43]

The results of prospective studies support the notion that peak bone mass may be achieved later rather than earlier in adulthood. Bennell et al. (1997) studied subjects aged 17 to 26 years for one year.[3] This study showed increases in the range of 2.2% in total body bone mass in the female subjects. In a longer study 156 women were followed for up to 5 years and peak bone mass was reported to be achieved between 28 and 30 years of age.[43]

There is also debate about how long peak bone mass is maintained before bone loss begins. There may be a plateau in bone mass or bone loss may occur shortly after peak bone density has been achieved. Two studies support the concept of early bone loss: 1) Matkovic et al. (1994) reported that the bone density of the proximal femur was about 15 percent lower in older premenopausal women compared to younger premenopausal women,[32] and 2) Bonjour et al. (1991) reported that the spines of adolescents (aged 14 to 19 years; n =24) had 10% higher trabecular bone density than young adults (25 to 35 years; n = 24).[4] Not all studies report a statistically significant decline in bone density with age during the pre-menopausal period.[43,35] The maintenance of bone mass in the lumbar spine and femoral neck has been reported in longitudinal studies of women in their thirties.[33,35] Individual variation may explain these conflicting results. This individual variation in the rate of change and direction of change in bone mass was highlighted by Mazess and Barden (1991) who reported that there was no detectable relationship between age and bone mass in a large group of subjects aged 20-39 years. However, 63 subjects decreased spine bone density by more than 2%, whereas 100 subjects showed change of less than 2% in either the positive or negative direction and a further 68 subjects gained more than 2%.[33]

In summary, there is clear evidence that the majority of bone mass is accrued during pre- and peri-pubertal years and that bone mass continues to be accrued at a slower rate in the post-pubertal years. There is still controversy about when peak bone mass is achieved and how long it is maintained, but the timing of the attainment of peak bone mass and bone loss is most likely a) site-specific and b) variable between individuals. This heterogeneity between individuals may be the result of the interaction between genetic and environmental determinants that influence bone mass.

The genetic determinants of peak bone mass

The variance in peak lumbar spine bone density varies between -20% and +20% of the mean. Family studies have shown that 60 to 90% of this variation between individuals is genetically determined.[30] It has also been found that the genetic effect is greater at the lumbar spine (up to 90%) than the femoral neck (up to 70%).[51]

A finding that is clinically important is that premenopausal women with a maternal family history of osteoporosis have low bone density,[1] and perimenopausal women with a family history of hip fracture also have low bone density.[55] Interestingly, daughters of mothers with hip fractures had low bone density at the hip, while daughters of mothers with spine fractures had low bone density at the spine[50,51] indicating that this familial association may also be site specific.

While a large proportion of the variance in bone density may be genetically determined, environmental factors also account for a clinically important proportion of the variance.[48] These influences may not be independent as it has been suggested that there is a common genetic control of both muscle mass and bone mass, both of which can also be affected by exercise.[40]

The osteotrophic effect of exercise on bone density in adult premenopausal women

Early studies of exercising subjects seemed to indicate that exercise could substantially improve bone mass. For example, the dominant arm in tennis players has higher bone mass than their non-dominant arm.[21] The reported differences between the two arms in competitive club players were between 8% and 13%, and even larger differences were reported in the professional players. However, cross-sectional data of mature athletes does not give an indication of whether the adaptation occurred in childhood or adulthood. Therefore, to investigate the effects of exercise on adult bone mass, a longitudinal approach is preferable.

The effect of aerobic exercise on bone density in the adult has been reviewed, and the following consensus has been reached: early cross-sectional studies were confounded by sampling bias and led to the belief that exercise in the adult could lead to large increases in bone density (up to 20%). Intervention studies tended to show mixed results ranging from small increases (2 to 3%), to no change or even bone loss.[15]

Weight training was hypothesised to have a greater osteotrophic effect than jogging or walking since weight training places a greater load on the skeleton to which it must adapt. Intervention studies in premenopausal women ranging from 4 to 24 months have shown inconsistent findings.[16, 18, 29, 47, 53] Some of these studies reported small but statistically significant changes of 1 to 2 percent; others showed no change in bone mass or bone loss. Friedlander et al. (1995) reported that the change in lumbar spine bone density after 2 years of aerobics and weight training in adult women (aged 28 years),[16] was similar to that found by Snow-Harter et al. (1992) after 8 months (1.3% for both studies),[53] indicating the possibility of a plateau in the adaptation to exercise. In the study the femoral neck bone density increased even less (0.5 ± 0.5%) with weight training but this was in contrast to a group of women who only did stretching exercises and who lost 1.9 ± 1.0% ($p<0.05$).

Although weight training places a high load on the skeleton, it is possible that the impact is not high since there is no momentum at the initial contact. The osteotrophic effect on the skeleton of high and low impact loading has also been investigated, albeit in postmenopausal women. Kerr et al. (1996) randomised 56 subjects to a high or low impact exercise group.[27]

Both groups exercised three times per week for 12 months. Increases were found in the high impact group at the trochanter, Wards triangle and ultra distal radius of between 1.7 ± 4.1% and 2.4 ± 4.3%). There was no increase at the femoral neck or other radial sites. There were no changes at any site (relative to baseline) in the low impact high repetition group and a control group lost bone density at most sites (changes between 0.8 ± 5.2 % and -1.4 ± 2.3 %).

Sports where impact loading is high, may also lead to increased bone density. Higher bone density has been reported in athletes who play volleyball and basketball,[45] and in college gymnasts.[46] The extent of the differences can be substantial (see Table 1). It should be remembered that firstly the results could be affected by selection bias, and secondly it can not be determined from these studies what proportion of these benefits were gained from exercise during childhood and adolescence, and what proportion was gained from exercise in adulthood.

The clinical relevance of exercise in the prevention of osteoporosis lies in the reduction of fracture risk late in life. It is generally accepted that a 10% increase in bone density is associated with halving the risk of femoral neck fracture.[22] Moderate exercise of any type in adulthood is unlikely to result in such large increases in bone mass. But a long term commitment to an active lifestyle, including exercise that places a mechanical load on the skeleton, through the pre-, peri-, and postmenopausal years may help reduce fracture risk by reducing bone loss and indirectly by reducing the risk of falling by improving muscular strength, coordination and balance.

The effect of menstrual irregularity on bone density

Research in the mid-eighties and subsequent research has confirmed the long term risk of osteopoenia associated with athletic amenorrhea.[11,31,44] The decrements in bone density are seen relative to both sedentary peers, and eumenorrheic athletic controls. The skeletal site most affected by menstrual dysfunction is the lumbar spine, a site containing a large proportion of trabecular bone.[10,31] The deficit in bone density relative to eumenorrheic athletes has been reported to be as high as twenty percent.[31] Up to four percent of trabecular bone can be lost in the first year of secondary amenorrhea and bone loss continues for at least two years.[38]

More recent studies have reported that oligomenorrhea is also associated with low bone density,[28,34] despite no episodes of amenorrhea.[34] There appears to be a relationship between bone density and the severity of current and previous menstrual history expressed either by category,[9] or the number of cycles per year since age thirteen years.[34] It has also been suggested that more subtle hormonal disturbances may affect bone mass, even when menses is regular. Women with more than one short luteal phase per year or anovulatory cycles, may lose bone mass due to decreased progesterone secretion.[42] But De Souza et al. (1997) followed exercising women for three months and reported that luteal phase insufficiency was associated with decreased progesterone, but not decreased bone density, whereas reduced estrogen production in the follicular phase, despite regular menses, was associated with lower bone density.[8]

Can exercise offset bone loss associated with menstrual dysfunction?

Despite lower bone density at the spine, amenorrheic athletes have either high or normal bone density at weight bearing sites. One explanation for this trend is that exercise may offset the negative effects of amenorrhea at the weight bearing sites. Two factors argue against this explanation: 1) bone density of the weight bearing sites may be higher than the non-weight bearing sites because of previous loading before exposure to estrogen deficiency and 2) trabecular sites are more at risk of early bone loss and cortical sites later bone loss, so that only trabecular bone loss is evident at the study time. Pearce et al. (1997) have reported lower bone density at the weight bearing sites only in ballet dancers with longer time periods of oligomenorrhea.[41]

Exercise with higher impact loading than ballet may offer protection at the weight bearing sites. In a group of figure skaters with a substantial history of menstrual irregularity bone density was enhanced in the lower limbs, but not in the lumbar spine.[52] Robinson et al. (1995) reported that gymnasts had higher bone density at all sites compared with runners who had a similar incidence of menstrual dysfunc-

TABLE 1: Lumbar spine and lower limb bone density in gymnasts, volleyball and basketball players compared to non-athletes.

Group	LS BMD g/cm²	Diff vs control %	FN BMD g/cm²	Diff vs Control %	Reference
Controls	0.11 ± 0.11		0.97 ± 0.10		no 46
Gymnasts	0.17 ± 0.13	+ 5.4 %	1.09 ± 0.12	+ 12.4 %	no 46
			Calcaneus BMD		
Controls	1.15 ± 0.03		0.424 ± 0.019		no 45
Volleyball	1.32 ± 0.04	+ 14.8 %	0.536 ± 0.017	+ 26.4 %	no 45
Basketball	1.29 ± 0.03	+ 12.1 %	0.575 ± 0.022	+ 35.6 %	no 45

Abbreviations: LS BMD = Lumbar spine bone mineral density; FN BMD = femoral neck bone mineral density; Diff = difference

tion.[36] It is possible however that the gymnasts' higher bone density was not due to a protective effect of exercise during the time period of menstrual irregularity, but rather may have been the result of exercise loading prior to the exposure of estrogen deficiency. Only longitudinal studies can clarify these issues.

Resumption on menses and its effect on bone density in previously amenorrheic athletes

Gains in lumbar spine bone density (6-7% per year) have been shown in the year or two immediately following resumption of menses in previously amenorrheic athletes.[11] These gains however, did not appear to be sufficient to completely reverse the original deficits in bone density compared with controls (see Table 2) and one study has shown no further increase in the third year after resumption of menses.[38] Other more long term studies in subjects with a prior history of menstrual irregularity who had been menstruating regularly for up to 10 years at follow-up still showed a 14-15% deficit in lumbar spine bone density.[26,35] These data suggest that there is irreversible bone loss in athletes with former menstrual irregularity .

One possible treatment for athletic amenorrhea is hormone replacement therapy (HRT) in the form of oral contraceptives. The effectiveness of hormonal therapies in the treatment of athletic amenorrhea is unknown. To date there has been only one retrospective clinical study and one randomized clinical trial investigating the effect of estrogen replacement therapy and oral contraceptive use on bone density in young women with hypothalamic amenorrhea. Cummings et al. (1996) reported that estrogen replacement therapy for 24 to 30 months increased vertebral and femoral neck bone density by 8 ± 1.2% and 4.1 ± 0.3% respectively in 8 amenorrheic runners.[7] In contrast, 5 amenorrheic runners who did not agree to take HRT experienced non-significant decreases of less than 2.5% in bone density at both sites. Hergenroeder et al. (1997) investigated the effect of 12-months of oral contraceptives, medroxy-progesterone or placebo on bone density in 15 amenorrheic women aged 14 to 28 years.[20] Four of the subjects had a current diagnosis of anorexia or bulimia nervosa, and one subject in each group did not exercise. Twelve months of oral contraceptive use led to a significant increase in bone density at the lumbar spine but not the femoral neck (5.4% and 3.7% respectively). After 12 months changes in bone density were not statistically significant for the medroxy-progesterone and placebo groups (lumbar spine: -10.2% and -0.7% respectively and femoral neck: +4.2% and -3.4% respectively). The results of these studies suggest that either HRT or oral contraceptive use may be effective in increasing lumbar spine bone density and femoral neck bone density, although the latter may only be significant after at least two years. Further studies are needed to confirm these results as the sample sizes were small and in the study by Hergenroeder et al. (1997) the subject sample was not homogenous (athletes and patients with eating disorders). It is recommended that athletes with amenorrhea be monitored for bone loss and early intervention be considered.[7,20]

The osteotrophic effect of exercise during growth

During childhood and adolescence there are large gains in bone mass that may be magnified by exercise. The first studies to show that large increases in bone density may be achieved during growth were unilateral loading studies. The strength of these studies is that the non-dominant limb controls for genetic determinants of bone density. The radius and humerus of adult tennis and squash players had 9% to 35% greater cortical thickness and bone mass in their playing arm compared to the non-playing arm.[21,24] Kannus et al even reported that the bilateral differences in bone mass were two to four times higher in those players who had started training before or at menarche (10 to 23%), compared to those who started more than 15 years after menarche (2% to 9%). Similarly, femoral neck, trochanter and distal radius bone density was greater (8-30%) in young elite gymnasts (aged 7 to 11 years).[2,13] The greatest differences were found in the arms, a weight bearing site in gymnastics. Selection bias is unlikely to explain a large proportion of the higher bone density in the gymnasts since Dyson et al.[13] reported that there were no significant differences in bone density between the mothers of the gymnasts and the mothers of the controls.

Does intense training in childhood confer benefits in bone density in adulthood?

Retired gymnasts have also been shown to have site specific higher bone density supporting the hypothesis that these childhood gains in bone mass can be maintained into adulthood.[2] Despite reduced physical activity in retirement, bone density was 1 to 1.5 standard deviations higher in the retired gymnasts at the weight bearing sites, but normal at the non-weight bearing sites compared to age-matched controls. This trend for retired athletes to have site specific surfeits in bone density related to the unique loading patterns

TABLE 2: Effect of resumption of menses on lumbar spine bone mineral density (g/cm²)

Baseline Control	Baseline MI	Difference vs control	Follow-up* Control	Follow-up* MI	Difference vs control	Reference
1.300	1.120	- 14 %	1.369	1.198	- 12.5 %	no 9, 10
1.250	1.050	- 16%	1.290	1.190	- 8 %	no 23
1.088	0.946	- 13 %	1.043	0.936	- 10 %	no 34, 35

* *Not all subjects were available for follow-up. Abbreviations: MI = menstrual irregularity*

associated with training in childhood and adolescence has also been reported in tennis players,[21] soccer players,[12] and weight lifters.[25]

Moderate physical activity and bone density in active non-athletic children

Improvements in bone density in competitive athletes may not be reflected to the same extent in active non-athletic children. Gunnes and Lehman (1996) reported that weight bearing exercise accounted for 5% to 16% of the variance in the change in bone density with age in children and adolescents depending on the site, age and gender.[19]

Nevertheless, several long term longitudinal studies have shown that exercise was a predictor for adult bone density.[6,37] Cooper et al. (1995) also investigated the relationship between bone density in adulthood and exercise. Women in the highest activity category had 12% higher bone density at the femoral neck than women in the lowest activity category.[6] Femoral neck bone density in the women in the moderate activity category was 7% higher than the least active group.

