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The South African Journal of Sports Medicine

The official publication of the South African Sports Medicine Association

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AHEAD IN COX-2 SELECTIVITY WITH A 3 YEAR TRACK RECORD.



SELECTIVE COX-2 INHIBITION WITH MELOXICAM IS ASSOCIATED WITH REDUCED NEED FOR HOSPITAL TREATMENT COMPARED WITH CLASSICAL NSAIDS

RESULTS

Parameter	Meoxicam	Paracetamol
Number of patients	100	100
Number of hospitalizations	10	25
Number of days of hospitalization	100	250

GASTROINTESTINAL TOLERABILITY OF MELOXICAM COMPARED TO DICLOFENAC IN OSTEOARTHRITIS PATIENTS

CONCLUSIONS

Meoxicam was found to be significantly better tolerated than diclofenac in patients with osteoarthritis. The incidence of gastrointestinal adverse events was significantly lower in the meoxicam group compared with the diclofenac group.



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Editorial



A few weeks ago I received a late night telephone call from a distraught father of an adolescent elite athlete who had been informed 3 days before the call to me that his son had tested positive for a stimulant at a national sports competition. The stimulant was one that is commonly found in medication used to treat upper respiratory tract infections. This

young athlete, who is not yet of legal age, complained of upper respiratory tract symptoms a few days before a national competition and went to see a specialist for treatment. His main concern was not to let down his team during the forthcoming competition. Trusting the treating doctor, he took whatever medication was required to get well before competing. A few days later he tested positive for the stimulant. This young athlete now faces a hearing, and if found guilty his team, whom he did not want to let down, may lose its medal. The long-term consequences on this young superstar's athletic career, and more importantly the psychological trauma on this child, cannot be quantified at this stage. It is sad that an incident such as this still occurs in a country that prides itself on its professional approach to sport.

Of particular concern is the fact that this young athlete was not aware of substances that are banned in his sport, and secondly that he put his trust in a specialist doctor for treatment of an illness a few days before a national event. At what age should an athlete be expected to take full responsibility for not taking a banned substance? Who takes responsibility for such a young athlete? These circumstances once again emphasise the important role that medical staff play in the sports medical care of athletes at this level. Surely the time has come for the care of athletes to be placed in the hands of well-trained, professional medical staff who are able to deal with and advise athletes on issues such as doping in sport.

This incident is a classical case of 'accidental doping'. It illustrates clearly that not enough is being done to educate medical staff, coaches and athletes at all levels, but particularly young athletes, on the issues surrounding doping in sport. I would like to see the authorities responsible for drug testing allocate more time and financial resources to educating athletes as well as medical staff in order to prevent a repeat of this unfortunate incident. A vigorous information campaign among athletes prior to participating in events where drug testing is likely to take place is required. Coaches and parents (in the case of minors) should be well informed by the authorities before sports events or out-

of-competition testing campaigns are undertaken. It is also important that medical personnel who treat athletes are fully aware of the rules and regulations pertaining to drug use in sports.

Sports medicine, as a discipline, has evolved at an incredibly fast rate over the last decade. The core body of knowledge in this field has increased to such an extent that there is even discussion about sub-specialisation within sports medicine. In South Africa, the interest in postgraduate training courses in sports medicine has paralleled this growth, and it is not inconceivable that sports medicine will be a registered specialty in South Africa within the next 3 years. Professional athletes require professionals to care for them. Currently medical doctors with a postgraduate sports medicine qualification are not recognised for their important and responsible role. Often this lack of professional recognition is not only from colleagues, but also from sports authorities; consequently sports medicine professionals in South Africa are not remunerated adequately for their services. The professional risk that a doctor takes in caring for a professional athlete is substantial and is probably as high, if not higher, than the professional risk that an obstetrician is exposed to in performing a delivery.

The *South African Journal of Sports Medicine* is an important tool for continued medical education. This first edition of the journal for 1999, although slightly delayed due to the fact that we had to find a new publisher, contains valuable information on a number of topics in sports medicine. The position statement published by the International Sports Medicine Federation on diabetes mellitus and exercise will be of value to any professional who cares for athletes with diabetes mellitus. This edition also contains a number of research articles pertaining to sports performance, and there are two papers that focus on heart rate responses during running and cricket.

On behalf of the Editorial Board I would like to thank the South African Medical Association's publication division for their willingness to assist us in the publication of this journal. I am convinced that their involvement will add considerable value to the journal, and we look forward to a long-term relationship with them.

Martin P Schweltnus

Editor-in-Chief

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The pathophysiology of compartment syndromes: microcirculatory compromise as a cause of exercise-induced limb pain

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Abstract

This paper summarises the pathophysiology of raised intracompartmental pressure (ICP) and its effects on capillary and hence nutrient blood flow. The important relationship of ICP to mean arterial pressure and diastolic pressure is discussed, as are the principles of diagnosis and management of raised ICP. In order to reduce morbidity there is a need to raise awareness of the consequences of raised ICP, and to make a diagnosis of raised ICP rather than wait for the functional impairment of a compartment syndrome to occur.

Introduction

The purpose of this contribution is to introduce the concept of tissue pressure within a closed compartment, to review the basic physiology of capillary blood flow and to relate these to the events that may lead to exercise-induced limb pain of vascular origin. There are two interrelated concepts, namely those of increased tissue pressure and reduced inflow pressure; although discussed as separate issues, these in fact usually occur synchronously in practice.

The apparent paradox of cell death and gangrene occurring in a limb in which bounding pulses can be felt, is emphasised. The effects of an increase in tissue pressure and arterial stenosis on capillary flow are discussed.

Arterial disease, particularly stenosis, is often thought of as the prerogative of the elderly, but recent-

ly the recognition of entrapments, injuries and congenital abnormalities has increased awareness of stenoses in young active individuals. Raised compartment pressures and compartment syndromes occur in a myriad of clinical situations and awareness is essential for diagnosis.^{1,4-7,9,12,15,17-20,22,25-27,29} No practitioner can afford to be unaware of the concepts involved.

Pathophysiology

Arterial stenosis

Any lesion occupying space in a vessel will alter the dynamics of blood flowing past the lesion. The energy wasted in turbulent flow causes a loss in potential energy that is measured as a fall in blood pressure distal to the stenotic lesion. This reduction in blood pressure results in a reduction in the pressure at the arteriolar end of the capillary bed and therefore relative hypoperfusion of the exercising muscle distal to the stenosis.²⁴

During exercise there is an increased demand for oxygen that is normally met by increased muscle blood flow. If this cannot be achieved because there is a problem with inflow, then claudication may result. Further, as muscle volume increases during exercise and is contained within a relatively inelastic osseofascial compartment, there is a tendency for the compartment pressure to rise. The relationship between inflow pressure and tissue pressure is critical in determining the adequacy of tissue perfusion and cellular oxygenation.

Compartment syndrome

Matsen¹⁶ has defined a compartment syndrome as damage to the cells caused by increased tissue pressure within a compartment, resulting in loss of function. We do not want to wait for loss of function before recognising and treating changes in tissue pressure, and it is our aim to diagnose an increasing compartment pressure and institute treatment before a compartment syndrome occurs.¹⁴ Increased tissue pressure in itself is not diagnostic of inadequate cell nutrition, rather it is tissue pressure relative to the inflow pressure that is crucial.^{8,28} Inflow pressure to a muscle group can be low, even in the presence of systemic hypertension, if there is a lesion in the axial artery that causes a loss of pressure.

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Muscle nutrient blood flow and the effects of stenosis and raised intracompartment pressure

Any fluid, including blood, will only flow along an energy gradient. To all intents and purposes this means a pressure gradient. Although there are some situations where blood will move against an apparent pressure gradient (for example, when standing the blood pressure in the foot will be higher than the pressure in the aorta), the kinetic and potential energy of the blood is sufficient to overcome this.²⁴

Therefore for the purposes of this discussion, in order for blood to circulate throughout the body there must be a gradual fall in blood pressure from the left ventricle, through the capillary beds and the venous system, to the right ventricle. The left ventricular pressure is reflected in the systolic blood pressure. The diastolic pressure reflects the peripheral resistance. It is convenient to think in terms of the mean arterial pressure (MAP), which is an expression of the effective pressure generated by pulsatile flow. It is calculated as the diastolic pressure plus one-third of the pulse pressure.

Blood pressure is affected by the stroke volume, i.e. the amount of blood that must be moved, and the peripheral vascular resistance against which the blood must be pumped. As peripheral vascular resistance increases, the blood pressure must increase to maintain the same flow. If the blood pressure cannot be increased then flow must fall.

At a micro-circulatory level the same principles hold. The driving force, or inflow pressure to the capillary bed, must be lower than the systemic blood pressure, but is derived from it. Regional inflow pressure may be reduced by a systemic hypotension or by the presence of a proximal stenotic lesion. The resistance to flow at this level is determined by the calibre and length of the capillaries and the pressure gradient across the capillary bed.²⁴ As capillaries are a single cell thick and have little inherent strength, their diameter is governed by the tissue pressure around them.² In addition to these factors the function of the capillary is to allow nutrients, including oxygen, to diffuse into the cells. For this to occur, Starling's hypothesis suggests that fluid leaves the arterial end of the capillary and fluid is returned at the venous end.

This is again, like all fluid shifts, a pressure-dependent process, with the driving forces being the arteriolar capillary pressure and the osmotic pressure of the interstitial fluid, and the opposing forces being the tissue pressure and the oncotic pressure that tends to keep fluid within the vessel.²⁴

Anything that disturbs this balance will interfere with cell nutrition. Therefore reducing the perfusion pressure by either lowering arteriolar pressure or increasing venular pressure, will tend to reduce flow. Increasing the tissue pressure or lowering capillary pressure will therefore secondarily affect oxygen delivery.

Increasing tissue pressure will also tend to cause collapse of the vessel, a tendency that is minimised by a reflex increase in venous pressure.³ While keeping the vessel open this also has the effect of reducing the

pressure gradient and thereby reducing flow.

Obviously this will be maximal in situations where there is no flow.

No flow will occur when the pressure gradient is reduced to zero. In fact flow ceases before this when the pressure difference is so low that the thickness of the blood becomes an important factor. The rheological features of blood are affected by many factors, including cell count and protein content, and in many clinical situations reducing the haematocrit will improve oxygen delivery to the tissues by improving the flow.²⁴

Any tendency to an increase in tissue pressure is usually accommodated by increase in volume of the tissue, or swelling. Compromise of capillary blood flow only occurs in those anatomical areas where swelling is not possible, or can only occur to a limited extent. A prime example is the intracranial haematoma that in the adult cannot be accommodated by increasing the volume of the skull, so pressure in the head rises. Similarly, any increase in pressure within an osseofacial compartment cannot be accommodated by increasing the volume of the compartment, consequently compartmental pressure will rise. In fact within any fascial space there is potential for a harmful increase in pressure and the importance of micro-compartments, such as within the epineurium, should not be forgotten.²³

As the microcirculatory pressures are much lower than pressures in the axial vessels, it is possible, in fact usual, to have normal axial artery pulses despite complete cessation of flow in the capillary beds.¹⁸ This is an important concept — the muscles of a leg may be gangrenous in the presence of arterial pulses.