These results however may be biased, as children who have a larger musculoskeletal mass may be more likely to participate in physical activity. Intervention studies are currently being conducted, but few have been published. Morris et al. (1997) conducted a ten-month exercise program structured into the school curriculum for pre-pubertal girls (aged 9 to 10 years). The bone density increased more in the exercise group compared with controls at the legs, arms, lumbar spine, and femoral neck.[36]

CONCLUSION

For exercise to have a role in the prevention of osteoporosis the osteotrophic response must be large enough to be considered clinically important. Also, the gains due to exercise must be maintained into adulthood and later in life. Large increases in bone density have been reported in children and adolescents involved in competitive exercise programmes with extensive training, but it is unknown how long these are maintained. The evidence that some retired athletes have site-specific higher bone density related to the unique loading patterns of their sport suggests that at least some of the benefits are not lost. In contrast, moderate weight bearing exercise in non-athletic children is associated with somewhat less benefit to bone density in adulthood. Exercise intervention in adulthood is even less positive for enhancing bone mass unless the impact is high, but the importance of exercise in adulthood is concerned mainly with conservation of bone.[39] It can be said that the adult skeleton is much more responsive to the adverse effects of unloading than to the beneficial effects of overloading.[39] Exercise may not always be beneficial however, especially if it is associated with the development of amenorrhea or oligomenorrhea which may lead to large deficits in bone density (up to 20%) due to either a failure to gain bone or bone loss. Recent evidence has also shown that even long term resumption of regular menses has failed to restore these deficits. The utility of exercise to offer protection or restore bone mass in amenorrheic athletes appears to be limited, alternative measures for bone gain must be investigated and promoted. In summary, exercise has an important role in increasing peak bone mass and bone strength during the growing years and maintaining bone mass in the premenopausal years. These findings support the promotion of weight bearing exercise in girls, teenagers and young women and highlight the importance of regular menstrual function.

REFERENCES

1. *Armamento-Villareal R, Villareal DT, Aviolo LV & Civitelli R. Estrogen status and heredity are major determinants of premenopausal bone mass. J Clin Invest 1992; 90, 2464-71.*
2. *Bass S, Pearce G, Bradney M, Hendrick E, Delmas P, Harding A & Seeman E. Exercise before puberty may confer residual benefits in bone density in adulthood: studies in active prepubertal and retired female gymnasts. J Bone Miner Res 1998; 13(2), 500-7.*
3. *Bennel KL, Malcolm SA, Khan KM, Thomas SA, Reid SJ, Brukner PD, Ebeling PR & Wark JD. Bone mass and bone turnover in power athletes, endurance athletes, and controls: A 12-month longitudinal study. Bone 1997: 20, 477-84.*
4. *Bonjour JP, Theintz G, Buchs B, Slosman D & Rizzoli R. Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. J Clin Endocrinol Metab 1991; 73, 555-63.*
5. *Carter DR, Bouxsein ML & Marcus R. New approaches for interpreting projected bone densitometry data. J Bone Miner Res 1992; 7, 137-45.*
6. *Cooper C, Cawley M, Bhalla A, Egger P, Ring F, Morton L & Barker D. Childhood growth, physical activity, and peak bone mass in women. J Bone Miner Res 1995; 10(6), 940-7.*
7. *Cummings DC. Exercise -associated amenorrhea, low bone density, and estrogen replacement therapy. Arch Intern Med 1996; 156, 2193-5.*
8. *De Souza MJ, Miller BE, Sequenzia LC, Luciano AA, Ulreich S, Stier S, Prestwood K & Lasley BL. Bone health is not affected by luteal phase abnormalities and decreased ovarian progesterone production in female runners. J Clin Endocrinol Metab 1997; 82, 2867-2876.*
9. *Drinkwater BL, Bruemner B & Chestnut III CH. Menstrual history as a determinant of current bone density in young athletes. JAMA 1990; 263(4). 545-8.*
10. *Drinkwater B, Nilson K, Chestnut C, Bremner W, Shainholtz S. & Southworth M. Bone mineral content of amenorrheic and eumenorrheic athletes. N Engl J Med 1984; 311, 277-81.*
11. *Drinkwater B, Nilson K, Ott S & Chestnut III CH. Bone mineral density after resumption of menses in amenorrheic athletes. JAMA 1986; 256, 380-2.*
12. *Duppe H, Gardsell P, Johnell O & Ornstein E. Bone mineral content in female junior, senior and former football players. Osteoporosis Int 1996; 6, 437-41.*
13. *Dyson K, Blimkie CJR, Davison S, Webber CE & Adachi JD. Gymnastic training and bone density in pre-adolescent females. Med Sci Sports Exerc 1997; 29(4), 443-50.*
14. *Faulkner RA, Bailey DA, Drinkwater DT, Wilkinson AA, Houston CS & McKay HA. Regional and total body bone mineral content, bone mineral density, and total body tissue composition in children 8-16 years of age. Calcif Tissue Int 1993; 53, 7-12.*
15. *Forwood M & Burr D. Physical activity and bone mass: exercises in futility? Bone Miner 1993; 21, 89-112.*
16. *Friedlander AL, Genant HK, Sadowsky S, Byl NN & Gluer C. A two-year program of aerobics and weight training enhances bone mineral density of young women. J Bone Miner Res 1995; 10(4), 574-85.*

17. Gilsanz V, Gibbens DT, Carlson M, Boechat MI, Cann CE & Schulz EE. Peak trabecular vertebral density: Comparison of adolescent and adult females. Calcif Tissue Int 1988; 43, 260-2.
18. Gleeson PB, Protas EJ, LeBlanc AD, Schneider VS & Evans HJ. Effects of weight lifting on bone mineral density in premenopausal women. J Bone Miner Res 1990: 5(2), 153-8.
19. Gunnes M & Lehman EH. Physical activity and dietary constituents as predictors of forearm cortical and trabecular bone gain in healthy children and adolescents: a prospective study. Acta Paediatr 1996; 85, 19-25.
20. Hergenroeder AC, O'Brian Smith E, Shypailo R, Jones LA, Klish WJ & Ellis K. Bone mineral changes in young women with hypothalamic amenorrhea treated with oral contraceptives, medroxyprogesterone, or placebo over 12 months. Am J Obstet Gynecol 1997; 176, 1017-25.
21. Huddleston A, Rockwell D, Kulund DN & Harrison B. Bone mass in lifetime tennis athletes. JAMA 1980; 244(10), 1107-9.
22. Johnston GC, Slemenda CW & Melton LJ. Clinical use of bone densitometry. N Engl J Med 1991: 324, 1105-9.
23. Jonnavithula S, Warren MP, Fox RP & Lazaro MI. Bone density is compromised in amenorrheic women despite return of menses: a 2-year study. Obstet Gynecol 1993; 81, 669-674.
24. Kannus P, Haapasalo H, Sankelo M, Sievanen H, Pasanen M, Heinonen A, Oja P & Vuori I. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. Ann Intern Med 1995; 123, 27-31.
25. Karlsson MK, Johnell O & Obrant KJ. Is bone mineral density advantage maintained long-term in previous weight lifters. Calcif Tissue Int 1995; 57, 325-8.
26. Keen AD & Drinkwater BL. Irreversible bone loss in former amenorrheic athletes. Osteoporosis Int 1997; 7, 311-5.
27. Kerr DA, Morton A, Dick I & Prince RL. Exercise effects on bone mass in postmenopausal women are site specific and load-dependent. J Bone and Miner Res 1996; 11, 218-225.
28. Lloyd T, Myers C, Buchanan JR & Demers LM. Collegiate women athletes with irregular menses during adolescence have decreased bone density. Obstet Gynecol 1988; 72, 639-42.
29. Lohman T, Going S, Pamenter R, Hall M, Boyden T, Houtkooper L, Ritenbaugil C, Dare L, Hill A & Aickin M. Effects of resistance training on regional and total bone mineral density in premenopausal women: a randomized prospective study. J Bone Miner Res 1995; 10, 1015-24.
30. Lutz J. Bone mineral, serum calcium, and dietary intakes of mother/daughter pairs. Am J Clin Nutr 1986; 44, 99-106.
31. Marcus R, Cann C, Madvig P, Minkoff J, Goddard M, Bayer M, Martin M, Gaudiani L, Haskell W & Genant H. Menstrual function and bone mass in elite women distance runners: Endocrine metabolic features. Ann Intern Med 1985; 102, 158-63.
32. Matkovic V, Jelic T, Wardlaw GM, Ilich JZ, Goel PK, Wright JK, Andon MB, Smith KT & Heaney RP. Timing of peak bone mass in Caucasian females and its implications for the prevention of osteoporosis. J Clin Invest 1994; 93, 799-808.
33. Mazess RB & Barden HS. Bone density in premenopausal women: effects of age, dietary intake. physical activity. smoking, and birth-control pills. Am J Clin Nutr 1991; 53, 132-42.
34. Micklesfield LK, Lambert EV, Fataar AB, Noakes TD & Myburgh KH. Bone mineral density in mature. premenopausal, ultramarathon runners. Med Sci Sports Exerc 1995; 27, 688-96.
35. Micklesfield LK, Reyneke L, Fataar A & Myburgh KH. Long-term restoration of deficits in bone mineral density is inadequate in premenopausal women with prior menstrual irregularity. Clin J Sports Med 1998; In press .
36. Morris FL, Naughton GA, Gibbs JL, Carlson JS & Wark JD. Prospective ten-month exercise intervention in premenarchel girls: positive effects on bone and lean mass. J Bone Miner Res 1997; 12(9), 1453-62.
37. Mosekilde L, Mosekilde L & Danielsen CC. Biomechanical competence of vertebral trabecular bone in relation to ash density and age in normal individuals. Bone 1987; 8, 79-85.
38. Otis, C.L. Exercise-associated amenorrhea. Clin Sports Med 1992; 11(2), 351-61.
39. Parfit M. The two faces of growth - benefits and risks to bone integrity. Osteoporosis Int 1994; 4(6), 382-98.
40. Parfitt AM. Genetic effects on bone mass and turnover-relevance to black/white differences. J Am College of Nutrition 1997: 16(4), 325-33.
41. Pearce G. Bass S. Young N, Formica C & Seeman E. Does weight bearing exercise protect against the effects of exercise - induced oligomenorrhea on bone density? Osteoporosis Int 1996; 6, 448-52.
42. Prior JC, Vigna YM, Schechter MT & Burgess AE. Spinal bone loss and ovulatory disturbances. N Engl J Med 1990: 323(18), 1221-7.
43. Recker RR, Davies K, Hinders SH, Heaney RP, Stegman MR & Kimmel DB. Bone gain in young adult women. JAMA 1992; 268, 2403-8.
44. Rencken ML, Chesnut III CH & Drinkwater BL. Bone density at multiple skeletal sites in amenorrheic athletes. JAMA 1996; 276(3), 238-40.
45. Risser WL, Lee EJ, Leblanc A, Poindexter GBW, Risser JMH & Schneider V. Bone density in eumenorrheic female college athletes. Med Sci Sports Exerc 1990; 22(5), 570-4.
46. Robinson TL, Snow-Harter C, Taaffee DR, Gills D, Shaw J & Marcus R. Gymnasts exhibit higher bone mass than runners espite similar prevalence of amenorrhea and oligomenorrhea. J Bone Miner Res 1995; 10(1), 26-35.
47. Rockwell JC, Sorensen AM, Baker S, Leahey D, Stock JL, Michaels J & Baran DT. Weight training decreases vertebral bone density in premenopausal women: a prospective study. J Clin Endocrinol Metab 1990; 71(4), 988-92.
48. Salamone LM, Glynne NW, Black DM, Ferrell RE, Palermo L, Epstein RS, Kuller LH & Cauley JA. Determinants of premenopausal bone mineral density: the interplay of genetic and lifestyle factors. J Bone Miner Res 1996; 11(10), 1557-65.
49. Schonau E, Wentzlik U, Dietrich M, Scheidhauer K & Klein K. Is there an increase in bone density in children? Lancet 1993; 342, 689-90.
50. Seeman E, Hopper JL, Bach LA, Cooper ME, Parkinson E, McKay J & Jerums G. Reduced bone mass in daughters of women with osteoporosis. N Eng J Med 1989; 320(9), 554-8.
51. Seeman E. Reduced bone density in women with fractures: contribution of low peak bone density and rapid bone loss. Osteoporosis Int Suppl 1994; 1, S15-25.
52. Slemenda C & Johnston C. High intensity activities in young women: site specific bone mass effects among female figure skaters. Bone Miner 1993: 20, 125-32.
53. Snow-Harter C, Bouxsein JL, Lewis BT, Carter DR & Marcus R. Effects of resistance and endurance exercise on bone mineral status of young women: A randomized exercise intervention trial. J Bone Miner Res 1992; 7(7), 761-9.
54. Theintz G, Buchs B, Rizzoli R, Sloman D, Clavien H, Sizonenko P & Bonjour J. Longitudinal monitoring of bone mass accumulation in healthy adolescents: Evidence for a marked reduction after 16 years of age at the levels of the lumbar spine and femoral neck in female subjects. J Clin Endocrinol Metab 1992; 75(4), 1060-5.
55. Torgerson D, Campbell MK & Reid DM. Life-style, environmental and medical factors influencing peak bone mass in women. Br J Rheum 1995; 34, 620-4.
56. Trotter M & Hixon BB. Sequential changes in weight, density, and percentage ash weight of human skeletons from an early fetal period through old age. Anat Rec 1974; 179, 1-8.
57. Welten DC, Kemper HCG, Post GB, Van Mechelen W, Twisk J, Lips P & Teule GJ. Weight-bearing activity during youth is a more important factor for peak bone mass than calcium intake. J Bone Miner Res 1994; 9(7), 1089-96. □

RESEARCH ARTICLE

The force absorption and rebound characteristics of cricket batting pads at four impact velocities

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ABSTRACT

Objective: An experiment was conducted to compare the ability to absorb impact forces and rebound characteristics of an "experimental" pair of pads with three pairs of cricket batting pads currently in use.
Design: The forces absorption and rebound characteristics of the "experimental" (P1) pair of pads was compared with three pairs of cricket batting pads (P2, P3 and P4) currently in use, at four impact velocities. Slow-medium (S1) (15.34 $m.s^{-1}$), Fast-medium (S2)(22.97 $m.s^{-1}$), Fast (S3)(30.68 $m.s^{-1}$) and Express (S4)(34.45 $m.s^{-1}$). These pads all showed differences in their structure and composition, with P1 manufactured from a polyurethane compound, while the inner part of P2, P3 and P4 was made of padding strengthened by a cane or plastic reinforcement rod, as well as a second spongy inner layer. The impact forces were measured using the drop test where a weighted ball was dropped vertically onto the surface of the batting pads with the vertical forces (N) measured on a Kistler Piezoelectrical multicomponent type 9281 A11 force platform. The rebound characterstics were determined by measuring the distance (m) the ball rebounded off the pad when delivered from a Brell Bowling Machine. The two- and one-way analysis of variance, with Fisher's method of multiple comparison, were used to test for significant differences ($p<0.01$) between the pads at the four impact velocities and Scheffe simultaneous confidence intervals were used to calculate the contrast in the pads.
Setting: This was a laboratory based test carried out in the Biokinetics Centre, Department of Human Movement Studies.
Results: The assessment of the force absorption characteristics showed that P1 was significantly better at absorbing the impact forces than all other pads at all impact velocities, with the exception of similar characteristics shown to P2 at S3 and S4. At the faster impact velocities, P1 and P2 showed similar force absorption characteristics, which were significantly better than both P3 and P4. The assessment ofthe rebound characteristics showed that P1 produced significantly greater post-impact rebound distance at all impact velocities. At the faster velocities P4 showed significantly smaller rebound characteristics than P1 and P3, with similar scores to P2 at S3. P2 showed significantly smaller rebound distance to P4 at S4.
Conclusions: The results showed significant differences between the impact kinetics of the cricket batting pads at various impact velocities. These differences were as a result of the differences in the structure and composition of the protective part of the pads, with some pads better able to absorb the impact forces of the ball providing the batsman with greater protection. Further, a number of other factors that need to be taken into account when choosing pads or in the manufacturing thereof, were highlighted.
Keywords: Cricket, equipment, force absorption, rebound, kinematics

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INTRODUCTION

Deaths from cricket date as far back as 1751 when, according to cricket lore, the passing of Frederick Louis, the Prince of Wales and father of George III, died hours after being struck on the head by a cricket ball.[1] It has been suggested that six deaths per year occur in the United Kingdom as a result of playing cricket.[2]

Besides back injuries to fast bowlers, the major areas of concern to players, coaches and administrators are impact injuries while batting, particularly to the head and face and the fingers.[1,3,4,5] The head and facial injuries were primarily as a result of being struck by the ball while batting without a helmet or with a helmet with ear-pieces only and included concussions, broken nose and cheek bones and lacerations requiring stitches, around the eyes, mouth and chin. Four of the eye injuries were as a result of the ball deflecting off the top edge of the bat while hooking and striking the eye on the side of the dominant hand.[6] Injuries to the fingers, as a result of impact from the ball, were found to be the most common while batting.[1,3,4,5]

Protective equipment such as batting gloves and

pads have been used for well over a century, while during the last two decades the batting helmet has become a standard part of the batsman's protective equipment. However, only two studies have aimed at evaluating this protective equipment in a sport where a solid ball with a mass 156g is propelled towards the batsman from a distance of approximately 18m at speeds up to 38m/s. Both studies recommended that the equipment manufacturers need to increase the protection offered by the batting glove.[7,8]

An investigation into the shock absorption of cricket batting pads in order to determine which factors influence the shock absorption capabilities and their ability to conform to standards showed adequate impact resistance was provided around the shin and ankle.[8] Six of the eleven pads did not meet the requirements for repeated drops on the knee roll, particularly for the higher temperature and humidity conditions. A high correlation was found between the peak deceleration and knee roll and shin thickness. Higher temperature and humidity had a negative effect on the force absorption characteristics, while the construction and pad thickness significantly improved the shock absorption characteristics.

Lower limb injuries occurring while batting were mainly hamstring, quadriceps and calf muscle pulls as a result of running between the wicket.[1,4,5,7,9] Although the literature does not report any serious impact injury to the protected part of the lower limbs, which may suggest that batting pads offer adequate protection, cricket equipment manufacturers continually need to assess new designs and material from which to manufacture their protective equipment. The final product not only has to meet the legal requirements of the sport, but has to balance the amount of protection offered to the batsman with a number of factors that may enhance or inhibit performance. These may include the following: comfort required to wear this protective equipment for long periods at a time; the pads need to be light so as not to have a negative effect on the running speed between the wickets; during 3-5 day matches where there are usually a number of fielders in catching positions in close proximity to the batsman it would be advantageous if the pads have a low ball-pad coefficient of restitution reducing the risk of "bat-pad" catches (the ball deflecting from the bat onto the pads and then caught by a fielder), while in limited-overs matches, where there are seldom fielders in close catching positions, the converse would apply whereby batsman may gain an advantage if the pads have a high ball-pad coefficient of restitution and can score runs or leg-byes from balls striking the pads.

Following on the assessment of the force absorption characteristics of cricket batting gloves by Stretch and Tyler (1995), the experiment aimed to evaluate and compare the force absorption and rebound characteristics of four pairs of cricket batting pads at various impact velocities and to compare a "new" pair of cricket batting pads with cricket batting pads currently in use. Secondly, the experiment sought to identify reasons, if any, for differences in the impact kinetics in order to assist manufacturers to modify batting pads to suit the modern game.

METHODS

One "experimental" (P1) and three pairs of cricket batting pads that are currently in use (P2, P3 and P4), supplied by different manufacturers, were used in this study to test the force absorption and the rebound characteristics of cricket batting pads. The structure and composition of the protective part of the pads differed in all four makes of the pads assessed. All the pads had two protective components, the outer protective layer that offered protection to the lower leg, knee and part of the upper leg, and a spongy padded layer that fitted inside the pad offering additional protection to the front of the lower leg. The part of P1 and P2 below and above the knee was made up of nine vertical segments or 'rolls', while that of P3 and P4 consisted of eight segments. At the knee area P2, P3 and P4 were similar with three horizontal segments or 'rolls', while P1 was made up of five horizontal segments. The outer part of the P1 consisted of a solid polyurethane compound, while that of P2, P3 and P4 was made of padding strengthened by a cane or plastic reinforcement rod in each of the 'rolls'. The second protective layer of the pads was very similar in thickness, size and composition, being made of a spongy layer.

The impact velocities used for both experiments were based on release velocities[10], taking into account a 14,3%[11] decrease in the ball velocity by the time it reaches the batsman. These were classified as slow-medium (S1), fast-medium (S2), fast (S3) and express (S4) (Table 1).

TABLE I: Release and impact velocities of the ball under match conditions and the calculated mass of the experimental "ball"

	Release Velocity ($m.s^{-1}$) (Abernethy, 1981)	Impact Velocity ($m.s^{-1}$) (Penrose et al 1976	Mass of Experimental "Ball" (g)
Slow-medium (S1)	17.9	15.3	630.8
Fast-medium (S2)	26.8	23.0	944.4
Fast (S3)	35.8	30.7	1262.8
Express (S4)	40.2	34.5	1416.6

The procedure used in the assessment of the force absorption characteristics of the four batting pads was based on the drop test.[12] In this test the dropping mass falls onto the surface of the batting pads (P1, P2, P3 and P4) at four impact velocities (S1, S2, S3, and S4), with the vertical forces measured by a Kistler Piezoelectrical multicomponent type 9281 A11 force platform (Figure 1) installed and calibrated to the manufacturer's specification. By means of the Piezoelectrical transducers an electric voltage, for the determination of the force exerted on the force platform, was obtained. A Kistler electronic amplifier type 9805 was attached to the piezoelectrical force platform by means of a 9-core cable in order to determine the force the ball exerted through the pads onto the force

platform. The voltage changes received from the piezo-electrical transducers passed through the amplifier to the computer which was fitted with an analogue to digital converter card (P.C. 30) to convert the impulses for storage and later retrieval and analysis.

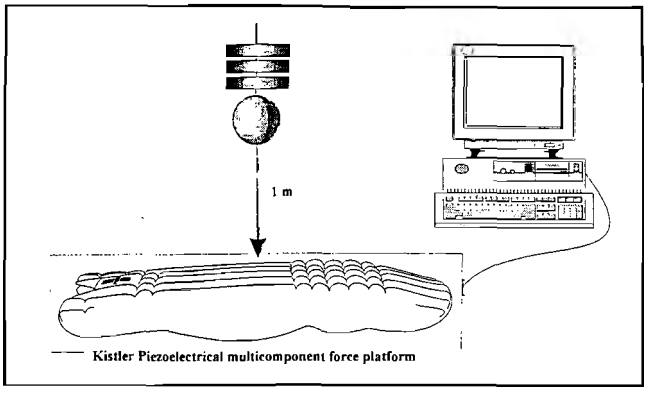

Figure 1: *Experimental set-up for measuring the force absorption characteristics of the cricket batting pads.*

The impact velocity of the ball under match conditions was simulated using an experimental "ball" which was weighted and dropped vertically onto the batting pad from a height of 1 metre. The weight of the experimental "ball" was determined as described below:

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

Momentum = mv (i)

where: m = Mass of ball (156 g)

v = Velocity of the ball at impact from Table I

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

Velocity = 2gd (ii)

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

d = Distance which remained constant at 1 metre

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

Mass = p/v

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

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

The pads were positioned on the force platform so that impact would occur just below the knee. At this site all the manufacturers ensured that maximum protection occurred by providing additional padding on the inside of the pad. The testing of the cushioning properties for one pad was carried out at all four impact velocities, before proceeding to the next pad. This ensured that the positioning of the pads for the various impact velocities was constant. Each pad was subjected to fifteen impacts with the weighted "ball" at S1, S2, S3 and S4 in order to simulate impact of the four release velocities.

The procedure used in the assessment of the rebound characteristics of the four batting pads required a cricket ball to be projected by a Brell Bowling Machine at four release against the batting pads, with the rebound distance measured (Figure 2). The bowling machine velocities were determined according to the guidelines set out by the manufacturer (Table 2). The following bowling machine settings were used: S1 was set at number 3, S2 was set between number 4 and 5, S3 was set between number 5 and 6, and S4 was set between number 6 and 7. The bowling machine projected a Kookaburra ball (156g) onto an artificial surface (astroturf) and then onto the pad which was positioned 3m away so that the angle of approach would be as close to 45° as possible. The pad was strapped onto a prosthesis which was secured onto a stable base and positioned at an angle of 90° to the testing surface. The small ball flight distance, as well as the securing of the pads in position, ensured a constant angle of approach of the ball prior toimpacting the pad. The rebound distance of the ball was measuredusing a tape measure positioned along the length of the cricket pitch. In addition, the testing procedure was video taped and the rebound distance was verified from this recording. The testing of the rebound characteristics for all four pads was carried out at S1, before adjusting the bowling machine speeds to S2, S3 andS4. This ensured that the pre-impact ball velocity was kept constant for the different pads.

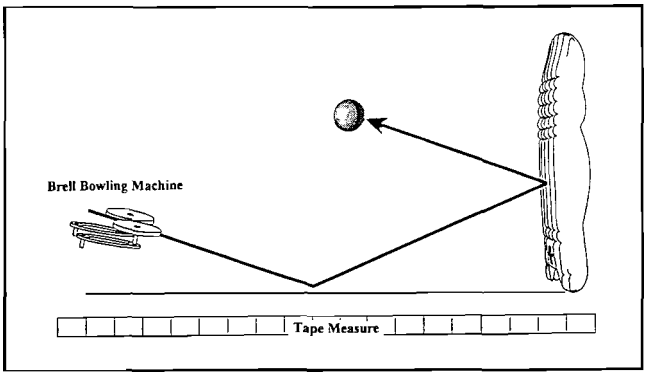

Figure 2: *Experimental set-up for measuring the rebound characteristics of the cricket batting pads.*

TABLE II: Brell Bowling machine release velocities

	Bowling machine setting (km/h)	Release Velocity ($m.s^{-1}$)	
1	45.58	12.66	
2	54.40	15.11	
3	65.70	18.25	S1
4	80.90	22.47	S2
5	102.98	28.61	S3
6	135.52	37.64	S4
7	182.25	50.63	

The statistical package MINITAB11 and SAS were used to compute single variable statistics and the following statistical tests and graphical tools aided in

checking the validity of the tests and to test the force absorption and rebound differences between the various batting pads at each of the four impact velocities.

- A one-way analysis of variance was used to examine whether any inter-pad differences occurred or whether the pads showed similar impact characteristics at the different impact velocities, with the Fisher method of multiple comparisons used to establish the significant differences,
- A two-way analysis of variance was conducted to test for significant interaction between the four pads and the different impact velocities, and
- Scheffe simultaneous confidence intervals were calculated to examine a contrast in the four pads.