Time is also an important parameter. The tolerance of tissue to ischaemia is varied, with neuronal tissue being most sensitive.²³ In clinical practice, therefore, early signs of ischaemia are often neuronal, such as paraesthesia or anaesthesia.

In summary, the pressure gradient across a capillary bed is small, much less than mean arterial pressure, but derived from it. If the systemic pressure is lowered, then the pressure gradient across the capillary bed will be lowered. Capillary blood flow will stop when tissue pressure exceeds or comes close to arteriolar inflow pressure. At this stage the axial artery pulses will still be clearly palpable. When capillary blood flow stops, cell death will ensue within a short time period that is dependent on the type of tissue involved, but which is shortest in neural tissue.

Management

Management can be aimed at either elevating the perfusion pressure by resuscitating the individual, or at reducing the tissue pressure by decompression of the compartment, or both. Definitive management of an arterial lesion by resection, bypass or disobliteration may be necessary for ultimate resolution of the problem.

Assessment may be clinically difficult. Particularly troublesome are children in whom it is difficult to elicit clinical signs or in whom co-operation is a problem. In patients with spinal injuries, be they traumatic or iatrogenic injuries such as those resulting from epidural anaesthesia, signs may be difficult to elicit. When the limb is encased in plaster, examination may be limited.²¹ For these reasons invasive compartment pressure monitoring is advised; the techniques and indices for intervention will be discussed by other authors.

Management is usually described in terms of surgical decompression, but a lot can be done while waiting for surgery, which may occasionally obviate the need for an operation. This has been aptly described as 'first aid for hypoxic cells'. All circumferential dressings should be removed, the limb should be kept at heart height as elevation decreases the arterial perfusion pressure, the patient should be resuscitated with crystalloid to restore volume and reduce haematocrit, and maximum oxygenation should be assured.¹³ Compartment pressure can be measured serially during these manoeuvres and compared with MAP. We believe that in children fasciotomy should be considered when compartmental pressure reaches MAP -30 mmHg, and in adults when it reaches diastolic -20 mm Hg.¹⁴ Time is of the essence and such patients should be regarded as true surgical emergencies.

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A quantification of competition-induced increase in heart rate during 10, 21 and 42 km races

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Abstract

Objective. Our previous study has shown that, at any running speed, heart rates (HRs) are higher during competition than during training. The aim of this study was to determine whether this competition-induced increase in HR (HR_{diff}) was affected by race intensity or perceived effort during the race.

Design. Male runners training at least 60 km/wk underwent a maximal oxygen consumption ($\dot{V}O_{2max}$) test 4 days before a 10 km ($N = 6$), 21 km ($N = 6$) or 42 km ($N = 5$) race. Two days before the race, subjects underwent a track test to determine the relationship between running speed and HR under non-competitive conditions ($r = 0.98 \pm 0.01$).

Results. The average HR during the 10, 21 and 42 km races were 163 ± 13 , 166 ± 10 and 156 ± 6 beats/min respectively, representing 90 ± 5 , 93 ± 4 and $84 \pm 4\%$ of laboratory-derived maximum HR (HR_{max}) ($P > 0.05$). The subjects ran the 10, 21, and 42 km races at 74 ± 5 , 75 ± 4 and $64 \pm 7\%$ of peak treadmill running speed. The HR_{diff} for the 10, 21 and 42 km races were 20 ± 7 , 15 ± 7 and 19 ± 13 beats/min respectively. The subjects' perception of fatigue measured after the track test and after each race were not different.

Conclusions. HRs during competition are higher than HRs at the same speed in a non-competitive situation. This HR_{diff} is independent of racing speed and cannot be explained by the perceived fatigue measured immediately after the race. In conclusion, HR measured during competitive exercise is not an accurate measure of exercise intensity.

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Introduction

In a previous study it was found that runners' heart rates (HRs) in 10 and 21 km competitive races were higher than their HRs at similar running speeds during training (HR_{diff}).¹⁴

This study raised two issues. Firstly, HR monitors are used widely by athletes during competition. Many of these athletes measure their HRs during competition in order to select an appropriate exercise intensity for that event. But the athlete will run slower than expected during a race should a racing target HR be calculated on a HR determined during training. Indeed, this very effect and its negative consequences on competitive athletes has already been documented in the lay press.⁵

Secondly, several factors including work load,¹ time of day,¹¹ state of fitness,⁴ dehydration,⁷ plasma volume,¹² duration of exercise⁶ and temperature^{2,10} may affect HR during exercise. However the mechanism causing the HR_{diff} is not known. Nor has the extent of the HR_{diff} been quantified. A better understanding of this phenomenon has practical application for competitive athletes and will contribute to a better understanding of the physiological mechanisms that affect HR during competition.

Accordingly, the aims of this study were to determine the HR_{diff} in runners in competitive races over varying race distances and to determine whether this increase in competition HR was affected by race intensity or perceived effort before and during the race.

Methods

Subjects

The study was approved by the Ethics and Research Committee of the University of Cape Town. Eight long-distance runners who were training at least 60 km/wk were recruited for each of 3 races (10 km, 21 km and 42 km). Informed consent was obtained from all subjects once the testing protocols had been explained.

Study design

Subjects were familiarised with all testing equipment in the orientation phase of this study. Thereafter 4 days before the race each subject underwent a maximum running test on a treadmill during which maxi-

imum oxygen consumption (VO_{2max}) and maximum HR (HR_{max}) were determined. Two days before the race subjects ran on an indoor track to determine their HR/running speed relationship in non-competitive circumstances. All laboratory and field tests were conducted between 5:30 and 8:00 a.m. to coincide approximately with the time of day during which the races were to be held.

During each race runners wore HR monitors (Vantage XL, Polar Electro Oy, Kempele, Finland) that recorded HRs continuously and split times for each kilometre. These data were then retrieved from the HR receiver after the race via an interface to a computer (Polar Electro Oy, Kempele, Finland). Following each laboratory field test and race the perceived level of fatigue of each subject was also evaluated.

Treadmill test

After a 5-minute warm-up, subjects started running on the treadmill at 12 km/h. The treadmill speed was increased by 0.5 km/h every 30 seconds. The test was terminated when the subject was unable to maintain the speed of the treadmill belt. Each subject breathed through a rubber mask that covered his nose and mouth. The expired air was directed to O_2 and CO_2 analysers (OxyconSigma, Mijnhart, Netherlands) for the measurement of oxygen consumption (VO_2), carbon dioxide production (CO_2) and respiratory exchange ratio (RER). The highest VO_2 measured during the test was recorded as the VO_{2max} . The equipment was calibrated before each test with a Hans Rudolph 3 litre syringe and a standard gas of known concentration. HR was recorded throughout the test using a Vantage XL HR monitor.

Track test

After a standard warm-up, subjects ran 1 000 m at a submaximal pace on an indoor tartan track. The starting pace was calculated to be 10 s/km slower than the subject's current 5 km racing pace. Each subject kept his running speed constant by pacing himself with a researcher who blew a whistle at regular intervals coinciding with the time the subject should have been at respective 100 m marks on the track. After a rest period of 2 minutes, the subject ran another 1 000 m, this time 10 seconds faster. This pattern was repeated until the subjects were unable to maintain the required running pace. The subjects usually completed 5 or 6 km before the onset of fatigue. After the test each subject's average HR for the last 60 seconds of each kilometre, and the average running speed for each kilometre, were calculated. Then the line of best fit between HR and running speed was calculated using a linear regression analysis. The ambient temperature during the test was between 19 and 20°C.

Race

Subjects wore HR monitors while competing in 10, 21 or 42 km races. Subjects were instructed to record the time splits for each kilometre by pressing the appropriate button on the HR monitor receiver. The HR was recorded every 5 seconds during the 10 and 21 km

races and every 15 seconds for the 42 km race. The average HR and average running speed were calculated for each kilometre during the race.

The temperature for the duration of the 10 km race was 19°C, between 16 and 19°C for the 21 km race and 20°C at the end of the 42 km race.

Measure of fatigue

The subject's perception of fatigue was evaluated after each indoor running test and race according to the Rose/Noakes running fatigue scale (0 = sluggish, exhausted, unable to run — 10 = best ever).⁸

Body composition

Subject's percentage body fat was predicted using the Durnin and Womersley anthropometric technique.³

Statistics

Descriptive statistics are presented as the mean \pm the standard deviation (mean \pm SD). A line of best fit was calculated between HR and running speed in the field test. The Pearson's correlation coefficient was calculated between these variables. The equation established for the relationship between HR and running speed for each subject was used to calculate his predicted HR based on the racing running speed. The difference between the subject's HR measured in the race and the HR predicted from the regression equation was determined using a paired *t*-test. A Spearman's ranked test was used to determine relationships between the fatigue scores and HR_{diff} . Statistical significance was accepted when $P < 0.05$.

Results

HR data were available for 6 subjects for the 10 and 21 km races and for 5 subjects for the 42 km race. The other subjects had poor HR recordings during the races as a result of malfunctioning HR monitors, and were excluded from further analysis.

General characteristics and laboratory data of the subjects participating in each race are shown in Table I. The relationship between HR and running speed determined in the field test under non-competitive

TABLE I. General characteristics and laboratory data of subjects participating in the 10, 21 and 42 km races (values expressed as mean \pm SD)

Variables	10 km (N = 6)	21 km (N = 6)	42 km (N = 5)
Age (years)	36.8 \pm 4.8	36.0 \pm 4.3	36.2 \pm 4.9
Mass (kg)	75.8 \pm 13.9	75.5 \pm 13.0	69.0 \pm 12.9
Stature (cm)	177.8 \pm 9.6	178.7 \pm 9.5	172.4 \pm 8.0
Per cent fat	14.5 \pm 4.4	15.9 \pm 4.6	12.9 \pm 4.0
VO_{2max} (ml O_2 /kg/min)	53.6 \pm 8.1	53.7 \pm 8.1	53.8 \pm 10.5
HR_{max} (beats/min)	182 \pm 8	181 \pm 10	181 \pm 7
PTRS (km/h)	18.8 \pm 1.3	18.4 \pm 1.3	18.9 \pm 1.7
RER	1.05 \pm 0.05	1.05 \pm 0.05	1.09 \pm 0.06

PTRS = peak treadmill running speed; RER = respiratory exchange ratio.

conditions was $r = 0.98 \pm 0.01$. (mean \pm SD).

The average running times for the 10, 21 and 42 km races were $43:25 \pm 5:15$, $92:48 \pm 7:51$ and $212:03 \pm 24:55$ (min:s). The subjects' personal best times for the 10, 21 and 42 km were $40:31 \pm 4:21$; $89:42 \pm 10:6$ and $190:27 \pm 33:43$ min:s respectively. Therefore, during the 10, 21, and 42 km races in this study the subjects ran 7, 3 and 10% slower than their personal best times for the same distances. The average HRs measured during all three races, and the average HRs predicted at the same running speeds on the basis of measurements taken during the indoor testing, are shown in Table II.