The one percent level of significance was used for all tests and confidence intervals.

RESULTS

The mean force absorption and rebound characteristics for the different batting pads at the four velocities are shown in Table 3. The one-way analyses of variance carried out on the data showed significant differences between the four pads for both rebound distance (F=5.81) and force absorption (F=7.80). The confidence intervals generated by Fisher's method indicate the significant differences (Table 4).

TABLE III: Force absorption (N) and rebound distance (m) values for the cricket batting pads at the various impact velocities

	Force Absorption (N)		Rebound Distance (m)	
	Mean	SD	Mean	SD
Speed 1				
P1	43.34	3.08	7.84	0.19
P2	51.04	3.67	7.44	0.12
P3	59.18	3.02	7.45	0.15
P4	59.89	2.91	7.16	0.09
Speed 2				
P1	69.61	3.00	9.27	0.20
P2	75.56	2.45	8.11	0.12
P3	87.88	2.47	8.64	0.15
P4	88.19	3.93	8.01	0.07
Speed 3				
P1	91.23	3.36	11.06	0.43
P2	91.60	2.60	10.07	0.11
P3	104.20	3.86	10.49	0.08
P4	107.45	5.88	10.07	0.08
Speed 4				
P1	108.13	4.40	12.52	0.19
P2	106.27	3.43	10.89	0.09
P3	120.97	4.62	11.54	0.31
P4	25.21	4.24	11.13	0.12

The assessment of the force absorption characteristics showed that P1 was significantly better at absorbing the impact forces than all other pads at all four impact velocities, with the exception of similar characteristics shown to P2 at S3 and S4. When comparing P1 and P2, P1 showed significantly better ability to absorb impact forces at the slower velocities than P2, with similar results recorded at the faster velocities (S3 and S4). P2 showed significantly better force absorption characteristics to P3 and P4 at all the impact velocities. P3 and P4 showed similar force absorption abilities at S1, S2 and S3, with P3 reflecting better force absorption characteristics at the fastest velocity (S4).

At the slower velocities P1 showed significantly greater ability to absorb the impact forces than P2, P3 and P4. At these slower impact velocities P2 was significantly better than both P3 and P4, while similar force absorption characteristics were found for P3 and P4. At the faster impact velocities, P1 and P2 showed similar force absorption characteristics, which were significantly better than both P3 and P4.

TABLE IV: Summary of the Fisher multiple comparison test for the force absorption and rebound distance of the cricket batting pads

	Force Absorption			Rebound Distance		
	P1	P2	P3	P1	P2	P3
Speed 1						
P2	-10.81*			25.49*		
	- 4.60			53-84		
P3	-18.95*	-11.25*		24.49*	-15.18	
	-12.74	- 5.04		52.84	13.18	
P4	-19.66*	-11.96	-3.812	54.16*	14.49*	15.49*
	-13.45	- 5.75	2.40	82.51	42.84	43.84
Speed 2						
P2	-8.89*			101.82 *		
	-3.00			130.45		
P3	-21.22*	-15.27*		49.15 *	-66.98*	
	-15.32	- 9.38		77.78	-38.35	
P4	-21.53*	-15.58	- 3.26	112.15*	-3.98	48.68*
	-15.63	- 9.69	2.64	140.78	24.65	77.32
Speed 3						
P2	-3.70			77.14*		
	4.30			122.19		
P3	-16.30*	-16.60*		34.81*	-64.86*	
	-8.29	- 8.60		79.86	-19.81	
P4	-19.54*	-19.85*	-7.25	76.81*	-22.86	19.47*
	-11.54	-11.84	0.76	121.86	22.19	64.53
Speed 4						
P2	-2.22			142.94*		
	5.95			181.72		
P3	-16.93*	-18.79*		78.61*	-83.72*	
	- 8.76	-10.62		117.39	-44.94	
P4	-21.17*	-23.03*	-8.32*	118.94*	-43.39*	20.94*
	-13.00	-14.86	-0.15	157.72	-4.61	59.72

**Significant difference (P<0.01)*

At all four velocities P1 recorded a greater post-impact rebound distance than the other pads. P2 showed similar rebound distances to P3 at S1 and to P4 at S2 and S3. P2 showed rebound distances that were significantly greater than P4 at S1 and significantly smaller than P3 at S2, S3 and S4, and P4 at S4. P3 was significantly greater than P4 at all the impact velocities, while P4 was significantly smaller than all the pads at all four the impact velocities, with the exception of recording similar rebound distances to P2 at S2 and S3. At the slower velocities P4 showed a significantly smaller rebound distances than P1, P2 and P3, with the exception of similar scores to P2 at S2. P3 recorded significantly smaller values than P1 at S1 and S2 and P2 at S1. P2 and P3 showed similar rebound characteristics at S1. At the faster velocities P4 showed significantly smaller rebound characteristics than P1 and P3, with similar scores to P2 at S3. P2 showed significantly smaller rebound distance to P4 at S4.

Further investigation yielded that the 99% Scheffe simultaneous confidence intervals for the contrast that involved a comparison of P2 with the other three pads, were significant at S2, S3 and S4, indicating that the force absorption and rebound distance characteristics of this pad rated the best. P2 did not differ sigffificantly from the other pads at S1.

DISCUSSION

The principal finding of this study was that there is a significant difference between the impact kinetics of the cricket batting pads at various impact velocities. These differences would be as a result of the differences in the structure and composition of the protective part of the pads, with some makes of pads better able to absorb the impact forces of the ball providing the batsman greater protection, while others were better able to reduce the rebound distance of the ball after impact.

From the findings it would appear that the batting pads best able to absorb the impact forces of a ball bowled at fast to express velocities would be those manufactured from a polyurethane material (P1). A second advantage, although not tested experimentally, is that these pads are very light and comfortable to wear and as a result they should not have as great a negative effect on the running speed of the batsman as the heavier pads currently used. In limited-overs cricket where an off-field umpire uses slow motion TV replays to decide whether a batsman is run-out or not, the player who runs fast between the wickets has a distinct advantage and the success or failure often depends on the speed with which he is able to run between the wickets.

The pads that are currently in use have a greater ability to reduce the rebound distance of the ball after impact with the pads. This reduced ball-pad coefficient of restitution would be as a result of the structure and composition of the protective parts of the pads which are less rigid.However, the rebound characteristic of the pads could either be an advantage or a disadvantage when batting, depending on the type of match and the match situation. In Test cricket, where it is more common for fielders to field in close proximity to the batsman, pads with a large post impact rebound distance of the ball could result in the batsman being caught "bat-pad" (the ball deflected from the bat onto the pads and then being caught by a fielder). Wearing the more traditional pads, which showed smaller rebound distance, would reduce this rebound distance thus reducing the risk of dismissal in this manner. The converse could, however, apply in limited-overs cricket matches where the fielders are normally a distance from the batsman. The batsman wearing pads with a greater post-impact rebound distance may have an advantage by being able to score runs or leg-byes from balls deflecting off the pads.

A disadvantage of P1, although not tested experimentally, was that the ball striking the pad made a similar sound to that of a ball striking the bat and could result in a batsman being incorrectly adjudged caught by the wicketkeeper or fielder.

The manufacturers are, however, in the difficult position of having to balance the impact absorption and rebound characteristics of the pads with the comfort required to wear them for long periods at a time. Cricket pad manufacturers, aware that the batting pads behave differently under various impact conditions due to the structure and composition of the protective layers of the pads, need to further investigate the impact properties of the components or combinations of components they use in their batting pads. Only through continual research in the design and composition of the materials used in the manufacture
of batting pads, will the players gain maximum protection and comfort.

REFERENCES

1. *Temple R. Cricket Injuries: Fast Pitches change the Gentleman's Sport. Phys Sportsmed 1982:10(6):186-192.*
2. *Blonstein JL. Medical aspects of amateur boxing. Proc R Soc Med 1966;59:6499.*
3. *Stretch RA. Injuries to South African cricketers playing at first-class level. Sports Med 1989:4(I):3-20.*
4. *Stretch RA. The incidence and nature of injuries in first-league and provincial cricketers. S Afr Med J 1993;83(5):339-342.*
5. *Stretch RA. The seasonal incidence and nature of injuries in schoolboy cricketers. S Afr Med J 1995;85(11):1182-1184.*
6. *Jones NP, Tullo AB. Severe eye injuries in cricket. Br J Sports Med 1986;20(4):178-179.*
7. *Stretch RA, Tyler J. The force absobtion characteristics of cricket batting gloves at four impact velocities. S Afr J Sports Med 1995;2(3):22©29.*
8. *Hyrosomallis C. Shock absorption of cricket leg guards and batting gloves. Paper presented at Australian Conference of Science and Medicine in Sport, National Convention Centre,Canberra, Australia. 1996.*
9. *Payne WR, Hoy G, Laussen SP, Carlson JS. What research tells the Cricket Coach. Sports Coach 1987;10(4):17-22.*
10. *Abernethy B. Mechanism of skill in Cricket Batting. Aust J Sport 1981;13(1):3-10.*
11. *Penrose T, Foster D, Blanksby B. Release velocities of Fast Bowlers during a Cricket Test Match. Supplement to Aust J Health Phys Ed Rec. 1976:71:2-5.*
12. *Nigg BM. The validity and relevance of tests used for the assessment of sports surfaces. Med Sc Sport Exercise 1990:22(1):131-139.* □

RESEARCH ARTICLE

Comparison of oral and infrared auditory canal thermometry in sports participants

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ABSTRACT

Objective: To investigate whether recently developed infrared auditory canal thermometers, which offer numerous advantages over conventional oral glass-mercury thermometers, provide measurements equivalent to those of the oral glass-mercury thermometers before and after exercise.
Design: Open trial
Setting: Sporting arenas and gymnasiums.
Interventions: Oral (T_{oral}) and auditory canal temperatures (T_{ac}) were recorded in 45 adult persons participating in one of four sports; field hockey, squash, high-impact aerobics or swimming.
Results: T_{ac} correlated linearly with T_{oral} before and after exercise, but there was considerable variability between the two measures of body temperature, with upper and lower 95 % prediction intervals determined over the range of T_{ac} differing by at least 2 °C. Although we did not measure core body temperature, post-exercise measurements of T_{ac} and T_{oral} did not indicate the expected rise in body temperature, and both sites appear to underestimate blood temperature. In addition, the ear canal thermometer recorded anomalously low readings of post-exercise T_{oral} at the site of the swimming pool.
Conclusions: Our data indicate that the relationship between auditory canal temperature and oral temperature is variable and unpredictable in sports participants. Auditory canal thermometry is unlikely to offer an improvement on oral thermometry for the screening of body temperature in the sports arena.

Key words: body temperature; exercise; tympanic membrane temperature; hyperthermia

INTRODUCTION

Hyperthermia and hypothermia constitute serious health risks, both for the elite athlete and the recreational sportsperson. Management of the risk requires the measurement of core body temperature, which is a particularly formidable logistic problem when events can attract tens of thousands of participants. Numerous techniques have been used, clinically and in the laboratory, to monitor body temperature. The general thermal status of the body is best represented by the temperature of mixed venous blood in the right ventricle or pulmonary artery.[3,16] However, temperature measurement at this site necessarily is invasive and usually is carried out only in critically ill patients. Other estimates of body core temperature trade off accuracy against practical considerations of convenience and compliance. In the sports arena, especially when there are multiple participants, one is constrained to use measures, at least for the primary screening of body core temperature, which forfeit accuracy for convenience. Even though it is documented that sublingual oral temperature (T_{oral}) is not a good index of blood temperature,[21] it is the measure of body temperature most commonly used in sports participants; it is used because it is convenient and socially acceptable. However, measurement of T_{oral} requires the voluntary co-operation of the subject and careful probe placement.[7] Glass-mercury thermometers are associated with risks of mercury spillage and cross-infection, and are slow, requiring at least three minutes of closed-mouth measurement and providing maximum accuracy after about eight minutes.[12] Furthermore, the accuracy of the reading, which is not good at best, can be influenced adversely by the ingestion of liquids, gum chewing, and open mouth breathing.[7,12,23]

Recently, "tympanic membrane" thermometers have become available commercially, at relatively low cost, and are being used increasingly in hospitals. These instruments record infrared radiation emitted from the tympanic membrane or, more usually, the terminal auditory canal, and offer several practical advantages over other devices used for body temperature measurement. The thermometers are non-invasive, easy to use, and provide a digital display of temperature in a few seconds.[22] Unlike oral thermometers, auditory canal thermometers are not influenced by recent liquid ingestion or inadvertent mouth opening,[23] and there is no discomfort or risk of infection for subjects. The usefulness of these thermometers has been demonstrated in pediatric patients, intensive care unit patients, nursing home residents, outpatients, and research laboratories.[6,22] The potential of the infrared auditory canal thermometer to simplify and shorten the process of temperature measurement makes it an appealing device for use in sports participants. If it is to be a viable sports thermometer, it should be no less accurate than the conventional oral thermometer. As with the oral thermometer, the auditory canal thermometer cannot be used to monitor the temperature of a person with heat illness, where a better measure of body core temperature is required; its role might be to screen for those sports participants who require more intensive monitoring.

The purpose of our study was to determine whether the infrared auditory canal thermometer provides measurements equivalent to those of the oral glass-mercury thermometer before and after exercise. We compared auditory canal and oral temperature measurements in adults participating in a variety of sports.

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METHODS

T_{oral} and auditory canal temperatures (T_{ac}) were recorded in subjects participating in four sports, at different times of day; field hockey, squash, high-impact aerobics, and swimming. In total, 45 healthy subjects volunteered, and their ages are shown in Table I. The protocol for our study was approved by the Committee for Research on Human Subjects, University of the Witwatersrand.

TABLE: I Subject ages

Sport	Age (yr.) Mean ± SD	Range
Hockey (n = 11)	20.5 ± 1.4	18 - 23
Squash (n = 10)	21.0 ± 1.2	19 - 23
Aerobics (n = 12)	22.8 ± 2.8	20 - 30
Swimming (n = 12)	22.9 ± 8.2	18 - 48

We measured T_{oral} using a glass-mercury thermometer placed in the subject's posterior sublingual pocket for three minutes. During this period, T_{ac} was recorded using an infrared thermometer, the Genius FirstTemp Model 3000A (Intelligent Medical Systems, Carlsbad, CA). Auditory canal temperatures were recorded in both ears, first in the right ear and immediately thereafter in the left ear. Temperatures were measured according to the manufacturer's instructions; the subject's head was gently restrained while the probe tip was inserted far enough into the ear canal to seal the tympanum from ambient air. To minimise operator variability, T_{ac} was measured by a single investigator. The subjects rested at ambient temperature, at the site of subsequent activity, for at least five minutes before we recorded rest temperatures. Post-exercise temperatures were recorded within two minutes after completion of exercise. Swimmers did not towel-dry themselves until both T_{ac} and T_{oral} had been measured; their ear canals were, however, dried before measurements of T_{ac} were taken. The duration of exercise for each sport (excluding warm-up time) was 70 minutes for hockey, 50 minutes for squash, 60 minutes for aerobics and 25 minutes for swimming. Post-exercise temperatures were not obtained from three subjects for technical reasons.

The glass-mercury thermometers and the auditory canal thermometer were calibrated after completion of the study, against a high accuracy quartz thermometer (Quat 100/200, Heraeus, Hanau, Germany). The oral thermometers were immersed in an insulated water bath set at a variety of controlled temperatures. The auditory canal thermometer was pointed at an infrared black body, constructed as a matt black re-entrant cone set up in the water bath. When calibrated, both the auditory canal thermometer and the oral thermometers had an accuracy of better than 0.1 °C over their measurement ranges.

Dry and wet-bulb ambient temperatures were measured at 10-minute intervals at each sports site using a sling psychrometer. A psychrometric chart was used to calculate vapour pressure and relative humidity. Environmental conditions at each site are shown in Table II. The heated swimming pool was located indoors in a health club complex, and pool temperature was about 25 °C.

TABLE II: Environmental conditions (mean ± SD, n = 4 to 8)

Sport	T_{db} (°C)	T_{wb} (°C)	RH (%)	VP (kPa)
Hockey	21.1 ± 1.1	12.1 ± 1.0	36 ± 4	0.9 ± 0.1
Squash	21.0 ± 0.7	13.8 ± 0.4	46 ± 6	1.2 ± 0.1
Aerobics	20.1 ± 0.5	13.7 ± 0.3	51 ± 2	1.2 ± 0.0
Swimming	18.0 ± 0.1	15.0 ± 0.1	74 ± 1	1.5 ± 0.1

T_{db} = dry bulb temperature; T_{wb} = wet bulb temperature; RH = relative humidity; VP = vapour pressure.

The relationship between T_{ac} and T_{oral} was evaluated using Pearson product-moment correlation analyses and linear regression. Values of $p < 0.05$ were considered significant.

RESULTS

Left T_{ac} was significantly correlated with right T_{ac} at rest before exercise ($r = 0.64$, $P < 0.0001$, $n = 45$; left T_{ac} = 12.7 + 0.64 right T_{ac}; right T_{ac} = 13.2 + 0.63 left T_{ac}), and mean left T_{ac} (35.9 ± 0.5 °C) and mean right T_{ac} (35.9 ± 0.5 °C) were the same. Furthermore, the slope of a regression line forced through the origin was not significantly different from one (Figure 1), so there was no particular bias for one side of the head. However, only about 40 % of the variability in the auditory canal temperature of one ear was associated with variability in the other ear. Measures of contralateral T_{ac} were not equivalent; the mean absolute difference between left T_{ac} and right T_{ac} was 0.3 ± 0.3 °C, and exceeded 1 °C for individual subjects. T_{ac} , in all further analyses, therefore was expressed as the mean of left T_{ac} and right T_{ac}.

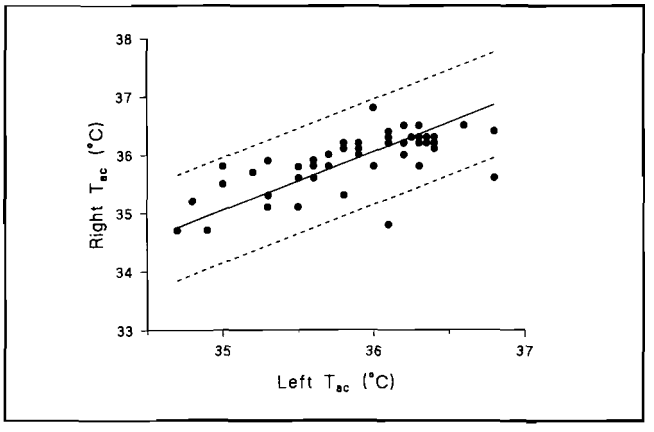

Figure 1: Left and right auditory canal temperatures in the same subjects, at rest, before participation in sports events. The linear regression line (solid line) and 95 % prediction intervals (dashed lines) of an equation constrained to go through the origin are shown. (Right T_{ac} = (1.00 ± 0.01) x left T_{ac}, $r = 0.52$, $P < 0.0001$, $n = 45$).

Mean oral and auditory canal temperatures recorded for participants in each sport, both at rest and post-exercise, are shown in Table III. Mean oral temperatures were typical of those usually recorded in young adults, in Johannesburg. At rest, T_{ac} and T_{oral} were significantly correlated (Figure 2), with T_{oral} somewhat

higher than T_{ac} at the lower temperatures, and somewhat lower at the high temperatures. However, the upper and lower 95% prediction intervals differed by 2 °C (Figure 2), implying that at any T_{ac}, T_{oral} could be substantially higher or lower than T_{ac} in individual subjects.

TABLE III: Oral (T_{oral}) and auditory canal (T_{ac}) temperatures of subjects recorded before and after exercise (mean ± SD)

	T_{oral} (°C)		T_{ac} (°C)	
Sport	Rest	Post-exercise	Rest	Post-exercise
Hockey (n = 10)	36.2 ± 0.4	35.5 ± 0.5	36.0 ± 0.3	35.6 ± 0.5
Squash (n = 10)	36.1 ± 0.5	35.8 ± 0.7	35.8 ± 0.6	35.3 ± 0.9
Aerobics (n = 11)	35.8 ± 0.6	36.2 ± 0.6	35.7 ± 0.6	36.0 ± 0.5
Swimming (n = 11)	35.9 ± 0.7	36.2 ± 0.9	36.0 ± 0.5	33.6 ± 0.7

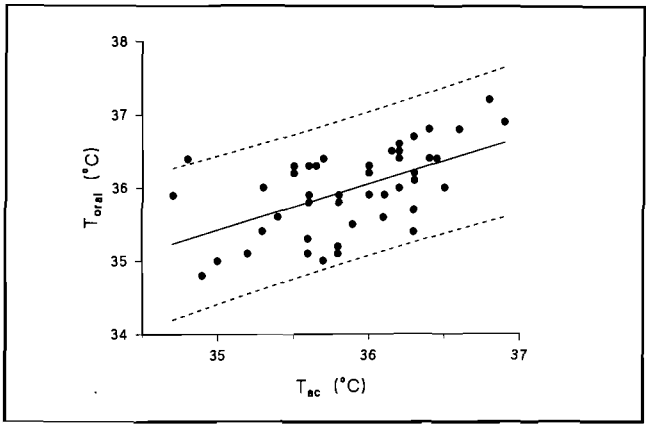

Figure 2: Oral temperature (T_{oral}) as a function of auditory canal temperature (T_{ac}, mean of both canals) in subjects at rest before exercise. The linear regression line and 95 % prediction intervals are shown. (T_{oral} = 0.63 T_{ac} + 13.3, r = 0.56, P < 0.001, n = 45).

Post-exercise temperatures for hockey, squash and aerobics participants are shown in Figure 3. Both T_{ac} and T_{oral} tended to drop during the sports events. T_{ac} and T_{oral}, for all three sports considered together, were still significantly correlated after the events. As with rest temperatures, variability between T_{ac} and T_{oral} of individuals was considerable, with the upper and lower 95 % prediction intervals differing by between 2.2 and 2.4 °C over the range of measured temperatures (Figure 3).

The mean T_{ac} measured after swimming was very low (33.6 ± 0.7 °C), so the temperatures after swimming were analysed separately. Measurements of T_{ac} and T_{oral} were significantly correlated (Figure 4). However, the mean absolute difference between T_{ac} and T_{oral} was 2.6 ± 0.6 °C, and the 95 % prediction intervals differed by between 2.9 and 3.3 °C over the range of measured T_{ac}. The greatest discrepancy between T_{ac} and T_{oral} was therefore evident in swimmers. We considered the discrepancy too great to be the result simply of an inherent difference between the measurement sites, and so measured T_{ac} in two observers, who had been at the poolside but not in the water; in both cases, T_{ac} was recorded as less than 34 °C.

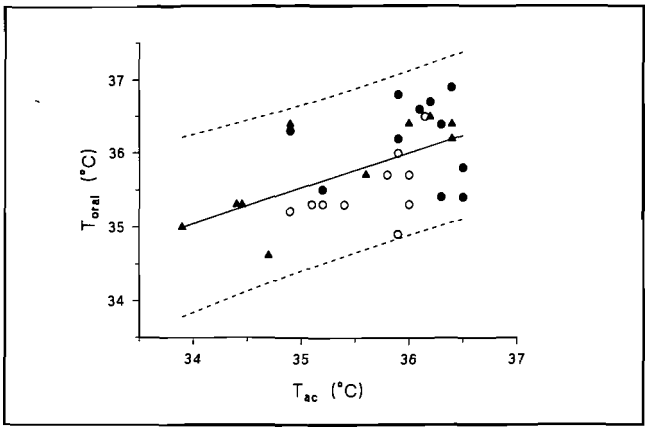

Figure 3: Oral temperature (T_{oral}) as a function of auditory canal temperature (T_{ac}, mean of both canals), after exercise in hockey players (open circles, n = 10), squash players (open triangles, n = 10), and aerobics participants (closed circles, n = 11). The linear regression line and 95 % prediction intervals are shown. (T_{oral} = 0.48 T_{ac} + 18.8, r = 0.54, P < 0.005, n = 31).

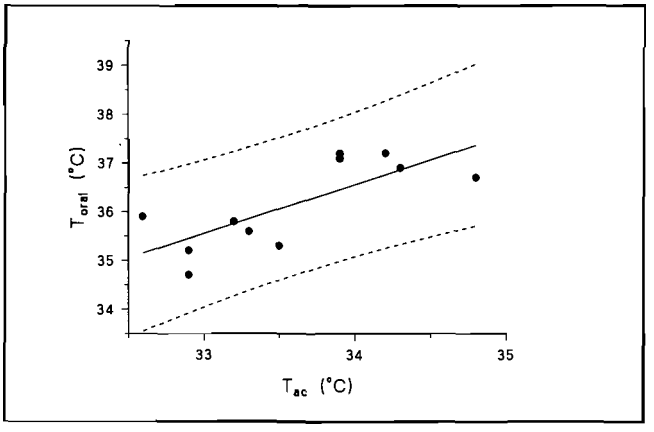

Figure 4: Oral temperature (T_{oral}) as a function of auditory canal temperature (T_{ac}, mean of both canals), after swimming. The linear regression line and 95 % prediction intervals are shown. (T_{oral} = 1.01 T_{ac} + 2.4, r = 0.76, P < 0.01, n = 11).

DISCUSSION

The main aim of our study was to determine whether temperatures indicated by an auditory canal thermometer bore a defined relationship to those indicated by an oral thermometer, when the measurements were made simultaneously in sports participants. If that had been the case, the speed and convenience of the auditory canal thermometer would make it an attractive instrument for screening participants in events which impose a risk of hyperthermia or hypothermia, replacing the oral thermometer, but not replacing the rectal, oesophageal or blood thermome-

ter necessary for monitoring the body core temperature of those with signs or symptoms of heat or cold illness. Our results indicate that the relationship between auditory canal temperature and oral temperature is variable and unpredictable in sports participants, before and after their events. Indeed, the results lead us to doubt whether either thermometer can be used safely to screen sports participants.

With neither thermometer does the problem lie with an intrinsic inaccuracy of the measuring instrument. When calibrated against a quartz thermometer, by water immersion in the case of the oral thermometer and using a re-entrant cone blackbody in the case of the infrared detector in the auditory canal thermometer, both thermometers achieved an intrinsic accuracy of 0.1 °C over the range of measured temperatures, better than necessary for physiological screening of body temperature. In the case of one sport, namely swimming, the main problem with the auditory canal thermometer resulted from its failure to record temperatures properly in the field. For all sports, a second problem arose, namely physiological variability and inconsistency between the temperatures indicated by the oral and auditory canal thermometers.

We detected the poolside problem with the auditory canal thermometer through the anomalously low readings of post-exercise T_{ac} recorded at the site of the swimming pool (Table III). We believe that the infrared thermometer was adversely affected by water, either in the air or condensing, as a result of the high relative humidity (74 %), or because of direct wetting (despite swimmers drying their ear canals). If water indeed caused the malfunction, sweat accumulation in the auditory canals of subjects may also affect the functioning of the infrared thermometer. We recorded auditory canal temperatures of less than 34.5 °C in three squash players who had sweated profusely (Figure 3). We do not know whether the problem is peculiar to the make of the thermometer we used, or whether it is a general problem of all infrared auditory canal thermometers. If it is a general problem, it is a serious deficiency; it not only would exclude the use of the thermometers for water sports (where hypothermia is the thermal risk), but also exclude them from use in environments where humidity contributes to the risk of hyperthermia. That does not mean only hot humid environments; several football deaths have been reported when T_a was below 24 °C, but the relative humidity exceeded 95 %.[14]

One of the physiological problems we encountered with the auditory canal thermometer was lack of bilateral symmetry between the temperatures of the two ear canals. Several papers describing the clinical use of auditory canal thermometers have claimed that the canals do have the same temperature, even in the presence of unilateral ear disease.[10,11,24] It may be that, in the clinical setting, the mean absolute difference of 0.3 °C between the two ear canals, which we observed, is considered tolerable. The criterion sometimes used to conclude that there is bilateral symmetry, namely that in a large group of subjects the mean difference between the two canal temperatures is not significantly different from zero, clearly is an inappropriate use of statistics, and reflects only variability between the subjects.