TABLE II. Average measured and predicted HRs for the subjects in the 10, 21 and 42 km races (values expressed as mean \pm SD)

Variables	10km (N = 6)	21 km (N = 6)	42 km (N = 5)
Average measured HR (beats/min)	163 \pm 13	166 \pm 10	156 \pm 6
Predicted HR (beats/min)	143 \pm 22*	151 \pm 13*	137 \pm 17*
HR _{diff} (beats/min)	20 \pm 7	15 \pm 7	19 \pm 13
% HR _{max}	90 \pm 5	93 \pm 4	86 \pm 4
% PTRS	74 \pm 5 [†]	75 \pm 4 [†]	64 \pm 7

* $P < 0.03$ predicted HR v. average measured HR in 10, 21, and 42 km races.
[†] $P < 0.01$ %PTRS 10 km v. %PTRS 42 km.
[‡] $P < 0.05$ %PTRS 21 km v. %PTRS 42 km.
 PTRS = peak treadmill running speed.

The HRs measured and predicted for each subject in the 10, 21 and 42 km races are shown in Figs 1, 2 and 3 respectively. The HRs measured during the races

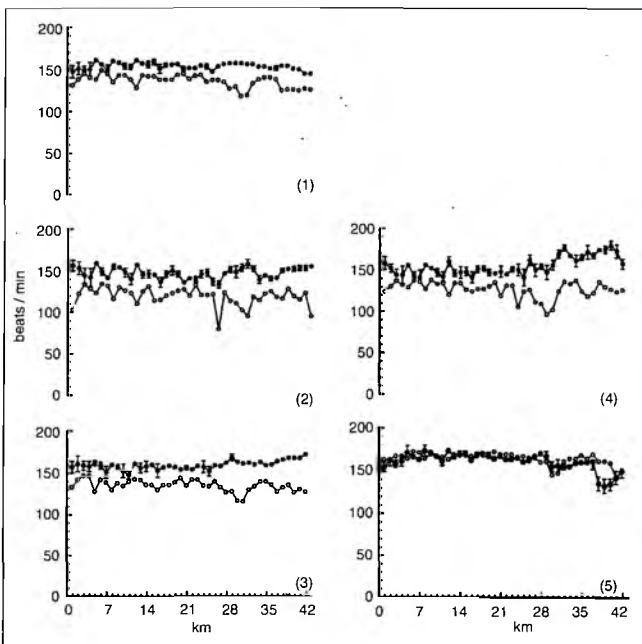


Fig. 1. Hearts rates of the subjects (N = 6) measured during the 10 km race are shown as ●. Each subject's heart rate predicted from the same running speed in non-competitive conditions is shown as ○.

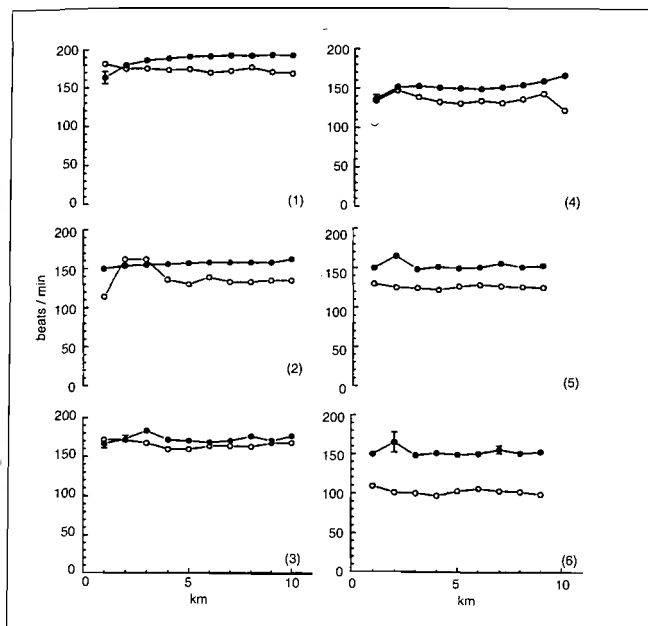


Fig. 2. Hearts rates of the subjects (N = 6) measured during the 21 km race are shown as ●. Each subject's heart rate predicted from the same running speed in non-competitive conditions is shown as ○.

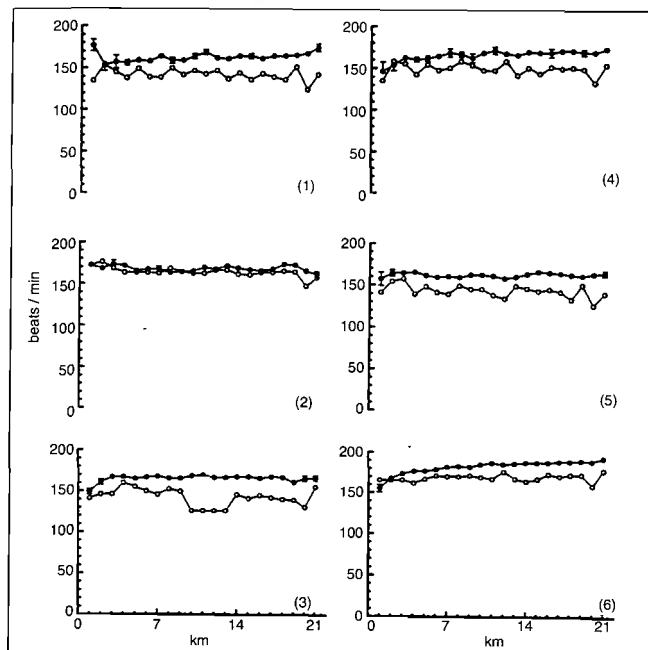


Fig. 3. Hearts rates of the subjects (N = 6) measured during the 42 km race are shown as ●. Each subject's heart rate predicted from the same running speed in non-competitive conditions is shown as ○.

were consistently higher than the HRs predicted from the running speeds during the races in all the subjects ($P < 0.03$).

The fatigue scores, according to the Rose/Noakes scale, after the races and track test are shown in Table III. There was no difference in these fatigue scores measured after the track test or after each race. The

TABLE III. Fatigue scores according to the Rose/Noakes scale after the track test and each race (values expressed as mean \pm SD)

Variables	10 km (N = 6)	21 km (N = 6)	42 km (N = 5)
Race	6.2 \pm 1.7	6.8 \pm 2.5	7.0 \pm 1.7
Track test	7.2 \pm 1.7	6.7 \pm 2.5	6.0 \pm 2.5

level of fatigue after each race, as measured by this questionnaire, did not explain HR_{diff} ($r = 0.01$, $P = 0.96$).

The magnitude of HR_{diff} was not explained by the subjects' VO_{2max} ($r = 0.44$, $P = 0.08$), perception of fatigue after the race ($r = -0.02$, $P = 0.93$), running speed during the race ($r = 0.17$, $P = 0.51$), or running intensity ($\%HR_{max}$) during the race ($r = -0.01$, $P = 0.97$). The graph of racing speed versus $\%HR_{max}$ and racing speed versus HR_{diff} are shown in Fig. 4. There were weak relationships between the laboratory measures of peak treadmill running speed (PTRS) and HR_{diff} ($r = 0.51$ and $P = 0.04$), and HR_{max} achieved during the treadmill test and HR_{diff} ($r = 0.64$, $P = 0.006$).

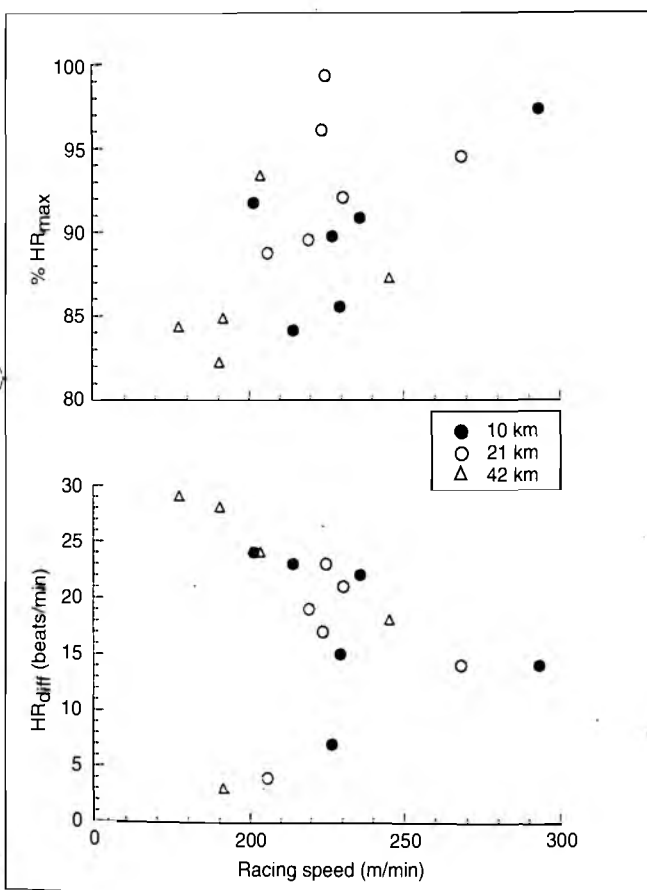


Fig. 4. Graphs of racing speed (m/min) v. $\%HR_{max}$ and HR_{diff} (beats/min) during 10 (N = 6), 21 (N = 6) and 42 km (N = 5) races.

Discussion

The major finding of this study was that HR measured during races of varying distances was consistently higher than the HR elicited at similar running speeds in non-competitive situations. This finding supports an earlier study done in this laboratory.¹⁴ The mechanism for the HR_{diff} is not known and cannot be ascribed to the perception of effort as the fatigue scores were similar after the non-competitive field test and after the races.

The next important finding was that the running intensity, as predicted as a $\%HR_{max}$, was not different between the 10, 21 and 42 km races (Table II) although there was a significant difference in the subjects' running speed (expressed as a percentage of PTRS) (Table II). This suggests that under competitive racing conditions the HR, expressed as a percentage of HR_{max} , is not an accurate marker of exercise intensity. This is in contrast to a non-competitive situation where HR as a measure of exercise intensity accurately reflects increases in running speed ($r = 0.98$).

It should be noted that the subjects ran 3 - 10% below their best times for the various race distances in this study. It is therefore tempting to conclude that the results could even underestimate the real extent of HR_{diff} in elite athletes. Had the subjects been racing with the intention of trying to better their personal best times, then it is possible that the HR_{diff} could have been greater.

The regulation of HR during exercise is complex^{9,13} and it is beyond the scope of this study to explain the mechanism causing HR_{diff} . It can, however, be concluded that the HRs during competition are higher than the HRs at the same running speed in non-competitive situations. In addition, this HR_{diff} cannot be accounted for by the subjects' perception of fatigue measured after the race or running speed or running intensity during the race (Fig. 4). Although there were weak relationships between the laboratory measures of PTRS and HR_{diff} ($r = 0.51$) and HR_{max} achieved during the treadmill test and HR_{diff} ($r = 0.64$), these relationships lacked the power needed to predict accurately HR_{diff} from laboratory measurements conducted before the race.

In summary, HR measured during competitive exercise is not an accurate measure of exercise intensity.

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Improved time-trial performance after tapering in well-trained cyclists

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Abstract

Objective. The purpose of this study was to determine whether cycling performance improved during a 14-day taper, the time course of any improvement, and the possible physiological reasons for any change in performance.

Design. Twenty trained male cyclists (> 250 km/wk) were randomly assigned either to a control group that continued training as usual, or to a reduced-training (tapering) group. Groups were further randomly subdivided and then pair-matched into a time-trial group (5 controls and 5 experimental subjects) who completed four 20 km time-trials on days 0, 4, 8 and 14 of the tapering programme, or a laboratory test group that performed a variety of laboratory tests instead of the time-trials. On the same days the remaining subjects participated in a laboratory-based trial in which maximum oxygen consumption, peak power output (PPO) and peak post-exercise blood lactate concentrations, anaerobic power and resting muscle glycogen concentrations were measured.

Setting. Trials were conducted in an exercise laboratory and at a cycling track.

Interventions. Experimental subjects reduced their training frequency to 66% and their weekly training duration to 60% of normal. Training intensity in both experimental and control groups was maintained.

Main outcome measure(s). Performance would be improved after tapering in both time trial and lab-

oratory subjects, with the physiological parameters measured in the latter group providing a physiological explanation for the improved performance.

Results. Time-trial performance became slower in the control subjects from day 0 (28.3 ± 0.3 min) to day 14 (29.3 ± 0.2 min), but became significantly faster ($P < 0.05$) in the reduced training (taper) group from day 0 to day 8 ($29.5 - 28.7 \pm 0.8$ min) and from day 8 to day 14 ($28.7 - 28.5$ min). Similarly, PPO increased significantly with reduced training from day 0 (382 ± 32 Watts (W)) to day 14 (404 ± 34 W; $P < 0.05$), but did not change in the control group. No other measured physiological variable changed significantly.

Conclusions. Similar to previous reports, these data demonstrate that a 14-day period of reduced training can improve exercise performance, both in field tests and in laboratory tests, in cyclists who habitually train 250 km or more per week. Eight to 14 days of reduced training were required for time-trial performance to be optimal. However, the mechanism for this effect was not identified by the measured physiological variables.

Introduction

Athletes frequently reduce their training load immediately before major competition or after a competitive season in order to allow recovery from the fatigue associated with racing and heavy daily training.^{7,10}

Recent findings show that a number of physiological and performance adaptations can be maintained even when training volume and frequency are reduced,^{9,10,13} provided that the usual exercise intensity is maintained.⁹ However, many athletes fear that any reduction in training volume for more than a few days will inevitably lead to a dramatic fall in fitness and impaired racing performance.

Studies of reduced training have been done on runners,^{10,11} swimmers¹² and cyclists.¹⁴ The aim of the present study was to determine the effect of a period of reduced training on the exercise performance of cyclists using both a field and laboratory test to evaluate performance changes and provide possible physio-

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logical mechanisms that might explain the findings. These tests included measures of neuromuscular mechanisms in addition to conventional measures of aerobic and oxygen independent 'fitness'. It was hypothesised that whatever mechanisms may be responsible for improvement in performance after tapering would apply equally whether the cyclist was endeavouring to improve performance in the laboratory or on the track.

Materials and methods

Twenty well-trained male cyclists volunteered to participate in this study, which was approved by the Research and Ethics Committee of the Faculty of Medicine of the University of Cape Town Medical School. All signed informed consent and were free to withdraw from the trial at any time. Entrance to the study was limited to cyclists who had consistently trained more than 250 km/wk throughout the year for at least the last year. This included racing and interval training. Before the study subjects were asked to record a 2-week training schedule in order to determine their training volumes, frequencies and intensities. Typically, training consisted of one training session of approximately 100 km on the weekend, with five other training sessions of approximately 60-minute duration during the week. Most training was done at moderate intensity (based on subject heart rate and rating of perceived exertion), with approximately one ride per week of high intensity.

Subjects were randomly assigned to control and experimental groups. Control subjects continued their usual training programme, whereas the experimental subjects followed a 14-day taper regimen during which training frequency was reduced to 66% and training duration to 60% of their normal training schedules. However previously documented training intensity was maintained throughout the study. Training was reduced in a single step rather than a graded reduction. Whereas it has been suggested that a graded reduction may be advantageous over a step reduction,¹² this applies to swimmers, not runners. Runners may need a greater reduction in training than is suggested in the studies cited by Houmard and Johns.¹² The control and experimental (reduced-training) groups were then further subdivided into laboratory and field groups, and then pair-matched. On the day before a test each subject performed a training session standardised according to previous training documented in his training log book.

After familiarisation with the procedures, cyclists in the two field groups performed four 20 km time-trials on an outdoor cycle track on days 0, 4, 8 and 14 of the study. Time-trials were conducted individually to prevent drafting. The control subjects were used to control for the influence of environmental factors, especially differences in wind speed and temperature. Matched subjects from both the control and experimental groups performed the time-trial on the same day and at the same time of day to avoid any diurnal variation

in performance measures, on the subject's own bike, and wearing the same type of clothing.

Subjects in the laboratory group were tested on days 0, 4, 8 and 14 for a number of physiological parameters, described subsequently.

Before the start of the performance trials all subjects were weighed and their per cent body fat was calculated from biceps, triceps, subscapular and supra-iliac skinfold measurements using a skinfold caliper (Holtan Ltd., Crymych, UK) according to the equations of Durnin and Wormersley⁶ as described by Brozek *et al.*²

Subjects in the laboratory trial reported to the laboratory on days 0, 4, 8 and 14 for the performance of a Wingate Anaerobic Test (WT) on a mechanically-braked Monark 818 cycle ergometer (Varberg, Sweden) that was calibrated by means of a weighted torque balance attached to the crank. The ergometer was modified with toe straps, adjustable racing handlebars and saddle, and interfaced with an Apple IIE microcomputer. The same seat height was used for each individual during each test. Sample periods of 0.5 seconds enabled calculation of peak power (WT-PP), taken as the highest average power for any 5-second period during the test and the mean power (MP) that was sustained throughout the test.

After a 5-minute warm up at a load and speed selected by the cyclist, subjects began pedalling at minimal resistance and gradually increased the cadence to 115 - 120 rev/min. When this cadence was attained, the full load (0.075 kp/kg body weight) was applied by an investigator and the computer programme was activated.⁷ Subjects were instructed to remain seated and were verbally encouraged to maintain maximal pedal rates throughout the test, i.e. to perform 'all out' from the start. Blood lactate concentrations were determined spectrophotometrically from venous blood (1 ml samples) drawn immediately on completion of the Wingate test and then at 1-minute intervals for 5 minutes via a cannula placed in an antecubital forearm vein before testing commenced.

After a 10-minute rest period, testing was conducted for determination of maximum isometric tension, maximum speed of contraction (V_{max}) and total work done on the same Monark bicycle ergometer as for the Wingate test. From rest, with the pedal 10° above the horizontal, a countdown of 3 seconds was given to the subject who subsequently pedalled as quickly as possible for 5 seconds at randomly chosen resistance settings of 3 kp (29.4 N), 5 kp (49 N), 2 kp (19.6 N), 7 kp (68.6 N), 4 kp (39.2 N) and 6 kp (58.8 N).¹⁵ Rest periods of at least 2 minutes were allowed between each exercise bout. The theoretical power output at zero load (P_0) was calculated from the zero point of the graph of power output versus resistance, V_{max} from a graph of velocity versus resistance and total work done from the area under the power/resistance graph.

After another 30-minute rest period, subjects performed maximal progressive exercise to exhaustion on an electromechanically braked cycle ergometer

(Excalibur, Lode, Groningen, the Netherlands).

For measurement of peak power output (PPO) and maximum oxygen consumption (VO_{2max}), air was inspired from a Hans Rudolph 2 700 one-way valve (Hans Rudolph Inc., Kansas City, Kansas, USA) connected to a Mijnhardt dry gas meter. A nose clip prevented nasal breathing. Expired air was passed through a 15 l baffled mixing chamber from which continuous sampling occurred by Ametek N-22M oxygen and CD-3A carbon dioxide gas analysers (Thermox Instruments, Pittsburgh, PA). Before each test, the gas meter was calibrated with a Hans Rudolph 3 l syringe, and the analysers were calibrated with air and a 4% CO_2 :16% O_2 :80% N_2 mixture. The cumulative value for each minute of the test for ventilation (V_i), oxygen consumption (VO_2), carbon dioxide production (VCO_2), and respiratory exchange ratio (RER) were printed out at the end of the test using standard formulae for calculations.

Subjects first pedalled at a workload of 3.33 Watts (W)/kg for 6 minutes, during which time submaximal VO_2 was measured. Blood was drawn from a forearm vein through a stopcock for subsequent measurement of blood lactate concentrations. Ratings of perceived exertion (RPE) were recorded at regular intervals during the exercise bout using the 1 - 10 point Borg scale. Heart rate was recorded on a Lohmeier M607 Monitor (Munich, West Germany). On completion of the submaximal test, the workload was increased by 50 W for 150 seconds. Thereafter the workload was increased by 25 W every 150 seconds until the subject voluntarily terminated the test. Venous blood was again drawn immediately via the indwelling cannula on completion of the test and then at 1-minute intervals for 5 minutes for subsequent determination of blood lactate concentrations.

Peak workload was calculated using the formula: Peak workload (W) = final workload (W) + (t/150 × 25),⁷ where t is time in seconds of the final workload completed. This battery of tests could lead to fatigue, with one test possibly affecting a subsequent test. However, this was consistent each time that testing was done and would affect all subjects.

Post-exercise muscle samples were obtained at the end of the trial from the right vastus lateralis of each cyclist, using the needle-biopsy technique as described by Bergstrom.¹ The sample was immediately frozen in liquid nitrogen and stored at -80°C for subsequent determination of muscle glycogen content according to the method of Lowry and Passonneau.¹³

Statistical analyses

Statistical significances of changes over time and between groups were assessed by means of a two-way analysis of variance (ANOVA) for repeated measures. When a significant F-ratio ($P < 0.05$) was found, a Scheffe test was used for *post hoc* analysis. Where necessary, between-group differences were measured with a Student's unpaired *t*-test. A value of $P < 0.05$ was regarded as significant.

TABLE 1. Physical characteristics before a reduced training protocol

	Laboratory groups		Field groups	
	Reduced-training group	Control group	Reduced-training group	Control group
Age (yrs)	22.6 ± 2.6	21.2 ± 1.2	26.0 ± 2.9	21.3 ± 0.4
Mass (kg)	75.2 ± 5.4	74.7 ± 4.3	73.9 ± 2.3	71.2 ± 2.7
Body fat content %	9.9 ± 0.8	10.5 ± 0.8	11.6 ± 1.1	10.8 ± 1.0
Peak power output (W)	382.8 ± 32.3	374.6 ± 19.1	-	-
Maximum oxygen consumption (l/min)	4.5 ± 0.2	4.7 ± 0.2	-	-

Values are means ± SD (N = 5 in each sub-group).

Results

Subjects' physical characteristics are shown in Table 1. Subjects in the laboratory group had high VO_{2max} values (4.6 ± 0.2 l/min; mean ± SD) and reached high PPO (394 ± 27 W). Subjects in the time-trial group did not do VO_{2max} or PPO tests as the only point of interest was whether their time-trial performance improved or not. There were no significant differences in age, mass or per cent body fat between the laboratory and field groups or in VO_{2max} and PPO in the control and reduced-training subjects in the laboratory group.