We decided to use the average temperature of the two auditory canals. The difference between that average temperature and oral temperature measured simultaneously was much greater than could be accounted for by the intrinsic inaccuracy of the thermometers. Despite similarities in mean temperatures (Table III) and statistical correlation between T_{ac} and T_{oral} (Figure 2, 3 and 4), the oral glass-mercury and infrared auditory canal thermometers yielded temperatures which were not interchangeable. In resting subjects, only 31 % of the variance in T_{ac} was associated with the variance in T_{oral}. Furthermore, for any particular value of T_{ac}, T_{oral} of an individual could be more than 1 °C above or below the mean expected value for the group (Figure 2). During exercise there are changes in heat balance at the head, for example those resulting from open-mouth breathing and scalp sweating, and one would expect the relationship between T_{ac} and T_{oral} to be even more variable than at rest. Indeed, we found greater inconsistency in the relationship between T_{ac} and T_{oral} after exercise. For example, at T_{ac} just below 36 °C, oral temperature was below 35 °C in one hockey player and almost 37 °C in one aerobics participant. We therefore have to conclude that there is no fixed relationship between auditory canal temperature and oral temperature in sports participants, at least for the sports and conditions we investigated.

The absence of a relationship between auditory canal temperature and oral temperature could arise because either one, or both, of the sites at which the thermometers measure temperature have a temperature not consistently related to blood temperature. Though we did not measure core body temperature, it is likely that neither site reflected blood temperature properly, since neither site demonstrated the expected rise in temperature associated with increased metabolic heat production during exercise (Table III).

Although it continues to be used in the sports arena, oral thermometry has been known, for at least thirty years, to provide an unreliable index of blood temperature during exercise.[21] Measurements of T_{oral} are lower than temperatures at deeper sites, in an inconsistent way, and are affected by both breathing patterns and ambient air temperature.[12,16] Head skin temperature also has a strong influence on oral temperatures,[15] and low skin temperature could have contributed to low oral temperature in the swimmers and when there was free evaporative cooling from the head. Infrared auditory canal thermometers are much newer devices, and experience with them outside the hospital setting is limited.[20,22] They are often referred to as infrared tympanic thermometers; the temperature at one particular site of the tympanum is thought by some to reflect the temperature of blood perfusing the brain.[1,2,4] However, the Genius thermometer which we used does not have the resolution necessary to measure temperature of the tympanic membrane only, and detects infrared radiation emanating from both the ear drum and the surrounding tissue, and the same is true for other makes of infrared thermometer.[6,8] Auditory canal temperature is lower than tympanic membrane temperature (T_{ty}).[20] True tympanic temperature can be measured by placing either a thermistor or a thermocouple in direct contact with the ear drum. Positioning of such a thermometer is a thoroughly unpleasant experience for subjects,[2,3] and contact tympanic thermometry cannot be used in the sports arena.

The fact that T_{ac} may underestimate body core temperature would not prevent its use, as a field thermometer, if the relationship between T_{ac} and blood temperature was fixed, in sports participants, and uncontaminated by other variables. However, that is not the case. In particular, auditory canal temperatures are influenced by head skin temperature.[3,13,15,17] Heat

loss from the human head constitutes a major component of total heat loss during exercise, particularly when there is additional convective cooling in sweating subjects;[3,19] fanning the face of hyperthermic subjects reduces T_{ty}.[5,18] The effect of head skin temperature on Tac arises not primarily from the pinna, but from the scalp directly above and behind the ear.[13] Hockey players, unlike the other sports participants in our study, exercised outdoors in windy conditions. The air movement, arising from ambient wind and the players' motion, would have induced convective and evaporative heat loss from scalps which may be the reason that post-exercise T_{ac} was lower than T_{ac} at rest.

In our view, therefore, auditory canal thermometry is unlikely to offer an improvement on oral thermometry for the screening of sports participants for hyperthermia or hypothermia, and we reaffirm that oral thermometry provides an inconsistent underestimate of blood temperature after sport. It remains remotely possible that T_{ac} does have some recognizable relationship with blood temperature in sports participants; the public nature of our study prevented us measuring rectal temperature (T_{rc}). Correlation of T_{ac} with T_{rc} in a variety of sports participants still needs to be done. In one recent study,[24] there was poor agreement between measurements of T_{ac} and T_{rc} in athletes with suspected exertion-induced heat exhaustion. T_{ac} on admission to the field medical centre was on average 1.2 °C lower than T_{rc}. In addition, statistical variability between the two measures of body temperature was such that measurements of T_{ac} could not be used confidently to predict a rectal temperature greater than 38 °C. The investigators concluded that the use of infrared tympanic membrane thermometry can result in misdiagnosis of heat exhaustion.[9] Therefore, it seems unlikely that there will be a recognizable relationship between T_{ac} and T_{rc} in sports participants.

ACKNOWLEDGEMENTS

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REFERENCES

1. *Benzinger TH. On physical heat regulation and the sense of temperature in man. Proc Nat Acad Sci USA 1959: 45: 645-659.*
2. *Benzinger TH, Taylor GW Cranial measurements of internal temperature in man. In: Hardy JD ed. Temperature: Its Measurement and Control in Science and Industry. New York: Reinhold Publishing Corp., 1963: 111-120.*
3. *Brengelmann GL. Dilemma of body temperature measurement. In: Shiraki K, Yousef MK eds. Man in Stressful Environments. Springfield: Thomas, 1987: 5-22.*
4. *Brinnel H, Cabanac M. Hyperthermia and human brain cooling. In: Shiraki K. Yousef MK eds. Man in Stressful Environments. Springfield: Thomas, 1987: 87-97.*
5. *Brinnel H, Cabanac M. Tympanic temperature is a core temperature in humans. J Therm Biol 1989: 14: 47-53.*
6. *Chamberlain JM, Terndrup TE, Alexander DT, Silverstone FA, Wolf-Klein G, O'Donnell R, Grandner J. Determination of normal ear temperature with an infrared emission detection thermometer. Ann Emerg Med 1995: 25:15-20.*
7. *Erickson R. Oral temperature differences in relation to thermometer and technique. Nurs Res 1980: 29: 157-164.*
8. *Fraden J, Lackey RP. Estimation of body sites temperatures from tympanic measurements. Clin Pediatr 1991: 30 (Suppl): 65-70.*
9. *Hansen RD, Olds TS, Richards DA, Richards CR, Leelarthaepin B. Infrared thermometry in the diagnosis and treatment of heat exhaustion. Int J Sports Med 1996: 17: 66-70.*
10. *Kelly B, Alexander D. Effect of otitis media on infrared tympanic thermometry. Clin Pediatr 1991: 30 (Suppl.):46-48.*
11. *Kenney RD, Fortenberry JD, Surratt SS, Ribbeck BM, Thomas WJ. Evaluation of an infrared tympanic membrane thermometer in pediatric patients. Pediatrics 1990: 85: 854-858.*
12. *Mairaux P, Sagot JC, Candas V. Oral temperature as an index of core temperature during heat transients. Eur J Appl Physiol 1983: 50: 331-341.*
13. *Marcus P. Some effects of cooling and heating areas of the head and neck on body temperature measurement at the ear. Aerospace Med 1973: 44: 397-402.*
14. *McArdle WD, Katch FI, Katch, VL. Exercise Physiology: Energy, Nutrition and Human Performance. 3rd Edition, Philadelphia: Lea and Febiger, 1991: 569.*
15. *McCaffrey TV, McCook RD, Wurster RD. Effect of head skin temperature on tympanic and oral temperature in man. J Appl Physiol 1975: 39: 114-118.*
16. *Mitchell D, Laburn HP. Pathophysiology of temperature regulation. Physiologist 1985: 28: 507-517.*
17. *Nadel ER, Horvath SM. Comparison of tympanic membrane and deep body temperatures in man. Life Sci 1970: 9: 869-875.*
18. *Nielsen B. Natural cooling of the brain during outdoor bicycling? Pflugers Archives 1988: 411: 456-461.*
19. *Rasch W, Samson P, Cote J, Cabanac M. Heat loss from the human head during exercise. J Appl Physiol 1991: 71: 590-595.*
20. *Shenep JL, Adair JR, Hughes WT, Roberson PK, Flynn PM, Brodkey TO, Fullen GH, Kennedy WT, Oakes LL, Marina NM. Infrared, thermistor, and glass-mercury thermometry for measurement of body temperature in children with cancer. Clin Pediatr 1991: 30 (Suppl.): 36-41.*
21. *Strydom NB, Wyndham CH, Williams CG, Morrison JF, Bredell GAG, Joffe A. Oral/rectal temperature differences during work and heat stress. J Appl Physiol 1965: 20: 283-287.*
22. *Terndrup TE. An appraisal of temperature assessment by infrared emission detection tympanic thermometry. Ann Emerg Med 1992: 21: 1483-1492.*
23. *Terndrup TE, Allegra JR, Kealy JAA. Comparison of oral, rectal, and tympanic membrane-derived temperature changes after ingestion of liquids and smoking. Am J Emerg Med 1989: 7: 150-154.*
24. *Terndrup TE, Wong A. Influence of otitis media on the correlation between rectal and auditory canal temperatures. AJDC 1991: 145: 75-78.* □

Brief report: Urinary catecholamine excretion during outdoor sport rockclimbing

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ABSTRACT

Objectives: The purpose of this study was to examine the response of urinary excretion of catecholamines after a bout of outdoor sport rockclimbing.
Design, setting & subjects: This study was undertaken outdoors in the Blue Mountains in NSW, Australia. Seven elite rockclimbers were recruited for the study. Each had previous experience with the climb route. The subjects were required to climb the route as fast as possible. Heart rate (HR) and urinary excretion of catecholamines (epinephrine, EPI; norepinephrine, NE) were measured pre-climb, immediately post-climb and 30 min post-climb.
Results & conclusions: The duration of the climb was ≈7 min, 36 s. HR increased from 74 bpm (pre-climb) to 157 bpm during the climb. The rise in catecholamine excretion was not significant compared to pre-climb samples at either of the sample times. It is concluded that training induced adaptations to this type of exercise coupled with the methodological constraints of the study are responsible for the non-significant increases in catecholamine excretion.

INTRODUCTION

Rockclimbing has increased in popularity in recent years. The follow-on has seen improvement in climbing standards and competitions.[1] However, unlike other popular sports there are not much scientific data on rockclimbers and rockclimbing with most of the available data related to typical climbing injuries.[1-3] More recently, however, anthropometric data has become available.[4,5] Data on the physiological responses and requirements of rockclimbing is somewhat scarce with inferences mainly being drawn from other related activities such as mountaineering and gymnastics. However, sport rockclimbing is unlike the more studied sports with the exercise itself characterised by isometric muscle contractions and gymnastic type movements. Furthermore, the sport may be considered 'high risk' which adds a stressful component to the physiological demands.

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Blood and urine catecholamine concentrations have been typically used as an index of sympathetic activity and have been shown to differ between trained and untrained subjects.[6] Because rockclimbing activity relies heavily on static muscular contractions, increased sympathetic activity would be expected.[7] Therefore, it would be of significant interest to evaluate the sympathetic drive elicited by a bout of outdoor rockclimbing. Furthermore, the common catecholamine response exhibited after similar or 'high risk' type activity such as rockclimbing is an increased epinephrine (EPI) and norepinephrine (NE) response.[8] For example, the urinary levels of EPI and NE in parachutist trainees were found to increase for initial jumps and then subsequently decline over the training period for both pre-jump and post-jump values. This decrease in EPI and NE is indicative of a reduced adrenomedulary discharge as a consequence of familiarisation to a previously stressful event.

This type of physiological response has yet to be studied in trained sport rockclimbers. Therefore, the aim of this study was to examine the response of urinary excretion of catecholamines after a bout of outdoor sport rockclimbing.

METHODS

Subjects: Seven competitive rockclimbers (6 male, 1 female) were recruited for this study. The mean climbing experience of the group was 8.9 ± 1.2 years with the most difficult outdoor ascent made without preview or fall ranging from 6b - 7a (UK grading system). All subjects were in good health as determined by a medical and health questiomaire. The study was approved by the University Ethics in Human Research Committee. The mean (± SE) age, height, mass and sum of nine skinfolds were 25 ± 1 years, 175.7 ± 2.7 cm, 62.6 ± 3.3 kg, and 61.3 ± 4.2 mm, respectively.

Climbing protocol: The climb was graded 5c and was conducted on a rockface in the Blue Mountains of New South Wales, Australia. All subjects had previous experience with the climb on at least three other occasions. The length of the climb was 24.4 m and overhanging by ≈ 4m throughout at an elevation of 890 m. On one occasion approximately 5 - 7 d prior to the climb a resting sample of urine was collected after subjects abstained from alcohol, caffeine ingestion and vigorous exercise for a minimum period of 24 h. The reasons for this were that the route to the climb was lengthy with subjects required to walk for approximately 20 min and in some instances the terrain physically demanding. Hence, a pre-climb sample at the climb site would have influenced the outcome of the urine concentration of catecholamines. Once arrived at the climb site subjects rested for at least 30 min before micturition. At

the climb site the subjects were briefed as to the conditions of the climbing protocol and prepared for climbing with a waist harness secured to a 10 mm dynamic rope fed through a ring bolt at the top of the rockface and to a belayer at the start of the climb. The briefing and preparation was approximately 20 - 30 min duration. Subjects were required to climb the route as quickly as possible. On completion of the climb each subject was lowered to the ground and given a specimen bottle for collection of urine. The specimen bottle and urine sample were returned within (5 min of completing the climb. On returning the urine sample the subjects were given a bottle containing 350 ml of water to ingest over a period of 15-20 min before giving another urine sample 30 min post - climb. During the climb heart rate (HR) was monitored continuously with a Sports Tester (Polar-Electro, Oy., Finland).