Changes in 20 km time-trial performance in the field group are shown in Fig. 1. Performance for both control and reduced-training (taper) groups were constant for days 0 and 4 of the trial. Thereafter performance in the control group was significantly impaired on days 8 and 14, whereas performance in the reduced training group improved significantly by day 14.

Similarly, PPO measured during the VO_{2max} test (VO_2 -PPO) increased steadily in the reduced training

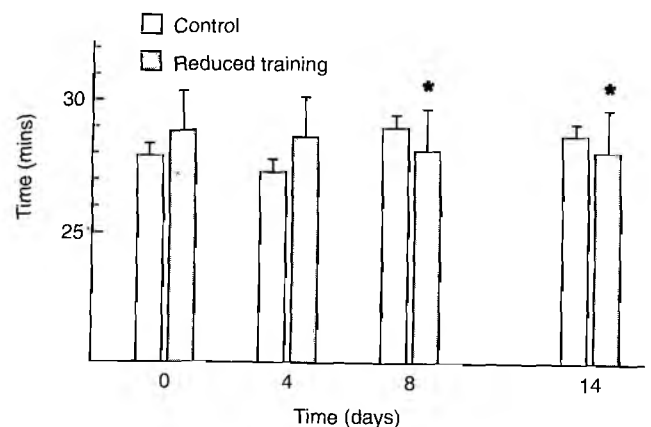


Fig. 1. Twenty km time-trial performance (min) in cyclists in control and reduced-training groups on days 0, 4, 8, and 14. Time-trial performance improved significantly in the reduced-training group on day 14 but was impaired on days 8 and 14 in the control group. Values are means ± SD. *Significant ($P < 0.05$) improvement from day 0.

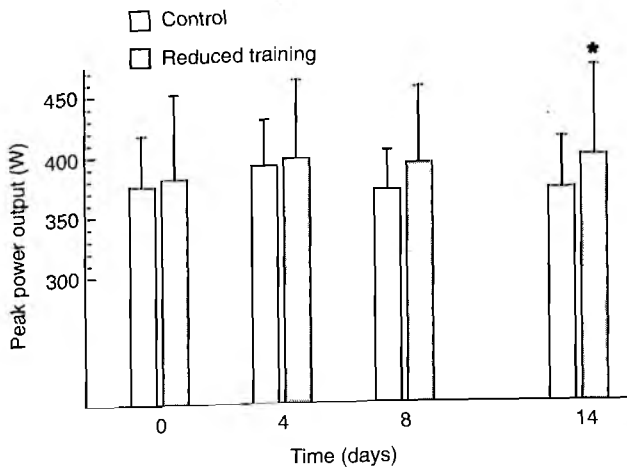


Fig. 2. Peak power output (PPO) (W) in cyclists in control and reduced-training groups on days 0, 4, 8 and 14. PPO was unchanged in the control group but increased significantly on day 14 in the reduced training group. Values are means \pm SD. *Significant improvement from day 0.

group in the laboratory study (Fig. 2). Peak power output was significantly higher in the reduced training group on day 14 than on day 0 (Fig. 2). In contrast, VO_2 -PPO was unchanged in the control group.

Per cent changes in VO_2 -PPO and time-trial performance in the different groups are listed in Table II. Time-trial performance improved by $2.8 \pm 3.9\%$ in the reduced-training field group but fell by $3.5 \pm 4.8\%$ in the respective control group. As a result, the performance difference between the groups changed by 6.3%.

VO_{2max} , blood lactate concentration at VO_{2max} , muscle glycogen content and submaximal oxygen consumption, heart rate, blood lactate concentrations and ratings of perceived exertion were not different between reduced training and control subjects in the laboratory group on any of the test days (Table III). Nor were there inter-group differences in peak power,

TABLE II. Per cent change in PPO and in time-trial performance in control and reduced-training groups from the start of tapering (day 0) to days 4, 8, and 14 of the reduced training period

	Reduced training		Control	
	PPO	Time-trial performance	PPO	Time-trial performance
Days 0 - 4	3.6 ± 10.2	0.5 ± 5.0	4.5 ± 8.4	1.4 ± 3.4
Days 0 - 8	3.3 ± 11.7	2.0* ± 2.9	-1.4 ± 11.4	-3.1 ± 7.3
Days 0 - 14	5.6* ± 9.2	2.8* ± 3.9	-1.2 ± 8.5	-3.5* ± 4.8

*Significantly different from the per cent change between day 0 and day 4 ($P < 0.05$).

Values are means \pm SD, $N = 5$ in each sub-group. A positive value indicates an improved performance, a negative value indicates a reduction in performance.

mean power, fatigue index or blood lactate concentrations measured after the Wingate test (Table IV) or in maximum isometric tension, maximum speed of contraction, or total work (Table V).

TABLE III. Cardiorespiratory and metabolic measures in the reduced training and control groups in the laboratory trial

Days		0	4	8	14
Maximum oxygen consumption (l/min)	RT	4.5 ± 0.2	4.5 ± 0.2	4.5 ± 0.3	4.6 ± 0.2
	CT	4.7 ± 0.2	4.6 ± 0.2	4.2 ± 0.1	4.3 ± 0.2
Muscle glycogen concentrations (mmol/kg wet weight)	RT	82.6 ± 7.8	79.5 ± 3.2	95.9 ± 13.5	83.7 ± 13.2
	CT	101.57 ± 16.9	5.3 ± 6.0	87.5 ± 5.6	100.6 ± 6.2
Submaximal VO_2 (l/min)	RT	3.3 ± 0.2	3.2 ± 0.2	3.2 ± 0.2	3.2 ± 0.2
	CT	3.3 ± 0.3	3.3 ± 0.1	3.4 ± 0.2	3.3 ± 0.2
Submaximal heart rate (beats/min)	RT	168.4 ± 10.4	161.2 ± 8.2	164.2 ± 10.8	162.2 ± 10.1
	CT	166.41 ± 1.4	156.4 ± 4.7	160.8 ± 3.2	157.4 ± 4.7
Submaximal blood lactate concentrations (mmol)	RT	4.1 ± 0.7	3.7 ± 0.7	3.7 ± 0.6	3.3 ± 0.6
	CT	4.4 ± 0.5	3.4 ± 0.7	3.1 ± 0.9	3.5 ± 1.1
Submaximal ratings of perceived exertion (units)	RT	1.5 ± 0.4	1.3 ± 0.3	1.5 ± 0.4	1.0 ± 0.2
	CT	1.9 ± 0.7	1.9 ± 0.6	1.0 ± 0.7	1.4 ± 0.5

Values are means \pm SD; $N = 5$ in each group.
RT = reduced training group; CT = control group.

TABLE IV. Skeletal muscle power output during the Wingate test and peak blood lactate concentrations in reduced training and control groups in the laboratory trial

Days		0	4	8	14
Peak power (Watts)	RT	1 014.0 ± 27.6	1 084.4 ± 36.1	1 102.4 ± 32.3	1 060.8 ± 60.5
	CT	1 024.5 ± 20.9	1 131.0 ± 56.8	1 140.0 ± 37.9	1 046.3 ± 41.6
Mean power (Watts)	RT	708.5 ± 44.8	698.0 ± 28.8	735.5 ± 27.9	727.9 ± 32.4
	CT	736.0 ± 44.5	718.7 ± 31.0	723.0 ± 38.3	698.9 ± 30.2
Blood lactate concentrations (mmol/l)	RT	10.5 ± 2.0	13.8 ± 1.8	12.7 ± 1.5	13.7 ± 1.2
	CT	10.2 ± 1.2	10.7 ± 1.4	10.3 ± 1.1	10.9 ± 1.0

Values are means \pm SD; $N = 5$ in each group.
RT = reduced training group; CT = control group.

TABLE V. Measures of skeletal muscle contractile function in reduced-training and control groups in the laboratory trial

Days		0	4	8	14
Po (Watts)	RT	172 ± 10	186 ± 10	161 ± 9	176 ± 16
	CT	168 ± 3	184 ± 22	170 ± 5	136 ± 32
V _{max} (m/s)	RT	16.0 ± 0.6	16.0 ± 0.5	17.0 ± 1.3	16.0 ± 0.5
	CT	16.7 ± 0.6	16.8 ± 0.4	16.7 ± 0.5	13.5 ± 3.0

Values are means ± SD; N = 5 in each group.
RT = reduced training group; CT = control group.

Discussion

The important finding of this study was that cycling performance in a field time-trial improved significantly by 2.8% in a reduced-training group over the 14 days of the study, whereas performance became significantly (3.5%) slower in a control group that continued their usual training during the same period (Fig. 1). This decline in performance is difficult to explain, but may have been due to boredom of the subjects with the testing regimen as it also occurred in the laboratory control group, whereas the experimental groups may have remained motivated since they seemed to know intuitively that they had performed better or worse than in their previous test. Alternatively the longer period of normal training load may have resulted in fatigue in the control subjects. PPO (Table II) but not VO_{2max} (Table III) also increased by 5.6% in the experimental group during this period, but was unchanged in the control group (Fig. 2).

Surprisingly, there were no other changes that might explain the superior time-trial performance or the increased VO₂-PPO of the reduced training subjects. Muscle glycogen content, measures of submaximal performance including heart rate, VO₂, blood lactate concentrations and ratings of perceived exertion were not different between reduced training and control groups in the laboratory trial, nor did they change during the duration of the experimental period. Similar results were found for measures of explosive muscle function measured with either the Wingate test or the isometric muscle function testing.

The improved time-trial performance after a period of reduced training in the well-trained cyclists in this study could not be explained by changes in physiological variables measured during submaximal cycling or in short duration tests of skeletal muscle function. Previously, Shepley *et al.*¹⁶ have shown increased citrate synthase activity, increased blood volume and an increase in muscle strength after a period of reduced training during which intensity was kept high. Changes in PPO were in the same direction as those of the time-trial performance (Table II).

In a study by Costill *et al.*⁵ improvements in performance in swimmers after reduced training were attributed to gains in muscular power. In the present study, improvements in both time-trial performance and PPO (muscular power) were found, but the underlying physiological determinant of this change was not elucidated. A similar conclusion was reached by Martin *et al.*¹⁴ who studied changes in cycling performance and isokinetic skeletal muscle function in a group of collegiate cyclists during 6 weeks of high-intensity aerobic interval training, followed by a 2-week taper in which training intensity and volume were reduced substantially. Both interval training and the taper produced increases in cycling performance (15% and 8%, respectively) and in isokinetic skeletal muscle function; but the time course of the changes was different. In particular, cycling performance was greatest after the first week of the taper, whereas isokinetic skeletal muscle function continued to increase throughout the 2-week taper. Martin *et al.*¹⁴ concluded that these findings were consistent with data which show that gains in muscle strength are not tightly linked to changes in performance with reduced training, and hence are not the sole determinant of changes in performance following a period of tapering or reduced training.

The lack of a significant change in VO_{2max} after 2 weeks of reduced training in the current study, despite increases in both PPO and performance, is surprising since an increase in PPO (presumably as a result of a period of recovery from hard training) is often accompanied by an increase in VO_{2max}, because more work can be performed. However, an unchanged VO_{2max} after a reduction in training has also been shown in the studies of Bryntesson and Sinning³ and Hickson and Rosenkoetter.⁸ In both studies VO_{2max} values were unchanged during 15 weeks' training at 33% of the normal training volume. These results and those of the present study confirm the finding that VO_{2max} can be maintained despite a large decrease in training load.

Since no significant differences were found in any of the physiological variables measured in this study, including VO₂, RPE, heart rate during submaximal exercise, resting muscle glycogen, post-exercise blood lactate concentrations, or in measures of *in vivo* skeletal muscle function, it would seem that these variables are not important in mediating performance changes with reduced training. Alternatively, current methods are not sufficiently sensitive to identify small changes that produce measurable changes in cycling performance.

In conclusion, this study shows that the performance of endurance-trained cyclists, like that of swimmers^{4,5} and runners,^{10,11} improves after a short period when training volume is reduced by 40% and training frequency by 34% while training intensity is maintained.

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Heart rate response of young cricket fast bowlers while bowling a six-over spell

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Abstract

Objective. To determine whether fatigue, measured as a percentage of maximum heart rate (% HR_{max}), occurred during a six-over bowling spell in fast bowlers.

Design. Twenty-one young elite or potentially elite fast bowlers (junior group $N = 11$; senior group $N = 10$) were recruited for the study. The bowler's heart rate was recorded during a six-over bowling spell in a turf cricket net. The accuracy of each delivery was recorded, and various anthropometric and flexibility data relevant to the physical demands of bowling were also collected.

Results. The junior (11.6 - 13.3 years) and senior (17.7 - 21.5 years) groups had similar run-up lengths of 15.9 ± 2.0 m and 15.2 ± 2.8 m, respectively. There were no differences between the groups for active and rest time during each over. No changes in the active/rest ratio occurred during the course of the experiment in either group. The average exercise intensity during the six-over spell was the same for juniors ($77 \pm 5\%$ HR_{max}) and seniors ($77 \pm 4\%$ HR_{max}). There were no differences between the dominant and non-dominant arm girths in the juniors, in contrast to the senior group who had a significantly greater dominant arm girth compared with their non-dominant arm girth ($P < 0.0003$).

Conclusion. There was no evidence in this study to limit the number of balls bowled by young fast bowlers to 36 or less, at least based on exercise intensity during bowling. Future studies need to address biomechanical factors during bowling that may predispose the bowlers to injury.

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Introduction

Cricket is a sport that has a 'moderate' injury risk.²¹ Numerous studies indicate that the incidence of injuries in cricket players is increasing rapidly.^{2,3,7,9,16,19,21} In part, the physical demands on cricketers would seem to be increasing. This is because in addition to technical skill, the modern cricketer must possess a high level of fitness, making him susceptible to injuries caused by repetitive training.

Forty-six per cent and 47% of the schoolboy injuries occurred during matches and practices respectively, and occurred fairly regularly throughout the season, with a slight increase during the early and latter part of the season.²⁰ The other injuries (7%) occurred during other nonspecific exercise training. It is known that over-use injuries are more common towards the end of the season.² Of particular relevance is the finding that many more 'adult-type' injuries are occurring in young cricketers, with bowling being the major cause of these injuries. Forty-seven per cent of the injuries sustained by young cricketers occurred to fast bowlers.²⁰ This was greater than in club and provincial cricket (42%).¹⁹ Injuries were predominantly to the lower back and included muscle tears, especially of the hip flexors, adductor longus and rectus femoris muscles. Studies have also shown that the lower backs of fast bowlers are particularly prone to injury, with the common injuries being stress fractures to the third, fourth and fifth lumbar vertebrae (11% in young Australian fast bowlers⁸), spondylosis (50% in Australian fast bowlers¹⁶), pedicle sclerosis, disc degeneration and bulging (21% of Western Australian players assessed⁴).

Damage to the epiphyses around the knee, traction apophysis, and compressive stress to the articular cartilage of the femur and talus were also common in young Australian fast bowlers. Thirty-eight per cent of the bowlers suffered one disabling injury during a single season, with stress fractures of the lumbar and/or sacral vertebrae (11%), and soft-tissue injuries to the back (27%) causing them to miss at least one match.⁷

The high incidence of back injuries in young fast bowlers is not the result of a single aetiological factor, but rather a combination of factors that may predispose these players to injuries. These factors included inherent physical and physiological factors such as postural defects, high physical demands, specific biomechanical demands of different bowling techniques, an escalation in training frequency and the duration of bowling spells

in both practices and matches.^{1, 3, 4, 5, 7, 10, 11, 13, 16, 20}

In order to address the problem of over-use injuries in fast bowlers, guidelines were drawn up for use in Australian cricket in 1989.⁵ In match play these guidelines varied from a limit of two spells of 4 or 6 overs for under-12 and under-16 bowlers respectively, to three spells of 6 or 8 overs for under-19 and senior bowlers respectively. According to the guidelines each spell was to be followed by a 1-hour break for all age groups. In addition, guidelines were developed for the quantity and quality of bowling to be carried out in practices. Other countries, including South Africa, have either accepted these guidelines with or without modifications, without necessarily legislating that these guidelines be enforced. But these guidelines have not been evaluated under modern playing conditions. Furthermore, little performance analysis has been done on cricketers. Consequently the energetics of fast bowling in cricket is not well understood and as a result players and coaches base their training and coaching on beliefs, hunches, trial and error, and the practice of successful players.

Studies on the energy demands of cricket players have shown that bowling in the nets and in a match have an energy cost of 33.5 kJ/min and 22.0 kJ/min, respectively.⁶ The values for batting and fielding were found to be even lower than those for bowling in a match. The estimated gross energy cost of bowling in a test match was 8.85 kJ/m²/min,⁸ while the energy expenditure of young slow bowlers was 7.5 kJ/min.¹⁵ The reason for these values being much lower than the values for the bowlers in the study by Fletcher⁶ is possibly because they were based on a number of assumptions with regard to run-up length.

It is evident that the incidence of cricket injuries, particularly over-use injuries in young fast bowlers, is a growing problem that will continue to escalate unless preventive steps are introduced. Data are lacking on the physical demands of bowling. The present study was conducted to determine the exercise intensity, based on heart rate, of young elite cricket fast to medium-fast bowlers, with the aim of determining whether there is cumulative fatigue measured as heart rate drift during a 6-over bowling spell. These data will contribute to the establishment of recommendations on the maximum number of consecutive overs a young fast bowler should be allowed to bowl. Together with providing a greater understanding of the physical demands of fast bowling, this study will provide coaches and fast bowlers with a better rationale for their training programmes.

Methods

Twenty-one young elite or potentially elite fast bowlers who were invited to participate in specialised training by the Eastern Province Cricket Board were selected for the study. These bowlers were divided into a junior group ($N = 11$), with the bowlers all under 14 years of age, and a senior group ($N = 10$), with the players all aged between 17 and 22 years of age.

The following anthropometric data were collected according to the descriptions of Ross and Marfell-Jones:¹⁷ mass (kg), stature (cm), biceps, triceps, subscapular, supra-iliac, abdominal, front thigh and medial calf skinfolds (mm) and right and left relaxed arm girth (cm). Body fat content was estimated as the sum of the seven skinfold measurements. The following tests were used to measure the flexibility of the subjects:¹³

1. Shoulder and wrist flexibility were assessed with the subject assuming a face-down position. The subject held a ruler with the arms straight and shoulder-width apart. The subject raised the ruler horizontally as high as possible while keeping his chin on the floor and elbows straight. The tester measured with a metre stick to the top centre level of the ruler. The tester used the metre stick to measure the subject's arm length from the acromial process to the tip of the middle finger. The best of three lifts was subtracted from the arm length to derive the score. The closer the arm lift gets to arm length, the better the score.

2. Back and hamstring flexibility were tested using the modified sit-and-reach test. The subjects removed their shoes and sat with their knees fully extended and feet shoulder width apart, placed against a 30 cm box. A ruler was positioned and held so that the 'zero' end of the ruler touched the extended fingers of the subject. The subject reached forward, palms down, along the measuring ruler four times and held the maximum reach on the fourth trial for 1 second. The score was recorded as the furthest point reached on the fourth trial.

3. Trunk and neck flexibility were assessed with the subject lying face down with his hands at the small of his back and an assistant holding his hips down. The subject raised upward, extending the trunk and neck as high as possible with the neck in the neutral position, and held the position for 3 seconds. The tester measured the highest point reached by the tip of the subject's nose. The distance between the tip of the nose and the seat of the subject's chair was measured to give his trunk and neck length. The best score of three lifts was subtracted from the trunk and neck length to give the score. The lower the score the better the flexibility.

Thereafter the subjects were allowed to warm up before starting the bowling trial. In order to simulate match conditions the bowler had to bowl at a batsman. After each delivery a trained observer gave a rating (1 - 5) for the line (the direction of the ball in relation to the batsman) and length (the proximity of where the ball bounced in relation to the batsman) of the delivery. The subjects worked in pairs, with one subject bowling and the other subject simulating fielding during the over. This subject simulated fielding on the boundary by walking in with the bowler for each delivery. A ball was also thrown for him to field and return during the over. After the completion of an over the subjects swapped position. This continued until both bowlers had each bowled 6 overs.

The subject's heart rate (HR) was recorded continu-

ously for the 6-over bowling spell in a turf cricket net by means of short-range telemetry using the Polar Sport Tester (Polar Electro, Finland). The HR monitor was placed on the subject before he commenced his warm-up routine. The recording of the HR began just before each subject's 6-over bowling spell. The receivers were programmed to measure and store the HR readings every 5 seconds.

The start of the collection of the HR data was synchronised with a stop watch in order to relate the HR to specific events during the data collection phase. These included the start and completion of each over, the start of the run-up, the completion of the follow-through, when the ball was fielded and any other tasks that the bowler performed. At the end of the testing session these data were downloaded into a computer using an interface and software purchased from the manufacturers (Polar Electro, Finland).

Each subject's exercise intensity was calculated for each over and expressed as the average HR as a percentage of the subjects maximum HR (% HR_{max}). The subject's maximum HR was determined in a field test immediately after the bowling experiment. Subjects were required to run 400 m at approximately 80% effort. Following a short rest subjects were instructed to run 800 m at maximal effort. The highest HR elicited during the test was defined as the maximal HR.¹²

Statistical analysis

All values are expressed as mean and standard deviation (SD). A Student's *t*-test was used to determine differences between the two groups of fast bowlers. A paired *t*-test was used to determine differences within a group. An analysis of variance with repeated measures was used to determine differences over time. Statistical significance was accepted for *P* values less than 0.05.

Results

The average temperature during the testing period, recorded at the Port Elizabeth meteorological office, was 22.3°C (ranging from 17.5°C to 22.8°C), with a wind speed of 18 knots (36 km/h) blowing across the run-up.