Catecholamine determination: The urine samples were collected in specimen bottles and acidified to pH 2-3 with 15 ml of hydrochloric acid. Immediately after collection the samples were stored on ice and subsequently frozen (-20°C) until analysis. All samples were analysed in the same run to avoid inter-assay variation. The method of determination was by high performance liquid chromatography according to Pillai.[9] This method requires less urine (0.5 ml vs. 4 ml) and is quicker with adjustment of pH facilitated by a visual indicator.

Statistics: The data were analysed by ANOVA with repeated measures on time for HR and for urinary catecholamine concentrations (pre-climb æ post-climb æ 30 min post-climb). When significant main effects were found Tukey's HSD *post-hoc* procedure was employed to locate the source of significant difference. Significance was accepted when $P < 0.05$. All values are expressed as means ± SE.

RESULTS

Climb duration and HR: The time taken for the climb was 7 min, 36 s (range 6 min 28 s - 9 min 54 s). Pre-climb HR was 74 ± 5 bpm and increased to 145 ± 10 bpm ($P < 0.05$) and 157 ± 8 bpm after 1 and 5 min of climbing, respectively. HR peaked at 83 bpm above resting values.

Urinary catecholamine concentrations: The results of the catecholamine analysis are shown in Figure 1. Resting urinary concentrations of EPI, NE and total catecholamines (CA_{TOT}) were 89.1 ± 20.1, 315.5 ± 78.8, 404.6 ± 98.3 nmoles.L^{-1}, respectively. The rise in urinary excretion of EPI was not significant ($P = 0.26$) with levels reaching 118.5 ± 21.0 nmoles.L^{-1} and 180.5 ± 73.3 nmoles.L^{-1} immediately post-climb and 30 min post-climb, respectively. A similar non significant ($P = 0.35$) finding for the urinary excretion of NE was observed with levels reaching 304.6 ± 42.6 nmoles.L^{-1} immediately post-climb and rising to 731.4 ± 430.3 nmoles.L^{-1} 30 min post-climb. CA_{TOT} were not significantly changed ($P = 0.34$) with values rising to 423.2 ± 60.1 nmoles.L^{-1} immediately post-climb and 911.8 ± 501.8 nmoles.L^{-1} (range 195 - 4407 nmoles.L^{-1}) 30 min post-climb.

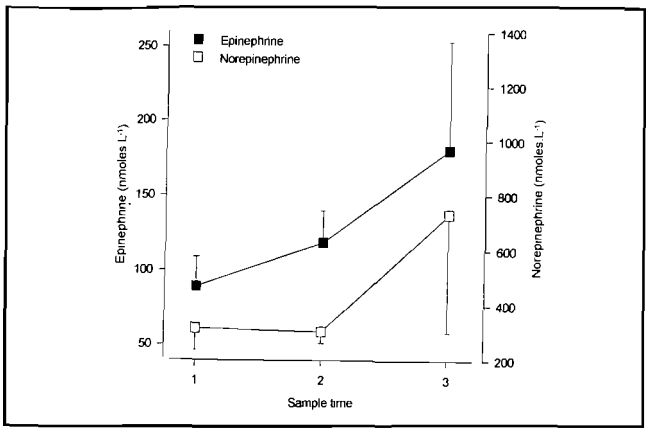

Figure 1: *Sympathetic activation as measured by catecholamine excretion in trained sport rockclimbers during outdoor climbing. Sample times are at pre-climb (1), immediately post-climb (2) and, 30 min post-climb (3).*

DISCUSSION

This is the first study to examine the sympathetic nervous system response in elite rockclimbers while climbing in the field. The levels of NE and EPI excretion observed in the pre-climb urine sample were within the normal range (EPI < 101 nmoles.L^{-1} ; NE < 701 nmoles.L^{-1}) as reported by the pathology laboratory (Barratt & Smith, Pathology, Orange, NSW, Australia). Furthermore, NE concentration was (3.5 times that of EPI and is considered normal.[10] Therefore, it is reasonable to assume that the sampling and determination procedures were satisfactory for this type of field experiment. Although urinary excretion of catecholamines is not ordinarily regarded as an accurate measure of sympathoadrenal activity,[11] the geographical setting and isolation of the investigation prevented the use of serial venous blood sampling.

Even though there was a tendency for CA_{TOT} concentration to increase after the climb, the post-climb sample was not statistically different compared to the pre-climb sample. The large SE associated with the 30 min post-climb CAT_{TOT} is most likely related to the large range of values recorded for both EPI and NE and the fact that one subject in particular responded with very high values for both amines. However, a very distinct pattern did emerge from the immediate post-climb sample to the 30 min post-climb sample for both amines. EPI excretion showed a more linear rise at all three sample times, whereas NE only showed an increase for the 30 min post-climb sample. This increase in post-climb NE can be partly explained by the delayed sympathetic spill from neuronal sites into the circulation and eventually into the urine.[8]

However, given that there was no statistical significance between samples at the various measurement times, it is difficult to speculate as to the role that the sympathetic nervous system may have played during the climb. Nonetheless, several points can be raised with respect to the present findings. For instance, previous work has shown increases in plasma catecholamine concentration when isometric handgrip exercise is performed at levels above 20% of maximum voluntary contraction (MVC).[7] Although MVC was not measured in this study, it would be reasonable to assume that subjects performed isometric exercise

above 20% MVC given that they were required to lift their own body weight and that the climb was overhanging by ≈ 4 m thereby increasing the negative effects of gravity. Furthermore, recent evidence indicates that trained climbers are able to generate significantly higher forces on grip strength tests compared to either recreational or non-climbers.[12]

Although, rockclimbing is characterised by intermittent isometric muscle contractions, some dynamic muscle work is also performed. An elevated HR of at least 30 bpm during short-term dynamic exercise is required to stimulate a rise in plasma catecholamine concentration.[13] In this study, HR increased by ≈ 83 bpm above the pre-climb value. Hence, a rise in catecholamine concentration would be expected. However, even at high work rates EPI levels are not always elevated compared to NE.[14] This might partly account for the non-significant increase in urinary concentrations of catecholamines observed in this study.

Exercise intensity is also a determinant of sympathetic stimulation. Previous work suggests that at least a 30% increase in VO_2 must be elicited for an increase in catecholamine concentration.[11] A recent investigation has shown that rockclimbers use less than 50% of VO_{2max} during a standard indoor climb.[15] However, work in our laboratory indicates that rockclimbers use up to 75% of VO_{2max} (≈ 44 $ml.kg.min^{-1}$) if the VO_{2max} is expressed as a function of graded climbing (unpublished observations). These data indicate that rockclimbing can elicit an exercise intensity above that which is normally required to stimulate sympathetic activity. Moreover, a given VO_2 elicited by dynamic arm work increases catecholamine concentrations to a higher level than if the same VO_2 is elicited by leg work.[16,17] Although the present findings are not statistically significant, the pattern of urinary catecholamine excretion seem to support previous findings.

There are at least three possible explanations why the rise in urinary catecholamine concentrations failed to reach statistical significance. First, plasma concentrations of catecholamines have been shown to be attenuated during acute exercise following training.[6,18,19] This sympathetic adaptation has also been observed in subjects with physically trained arms whilst performing handgrip exercise.[20,21] This might partly explain the blunt catecholamine response in this study. Second, the results support the notion that trained individuals do not necessarily perceive the activity as 'high risk' as less trained individuals who exhibit signs of subjective fear.[8] Also, the methodological features of the study may have contributed to a reduced sympathetic response than would have otherwise been expected. For instance, the climbers were secured to a top rope rather than lead climb and all climbers had previous experience on the route, all of which may have attenuated the subjective fear.

In summary, elite sport rockclimbers do not exhibit significantly elevated catecholamine concentrations as measured in urine after a bout of moderatley difficult outdoor climbing. This may be due to training induced adaptations to the exercise and the methodological constraints of the investigation. It is suggested that further field work be undertaken to confirm these findings so that physiological responses to such a highly skilled activity be quantified and used in order to provide a practical approach to training and preparation for competition and that the stresses related to this activity be understood.

REFERENCES

1. *Bollen SR. Soft tissue injury in extreme rockclimbers. Br J Sp Med 1988; 22: 145-147.*
2. *Bollen SR, Gunson CK. Hand injuries in competition climbers. Br J Sp Med 1990; 24: 16-18.*
3. *Holtzhausen LM, & Noakes TD. Elbow, forearm, wrist, and hand injuries among sport rockclimbers. Clin J Sport Med 1996; 6: 196-203.*
4. *Watts PB, Martin DT, & Durtschi S. Anthropometric profiles of elite male and female competitive sport rock climbers. J Sports Sc 1993; 11:113-117.*
5. *Holtzhausen LM, Schwellnus MP, & Noakes TD. Anthropometric and muscle strength measurements of competitive South African sport rockclimbers. Sth Af J Sports Med 1996; 4:13-18.*
6. *Mazzeo RS. Catecholamine responses to acute and chronic exercise. Med Sci Sports Exerc 1991; 7: 839-845.*
7. *Seals DR, Chase PB, Taylor JA. Autonomic mediation of the pressor response to isometric exercise in humans. J Appl Physiol 1988; 64: 2190-2196.*
8. *Hansen JR, Stoa KF, Blix AS, & Ursin H. Urinary levels of epinephrine and norepinephrine in parachutist trainees. In: Ursin H, Badde E, & Levine S eds. Psychobiology of stress: A study of coping men. New York: Academic Press, 1978, 63-74.*
9. *Pillai DN. Inexpensive alternative to kit methods for extraction of urinary catecholamines. Clin Chem, 1987; 33: 11.*
10. *Karki NT. The urinary excretion of noreadrenaline and adrenaline in different age groups, its diurnal variations and the effect of muscular work on it. Acta Physiol Scand 1956; 39: 132 (suppl).*
11. *Galbo, H. Hormonal and metabolic adaptation to exercise. Thieme-Stratton Inc, 1983: 7-8.*
12. *Grant S, Hynes V, Whittaker A, Aitchison T. Anthropometric, strength, endurance and flexibility characteristics of elite and recreational climbers. J Sports Sc 1996; 14: 301-309.*
13. *Christensen NJ, Brandsborg O. The relationship between plasma catecholamine concentration and pulse rate during exercise and standing. Eur J Clin Invest 1973; 3: 299-306.*
14. *Galbo H, Holst JJ, Christensen NJ. Glucagon and plasma catecholamine responses to graded and prolonged exercise in man. J Appl Physiol 1975; 38: 70-76.*
15. *Billat V, Palleja P, Charlaix T, Rizzardo P, & Janel N. Energy specificity of rockclimbing and aerobic capacity in competitive sport rock climbers. J Sports Med Phys Fit 1995; 35: 20-24.*
16. *Blomqvist CG, Lewis SF, Taylor WF, Graham RM. Similarity of the hemodynamic responses to static and dynamic exercise of small muscle groups. Circ Res 1981; 48, Suppl 1, 87-92.*
17. *Davies, CTM, Few J, Foster KG, Sargeant AJ. Plasma catecholamine concentration during dynamic exercise involving different muscle groups. Eur J Appl Physiol 1974; 32: 195-206.*
18. *Peronnet F, Cleroux J, Perrault H, Cousineau D, de Champlain J, Nadeau R. Plasma norepinephrine response to exercise before and after training in humans. J Appl Physiol 1981; 51: 812-815.*
19. *Winder WW, Hickson RC, Hagberg JM, Ehsani AA, McLane JA. Training-induced changes in hormonal and metabolic responses to submaximal exercise. J Appl Physiol 1979; 46: 766-771.*
20. *Sinoway L, Rea R, Smith M, Mark A. Physical training induces desensitization of the muscle metaboreflex. Circulation 1989: 80 Suppl: II 289.*
21. *Somers VK, Leo KC, Green MP, Mark AL. Forearm training alters the sympathetic nerve response to isometric handgrip. Circulation 1988; 78 Suppl: II 177.* □

Position Statement: Code of Ethics in Sports Medicine International Sports Medicine Federation (FIMS)

The following Statement was approved by the FIMS Executive Committee on 23 September 1997

1. Medical ethics in general

The same ethical principles that apply to the practice of medicine shall apply to sports medicine. The main duties of a physician include:

- Always make the health of the athlete a priority.
- Never do harm.
- Never impose your authority in a way that impinges on the individual right of the athlete to make his/her own decisions.[1]

2. Ethics in Sports Medicine

Physicians who care for athletes of all ages have an ethical obligation to understand the specific physical, mental and emotional demands of physical activity, exercise and sports training.

A different relationship exists between sports medicine practitioners, their employers, official sports organization, professional colleagues and the athletes.[2] In sports medicine there is also a link between the pathologic concern and specific recreational and professional activity. An athletic injury has a direct and immediate impact on the participation in this activity that may have psychological and financial implications. The most obvious difference between sports medicine and other aspects of medicine is that the athletes treated are generally healthy.

Ethics in sports medicine should also be distinguished from law as it relates to sport. One refers to morality the other to a set of enforceable social rules.[2] Although it is desirable that the Law be grounded in moral principles and that matters of moral importance should be given legal backing in many instances, not everything that is illegal is immoral and similarly not every immoral behaviour is against the law. Thus when speaking of ethics in sports medicine, one is not concerned with etiquette or law, but with basic morality.

3. Special Ethical Issues in Sports Medicine

The physician's duty to the athlete must be his/her first concern and contractual and other responsibilities are of secondary importance. A medical decision must be taken honestly and conscientiously.

A basic ethical principle in health care is that of respect for autonomy. An essential component of autonomy is knowledge. Failure to obtain informed consent is to undermine the athlete's autonomy. Similarly, failure to give them necessary information violates the right of the athlete to make autonomous choices. Truthfulness is important in health care ethics. The overriding ethical concern is to provide information to the best of one's ability that is necessary for the patient to decide and act autonomously.