The general characteristics of the subjects are

TABLE I. General characteristics of the junior (*N* = 11) and senior subjects (*N* = 10)

Variables	Junior		Senior	
	Mean	SD	Mean	SD
Age (years)	12.7	0.5	19.0	1.4
Mass (kg)	45.6	5.2	78.0	10.6
Stature (cm)	158.3	5.8	184.9	5.3
Sum of 7 skinfolds (mm)	57.4	13.2	76.8	21.6
Dominant arm girth (cm)	22.4	1.2	29.3	2.8*
Non-dominant arm girth (cm)	22.0	1.5	28.4	2.8

* Dominant arm v. non-dominant arm *P* < 0.0003 (seniors). All the variables of the juniors are significantly lower than those of the seniors (*P* < 0.00001).

shown in Table I. The ages of the junior group ranged from 11.6 to 13.3 years and the seniors from 17.7 to 21.5 years. The majority of the group were right handed (juniors 8/11, seniors 9/10). There were no differences between the dominant and non-dominant arm girths in the juniors. In contrast, the arm girth of the dominant arm was 0.89 cm ± 0.50 cm greater than the non-dominant arm (*P* < 0.0003) in the senior group.

TABLE II. Flexibility characteristics of the junior (*N* = 11) and senior subjects (*N* = 10)

Variables	Juniors		Seniors	
	Mean	SD	Mean	SD
Shoulder and wrist flexibility (cm)	33.5	11.7	41.1	11.9
Back and hamstring flexibility (cm)	44.5	4.5	47.1	10.4
Trunk and neck flexibility (cm)	16.2	8.0	30.6	8.9*

* Significant difference (*P* < 0.001).

The flexibility measurements are shown in Table II. The shoulder and wrist, and back and hamstring flexibility were similar between groups. However, the junior subjects were significantly (*P* < 0.001) more flexible in the trunk and neck flexibility test than the senior subjects (16.8 ± 8.0 v. 30.6 ± 8.9 cm).

The length of the run-up to the bowling crease was similar in each group (15.9 ± 2.0 v. 15.2 ± 2.8 m) (juniors v. seniors). The active and rest time comprising each of the 6 overs bowled in the study are shown in Table III. There was no difference between groups for active and rest time. Furthermore, there were no changes in the active/rest ratio during the course of the experiment in either group.

TABLE III. The active and rest time (seconds) during each of the 6 overs of the junior (*N* = 11) and senior (*N* = 10) subjects

Variables	Juniors		Seniors		
	Mean	SD	Mean	SD	
Over 1	Work (s)	156	31	150	19
	Rest (s)	254	75	246	26
	Work: rest	0.64	0.26	0.61	0.08
Over 2	Work (s)	143	24	152	20
	Rest (s)	229	47	238	41
	Work: rest	0.64	0.10	0.65	0.10
Over 3	Work (s)	153	46	152	20
	Rest (s)	226	50	254	32
	Work: rest	0.68	0.17	0.60	0.10
Over 4	Work (s)	150	38	152	23
	Rest (s)	220	34	226	23
	Work: rest	0.69	0.19	0.68	0.12
Over 5	Work (s)	146	18	156	21
	Rest (s)	219	24	225	22
	Work: rest	0.67	0.07	0.70	0.13
Over 6	Work (s)	143	21	156	22

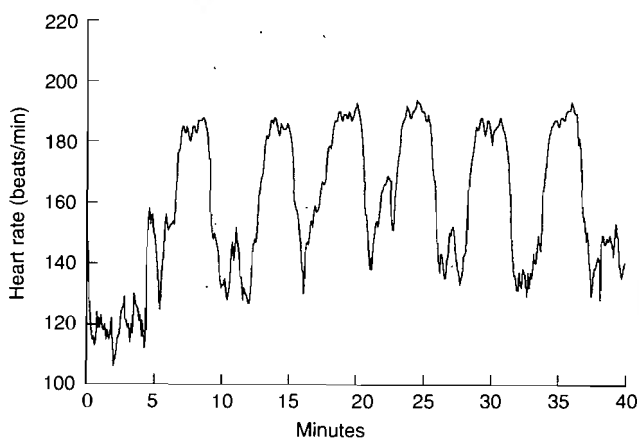


Fig. 1. A typical heart rate response during the 6-over trial.

TABLE IV. The average HR (beats/min) and exercise intensity (%HR_{max}) for each of the 6 overs for the junior (N = 11) and senior (N = 10) subjects.

Variables	Juniors		Seniors	
	Mean	SD	Mean	SD
Over 1	Average HR	153	149	11
	% HR _{max}	74	75	5
Over 2	Average HR	157	152	10
	% HR _{max}	76	76	5
Over 3	Average HR	159	153	10
	% HR _{max}	77	77	5
Over 4	Average HR	160	155	10
	% HR _{max}	78	78	4
Over 5	Average HR	161	155	10
	% HR _{max}	79	78	4
Over 6	Average HR	162	156	10
	% HR _{max}	79	78	4
Average	Average HR	159	153	10
	% HR _{max}	77	77	4

A typical HR response of a subject during the 6-over trial is shown in Fig. 1. The average HR and exercise intensity (expressed as %HR_{max}) for each of the 6 overs is shown in Table IV. The maximum HR determined in the field test was higher ($P < 0.01$) for juniors (206 ± 5 beats/min) than for seniors (199 ± 7 beats/min). However, throughout the 6 overs the average HR and %HR_{max} were similar between groups and did not change as the experiment progressed.

The rating score for each of the 36 deliveries (6 overs) for the junior and senior players are shown in Fig. 2. There were no differences between groups or over the course of the 36 deliveries.

Discussion

The primary finding of this study was that the exercise intensity of the junior and senior fast bowlers was similar, showing no indication of cumulative fatigue as the bowling spell progressed. In addition, the accuracy of each delivery did not change in either group over the 36 balls. Therefore, based on exercise intensity and

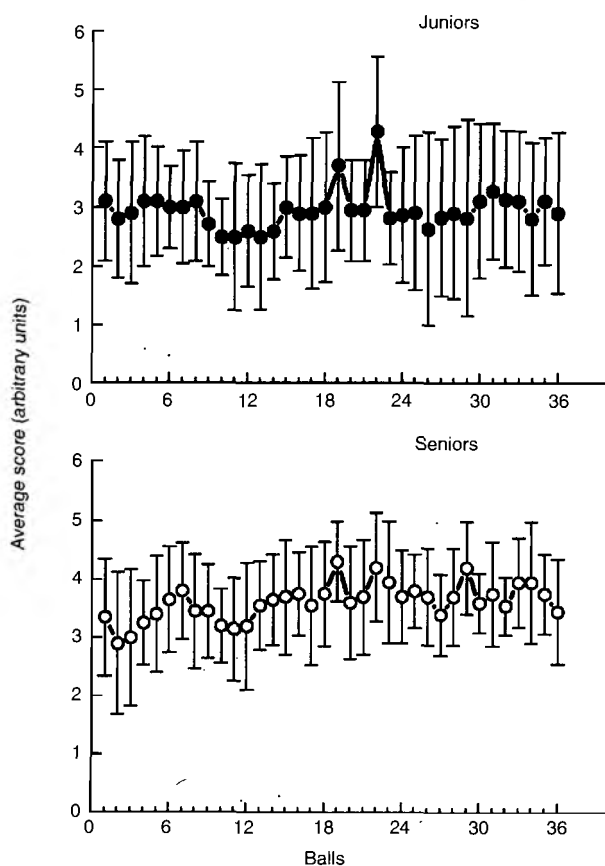


Fig. 2. Rating score for each of the deliveries of the 6 overs for the junior (N = 11) and senior (N = 10) subjects.

accuracy of each delivery, there is no reason to suggest that there was any acute fatigue affecting bowling performance after 36 deliveries.

The dominant arm girth of the senior players was greater than their non-dominant arm girth, which was in contrast to the junior players who had similar dominant and non-dominant arm girths. The hypertrophy of the dominant arm can possibly be attributed to the physical demands of the bowling action. Another study¹⁸ on cricket bowlers showed that calf girth measurements were greater in the leg opposite the bowling arm of each player. This leg is used as an effective lever during the bowling action.¹⁸ The hypertrophy of the trunk and neck in the senior players in this study, supported by the findings of Stretch,¹⁸ suggests that the bowling action places stresses on the body that result in asymmetrical adaptations. Although it is tempting to conclude that these asymmetrical adaptations place the bowler at risk of injury, this will have to be determined in future studies.

In summary, there are no grounds (based on the possibility of fatigue-induced injury) for restricting the number of consecutive overs bowled to less than 6. However this study supports the concept of limiting the number of overs to 6 as it shows that there is no associated fatigue. The decreased flexibility of the

trunk and neck and the increase in dominant arm girth in the older bowlers suggests that adaptations, possibly as a consequence of the repetitive loading during the bowling action, take place. Whether these adaptations increase the bowler's risk of injury remains to be determined. Further studies of bowlers bowling more than 6 overs need to be conducted in order to determine whether acute fatigue is measurable using the techniques in this study. Furthermore, studies need to be conducted to investigate biomechanical aspects of bowling to determine whether any limits need to be placed on bowling at practices and during matches, with the overall aim of reducing the risk of injuries associated with bowling.

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The consequences of the female athlete triad in a mature woman runner: a case report

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Introduction

The female athlete triad is a triad of disorders observed in adolescent and adult female athletes and includes disordered eating, amenorrhoea and osteoporosis,¹¹ the severity of which is determined primarily by prior menstrual history.⁹ It is not restricted to elite athletes but includes also physically active girls and women participating in a wide variety of sports. It has recently been shown^{5,10} that if regular menses are resumed there is an initial increase in bone mineral density (BMD);^{2,4,7} however even after several years it is still significantly lower than in women who menstruate regularly, and our previous study¹⁰ suggests that in women over 30 years of age there is little chance of significant increase in BMD.

Case report

A 29-year-old female runner was investigated at baseline, 1 year after she had started training for marathons, and again 3 years later. She participated in a 56 km ultramarathon three times in the period between the two tests, and her best time was 4 hours and 42 minutes. Her exercise schedule consisted of running five times a week for approximately an hour at a time. When training for a marathon competition her weekly running distance was 75 - 95 km. She also participated in aerobic dance exercise four times a week and circuit weight training three times a week. She did not change her training significantly between tests 1 and 2.

Information such as age at menarche, the estimated number of periods every year since menarche, the use of oral contraceptives, fertility drugs or hormone injections, parity and total months of breast-feeding, was collected and used to calculate a modified menstrual history index (MHI).⁹ She had started menstruating at the age of 14 years.