The highest respect will always be maintained for human life and well-being. A mere motive of profit shall never be permitted to be an influence in conducting sports medicine practice or functions.[3]

4. The Athlete-Physician Relationship

The physician shall not allow consideration of religion, nationality, race, party politics or social standing to intervene between his/her duty and the athlete.

The basis of the relationship between the physician and the athlete should be that of absolute confidence and mutual respect. The athlete can expect a physician to exercise professional skill at all times. Advice given and action taken should always be in the athlete's best interest.

The athlete's right to privacy must be protected. The regulations regarding medical records in health care and medicine shall also be applied in the field of sports medicine. The sports medicine physician should maintain a complete and accurate record of the patient.

In view of the strong public and media interest in the health of athletes, the physician should decide with the athlete what information can be released for public distribution.[1]

When serving as a team physician, the sports medicine physician assumes the responsibility to athletes as well as team administrators and coaches. It is essential that each athlete is informed of that responsibility and authorizes disclosure of otherwise confidential medical information, but solely to the specific responsible persons and for the expressed purpose of determining the fitness of the athlete for participation.[4]

The sports medicine physician will inform the athlete about the treatment, the use of medication and the possible consequences in an understandable way and proceed to request his or her permission for the treatment.

The team physician will explain to the individual athlete that he or she is free to consult another physician.

5. Training and Competition

Sports medicine physicians should oppose training and practices and competition rules as they may jeopardize the health of the athlete. In general, the physician shall obtain knowledge of the specific and mental demands made of athletes when they participate in sport activities. Relevant aspects in this respect include expertise, effectiveness and efficiency, and safety.[5]

If the athletes concerned are children or growing individuals, the physician must take into consideration the special risks that the sport in questions may represent to persons who have not yet reached physical or psychological maturity. When the sports participant is a growing individual, the sports physician must ensure that the training and competition are appropriate for the state of growth and development.[4] The physician shall contribute to the spreading of information or the special conditions that pertain to young people training and competing. It is vital that this information also reaches the young athletes, parents, guardians, and trainers.[1]

6. Education

Sports medicine physicians should participate in continuing education courses to improve and maintain the knowledge and skills that will allow them to provide optimal advice and care to their patient athletes.[6] Knowledge should be shared with colleagues in the field.

7. Health Promotion

Sports medicine physicians are obligated to educate people of all ages about the health benefits of physical activity and exercise.

8. Injuries and Athletes

It is the responsibility of the sports medicine physician to determine whether the injured athletes should continue training or participate in competition. The outcome of the competition or the coaches should not influence the decision, but solely the possible risks and consequences to the health of the athlete.

If the physician considers that a certain sport entails major risks he should try to eliminate the risk by exerting pressure on the athletes as well as on the relevant decision makers.

Injury prevention should receive the highest priority.

9. Therapeutic Exercise

When supported by scientific research, a detailed exercise prescription should be part of the therapeutic plan for an athlete recovering from injury or disease.

10. Relationship with Other Professionals

The sports medicine physician should work in collaboration with professionals of other disciplines. The sports medicine physician should cooperate with physical therapists, podiatrists, psychologists, sport scientists including biochemist, biomechanics, physiologists, and others. The sports medicine physician has the final responsibility for the health and appropriate medical specialists in the prevention, treatment and rehabilitation of disease and injury. The concept of interdisciplinary team work is fundamental to the practice of sports medicine.

A sports medicine physician should refrain from publicly criticizing fellow professionals who are involved in the treatment of athletes.

A sports medicine physician should behave in relation to his colleagues and coworkers as he would like them to behave towards him.

When a sports medicine physician recognizes that the athlete's problems are beyond his level of expertise, it beholds him to advise the athlete of other persons with the necessary expertise and refer the athlete to such appropriate persons for assistance.

11. Relation to Officials, Clubs, etc.

At a sport venue, it is the responsibility of the sports medicine physician to determine when an injured athlete can participate in or return to an event or game. The physician should not delegate this decision. In all cases, priority must be given to the athlete's health and safety. The outcome of the competition must never influence such decisions.

To enable the sports medicine physician to undertake this ethical obligation the sports medicine physician must insist on professional autonomy and responsibility for all medical decisions concerning the health, safety and legitimate interest of the athlete. No third party should influence these decisions.[3]

No information about an athlete may be given to a third party without the consent of the athlete.

12. Doping (see FIMS Position Statement)

The sports medicine physician should oppose and in practice refrain from using methods to improve performance artificially such as those prohibited by the IOC.[4]

The physicians have forcefully opposed the use of methods that are not in accordance with medical ethics or scientifically proven experience. Thus, it is contrary to medical ethics to condone doping in any form. Neither may the physician in anyway mask pain in order to enable the athlete's return to practicing the sport if there is any risk of aggravating the injury.[1]

13. Research

Research should be conducted following the ethical principles accepted for research in animals and human subjects. Research should never be conducted in a manner which may injure athletes or jeopardize their athletic performance.

REFERENCES

1. *Code of Ethics. Swedish Society of Sports Medicine.*
2. *Hodge KP. Character building in sport: fact or fiction? New Zealand Journal of Sports Medicine 17(2):23-25, 1989.*
3. *Code of Ethics. Sports Medicine Australia.*
4. *Principles and Ethical Guidelines of Health Care for Sports Medicine. International Olympic Committee.*
5. *Code of Ethics. The Netherlands Association of Sports Medicine.*
6. *Code of Ethics. The American College of Sports Medicine.*

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SASMA NEWS

Welcome to the first of a regular feature in the new Journal. As one of the changes to the Journal, these pages will contain local and international news, other features, and details of congresses.

Privileged healthcare professionals who received Froben's new Sportsmed Quarterly would have been intrigued to discover the existence of a new sports medicine organisation, SASMO. This "Organisation" in the end turned out to be the "Association" in mis-spelt guise. Acronyms are well loved in SA. But just what are SAGCA, NOCSA, NSC, IOC, ANOCA, SISA, SSISA, ACSM, ETC? We'll investigate some of these in the months to come.

What is SISA?

The Sports information and Science Agency (SISA) is an agency which at present is fully funded by the Department of Sport and Recreation. According to the secretariat Mr. Galant, Directors are made up from equal representation from the Department, the National Sports Council and the National Olympic Committee. SASMA members may remember their involvement in funding part of the SASMA Biennial General Meeting in Sun City last year. They have also collaborated with SASMA in bringing out the position statement on AIDS in sport.

The aim of the agency is the rendering of support services to sportspeople in the Medical, Scientific and Physiotherapy fields, and also in the areas of information and technology. They see SASMA as being an important client of SISA in advising and rendering quality sports medicine. SISA plan to have the various aspects of Sports Medicine and Science accredited, and that effectively means activities in all the fields in which SASMA members are currently involved. At present they have accredited nineteen Institutions in South Africa (Technicons, Universities, and the Sports Science Institute), and the plan is to accredit the clinicians in the future. The Sports Science and Medicine Listing book that SASMA members received recently is the first stage in this process. It has also been sent to the 120 Sports Federations as a source of information (without specific guarantee of quality!), to enable them to make their own decisions in health care, as the accredited infrastructure is, not yet in place.

Commonwealth Games 1998 (and SACGA)

The Queen's baton passed through SA on its first visit to this country, as part of its relay run to Malaysia. The South African Commonwealth Games Association (SACGA) is the body responsible for organising this event every four years.

Supporting the athletes in one of the world's major sporting events is a medical and scientific team. The stated aims are to ensure that the physical well-being of the athletes are carefully monitored, and timeous intervention instituted when necessary, and also to identify, encourage and promote participation of sports physicians, physiotherapists and sports scientists from the so-called disadvantaged communities in the support structure.

The team provides support in three fields around the country.

Scientific: Ms. Noo Scales & Mr. Justin du Rant (Cape Town), Prof. Geoff Rodgers (Johannesburg), Dr. Yoga Coopoo & Prof. Maurice Mars (Durban), Drs. Ian Cook, Jones Cilliers & Jacques Rossouw (Pretoria).

Medical: Profs Wayne Diesel & Martin Schwellnus (Cape Town), Drs. Ponky Firer & Demitri Constantinou (Johannesburg), Dr. Mike Marshall (Durban), Dr. Philda de Jager (Pretoria).

Physiotherapy: Prof. Wayne Diesel (Cape Town), Ms. Tanya Bell (Johannesburg), Ms. Joyce Morton (Durban), Ms. Jaqui McCord (Pretoria).

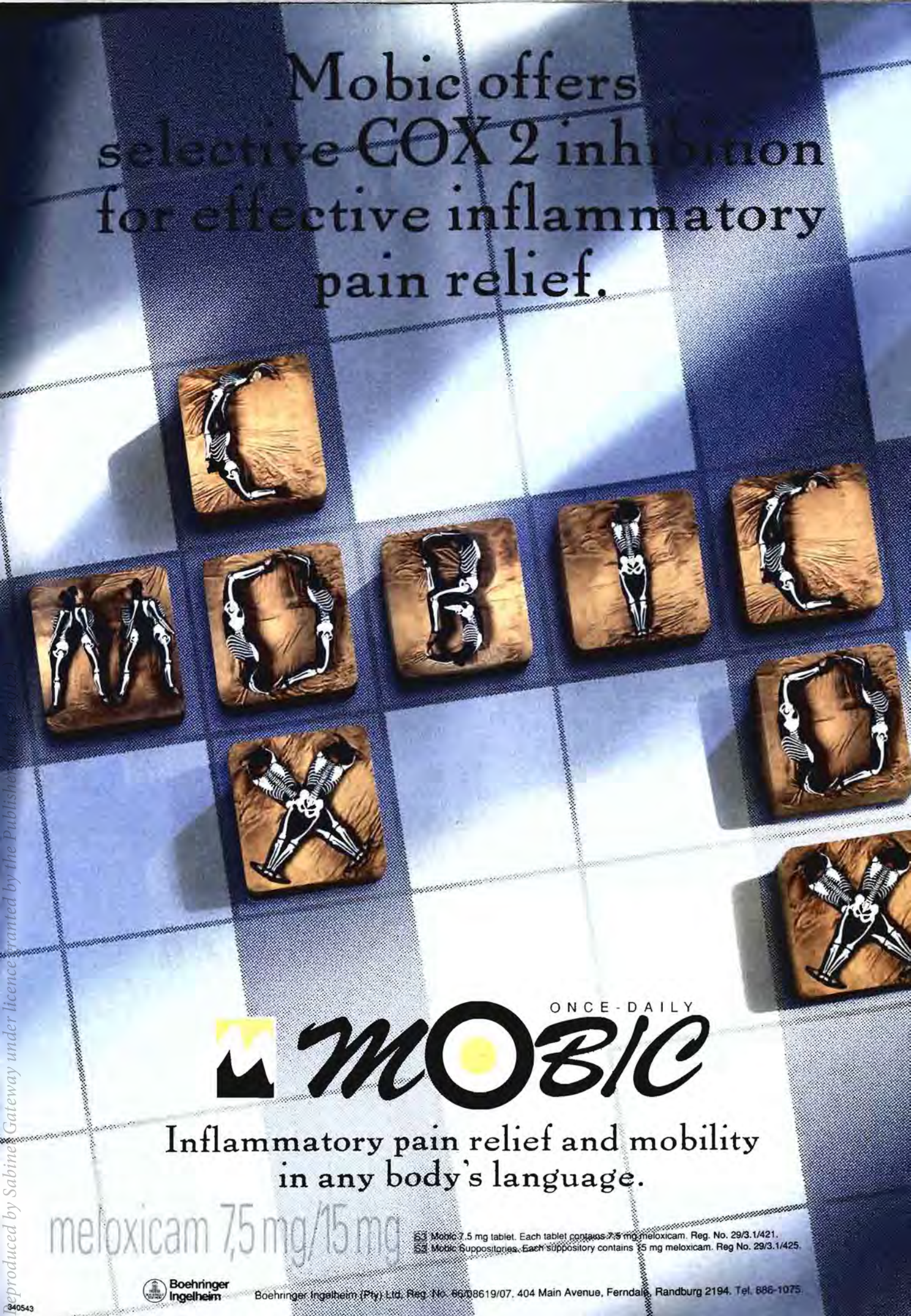
Mr. Khalid Galant coordinates the scientific evaluation and monitoring of athletes through the High Performance programme of SISA. The ubiquitous Dr Ismail Jakoet is the appointed Chief Medical Officer, and oft-quoted Prof. Tim Noakes, the Medical Advisor.

The medical teams continue to have a strong, albeit dubious influence on the physical appearance of our national teams. One of the major inputs in the field of physiotherapy has been the appearance of tattoos on the hides of the Springbok rugby team. This was taken further at the Commonwealth Games where the medical team won distinction by being fined for not dressing properly. Apart from these sartorial slipups, tours such as the Commonwealth Games have served to expose various sporting codes to current views and good sports medicine practices. Many coaching or management practices may seem outdated or inappropriate to the medical team. There in certainly a perceived need to educate coaches and managers, but perhaps the tour itself is not the best place to start doing this. These sporting disciplines and personnel may benefit from being more involved in SASMA activities.

Other Events

Remember that our own SASMA Congress has moved from its usual March slot to September 1999, to coincide with the All Africa Games, and will be held in Johannesburg, rather than Cape Town. This allows the conference to receive sponsorship from ANOCA and NOCSA.

Current SASMA President Prof. Wayne Derman says that the conference will be an "African Renaissance" project at the request of the African Renaissance Committee of the Deputy President (is that ARCODP?) Incoming SASMA president Dr Philda de Jager represents a departure from recent history and illustrates the benefit of having a rotating presidency. Being the first female president, and the first inland president for some time. We hope she will be able to keep her head above water a little better than she has done in her sporting life ... (her women's underwater hockey team returned as World Champions from their tour to the US). □



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