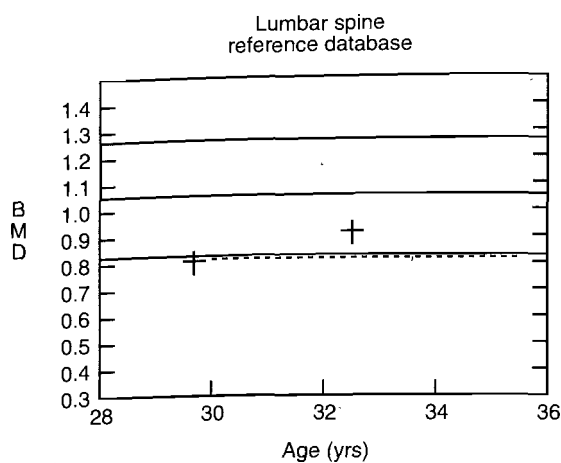
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At baseline she was amenorrhoeic and had experienced on average 2 periods per year for the preceding 3 years. Before that she had been either oligomenorrhoeic (3 years) or amenorrhoeic (6 years) since the age of 18. Her MHI at baseline was 6.50 periods/year. Her menstrual irregularity before baseline coincided with a rapid loss of weight — 38 kg in 21 weeks, achieved through dieting. She was 170 cm tall and had a body mass of 58 kg (her lowest weight as an adult) at baseline and 60 kg at follow-up. The most she had ever weighed as an adult was 96 kg (body mass index (BMI) = 33.2 kg/m²) a year and a half before baseline. At baseline her per cent body fat was 32%, but it had dropped to 26% at follow-up. Lean body mass had increased from 68% to 74% in this time, probably as a result of the inclusion of more weight training in her exercise routine. MHI had also increased to 9.25 at follow-up due to the regulation of her menstrual cycle. She has two children aged 12 and 8 years and had breast-fed for a total of 5 years. She had taken oral contraceptives for only 2 years after giving birth to her second child.

Previous dairy product intake was reported as the estimated number of portions consumed per week during four stages of life. One portion was equivalent to one cup of milk, 175 ml of yoghurt, a rounded scoop of ice-cream, a 250 g tub of cottage cheese, or a cheese meal or sandwich. As a child her dairy intake consisted of canned milk diluted with water. She consumed an estimated 10 portions of dairy per week during her junior school years, 21 portions/week during high school, and since leaving school between 2 and 4 portions/week until after baseline, when her intake increased. Calcium intake at test 1 was 708 mg and at test 2, 548 mg. Total energy intake was 4 492 kJ/day at test 1, and 3 940 kJ/day at test 2, constituting 48% and 43% of the recommended daily allowance (RDA) for adult women. She took no dietary supplements or regular medication except for treatment for insomnia, for which condition she was using flunitrazepam (Rohypnol, Roche).

A Hologic QDR-1000 (version 4.20) dual-energy radiograph bone densitometer was used to measure BMD of the lumbar spine (LS) and left proximal femur (F). The repeatability of this method has been described previously.¹⁰ LS BMD was 0.796 g/cm² at baseline, increasing to 0.893 g/cm² at follow-up, an annual increase of 4% (Fig. 1), but a total of 12% in 3 years. The T-score for the second scan was -1.40 while



Region	Rate of change	±SD	% Change	±% SD
L1 - L4	+0.0318	0.0042	+3.99	0.52

Fig. 1. Lumbar spine BMD of the subject in this case report is indicated relative to a reference database by two crosses corresponding to age 29.6 and 32.7 years. The table indicates that the percentage increase of BMD was 3.99%/year.

the Z-score was -1.37 (reference curve for American females, Hologic QDR-1000, October 1984). BMD of L1 was just below the fracture threshold of 0.827 g/cm^2 and L 4 showed significant osteopenia. F BMD was normal at baseline and follow-up (1.030 g/cm^2 and 1.122 g/cm^2 respectively). The T-score for the second scan was $+1.13$, while the Z-score was $+1.16$ (reference curve for American females, National Health and Nutritional Examination Survey (NHANES) update). BMD of the femoral neck was 0.934 g/cm^2 at test 1 and 0.978 g/cm^2 at test 2. Since the first scan, all areas in the left hip had increased in BMD. The annualised changes were: neck of the femur: $+1.53\%$ per year, greater trochanter: $+3.41\%$ per year, inter-trochanteric space: $+2.67\%$ per year and the total proximal femur: $+2.92\%$ per year. All the areas of the hip had a positive T-score at the second test.

Discussion

The annualised changes in BMD of this subject were substantial despite her age. This provides further evidence of the positive effect of the resumption of regular menses on BMD in a subject with a severe history of menstrual irregularity combined with a bad profile of other risk factors for osteoporosis. However, BMD of the lumbar spine, which is predominantly trabecular bone and therefore more sensitive to hormonal changes, was still significantly lower than that of age-matched normals (86%) at test 2. A follow-up investigation is necessary to see whether BMD will continue to increase or whether, as found by Keen and Drinkwater⁵ and Micklesfield *et al.*,¹⁰ it will eventually plateau before reaching the level of age-matched normals.

In this case low lumbar spine BMD had a multifactorial origin, including lifelong lower-than-recommended calcium intake, extreme weight loss (though no clinical treatment for an eating disorder) and a substantial history of amenorrhoea and oligomenorrhoea, even before starting to run later in life. Her menstrual irregularity was associated at times with breast-feeding, extreme weight loss or training for an ultramarathon. At baseline, the subject was informed of the status of her bone health and advised of the lifestyle factors that contributed to this. She desisted from further weight loss and decreased year-round running training, although not when training for an ultramarathon (approximately 6 months per year). Her menses regularised spontaneously within 1 year of test 1, coinciding with the removal of an ovarian cyst. Regular menses probably exerted a large influence on the gain in BMD in the subsequent 3 years. However, regular weight training may also have contributed. Heinrich *et al.*⁸ have suggested that weight training may be a better stimulus for improving bone status than running.

The subject was 32 years of age at test 2. Although several studies would predict that by this age peak bone mass would already be achieved,^{1,8} other studies would infer that bone mass could increase up to the age of 34 years.⁶ If lumbar spine BMD continued to increase at the present rate, i.e. 4% /year, this subject would reach the mean level of 'young normals' in another 4 years. However, our other longitudinal data¹⁰ indicate a $< 1\%$ /year increase in previously irregularly menstruating women with a mean age of 36.1 ± 5.05 years. These subjects had already menstruated regularly for an average of 11.7 ± 7.9 years, and were on average older than this subject. It is not known whether the better rate of improvement of BMD in this subject was related to age, severity of previous irregularity at baseline, or weight training, and whether improvement will continue at the same rate or slow down.

Conclusion

This case illustrates that a large initial increase in lumbar spine BMD with the resumption of menses is possible even at a relatively older age than previously reported. The detrimental effect of menstrual irregularity on BMD is not restricted to the very lean, elite athlete, but should be of concern to runners of any standard, particularly those with multiple risk factors for osteoporosis such as those in this case study.

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Position Statement: Diabetes Mellitus and Exercise International Sports Medicine Federation (FIMS)

The following Statement was approved by the FIMS Executive Committee, 26 October 1996

Introduction

Diabetes mellitus is a common metabolic disease characterized by insulin insufficiency resulting in impaired ability to transport glucose across the cell membrane for its subsequent oxidation. Also, muscle and liver glycogen re synthesis, triglyceride synthesis in adipose cells, inhibition of their breakdown (antilipolytic effect), as well as protein synthesis and storage (anabolic effects) are being impaired. Insulin insufficiency thus leads to metabolic disturbances leading to such common symptoms as fatigue, weakness, weight loss, hunger, overeating, polyuria, and signs of glycosuria, and ketosis.

Though being clinically silent for many years, diabetes mellitus often leads to serious pathological complications of various organ systems (eyes, kidneys, peripheral nerves, coronary and peripheral arteries) which may substantially impair quality of life and reduce life expectancy.

There are two distinct forms of diabetes, termed insulin dependent diabetes mellitus (IDDM) and non insulin dependent diabetes mellitus (NIDDM).

IDDM is an auto-immune disease in which the body attacks and ultimately destroys insulin producing pancreatic beta-cells. In addition to a genetic component, evidence supports a viral infection triggering an auto-immune process either due to similarities with beta cell protein or sensitization to destructed beta-cells.

The key pathogenetic factor of NIDDM is relative insufficiency of insulin due to insulin resistance and/or defective insulin secretion. Insulin resistance is often associated with hypertension, lipid disturbances, and obesity. Apart from genetic dispositions, diet and obesity, animal experiments as well as epidemiological data suggest that a lack of physical activity may also contribute to a relative deficiency of insulin.

Diabetes may be precipitated by, or a similar syndrome brought about by endocrine disorders (e.g., hypercortico steroidism, acromegaly, hyperthyroidism, pheochromocytoma), drugs (e.g., glucocorticoids, thyroid hormones, contraceptives, thiazides), and pancreatic or liver disease.

Rationale for exercise in prevention and therapy

Due to an insulin-like effect on muscle contraction (an increase of membrane permeability to glucose) exercise has a potential to increase insulin sensitivity, lower blood glucose and increase its utilization. Improved glucose tolerance positively influences the glycemic profile that can be detected by lower concentration of glycosylated hemoglobin. A better glycemic profile may postpone and reduce the risk of late complication. Since

this effect is rather short-lived, regular frequent exercise sessions are needed to maintain such a benefit.

Also reducing body fat due to the increased energy expended and its effect on the basal metabolic rate may indirectly but significantly decrease insulin resistance.

In addition to obesity, exercise has the potential to favourably alter other risk factors of cardiovascular disease, namely elevated blood lipids and hypertension. In this way an already increased risk of coronary heart disease (3 times higher than general population) may be reduced.

Last but not least, exercise may reduce psychological stress, positively influence a feeling of well being, and improve quality of life.

Exercise guidelines

Clearance by a knowledgeable physician is recommended prior to the initiation of an exercise program. In addition to a general assessment, screening should include an exercise stress test to detect latent cardiovascular disease. Requisites include an absence of ketoacidosis and glycemia under 300 mg %. When late complications are evident, such as hypertension or renal impairment, the risks and benefits should carefully be considered.

During the initial stages of an exercise program, close medical supervision which includes blood glucose monitoring is strongly recommended in order to adjust diet and medication (insulin or PAD doses) to the exercise altered metabolic situation.

Modes of exercise

Aerobic activities carried out at moderate intensity such as brisk walking, cycling, jogging, running, or cross country skiing are preferred modes of exercise. Since the majority of diabetics are obese, non weight bearing exercise like cycling and swimming may pose less stress on the locomotor system and contribute to better compliance. General daily activities of a habitual nature are encouraged in addition to the exercise sessions.

In the past, resistance exercise has not been recommended because of the potential for a dangerous increase in blood pressure, especially in those with vascular complications. Recent findings indicate that appropriate forms of resistance exercise are safe and may potentiate the positive effects of aerobic exercise. A circuit training approach aimed at all major muscle groups is recommended. The resistance should allow 10 to 12 comfortable repetitions.

Intensity of exercise

The exercise intensity should be between 50 and 70% of VO_{2max} . Higher intensities excessively activate the sympathoadrenal system with subsequent increase of glycemia. With caution, heart rate can be used as an indicator of intensity. In patients with autonomic neuropathy, heart rate may not accurately reflect exercise intensity. As an alternative, perceived exertion of METs (metabolic equivalents) should be used for the exercise prescription.

Duration of exercise

Exercise sessions between 20 and 60 minutes are recommended. Less than 20 minutes yields little cardiovascular benefit, longer exercise tends to increase the risk of hypoglycemia.

Frequency of exercise

Daily exercise is suggested because such an approach enables easier insulin adjustment and diet planning. A more realistic and practical goal may be 4 to 6 sessions a week.

Practical remarks

- Patients should be educated about the effects and potential risks of exercise, namely hypoglycemia.
- The participants should wear identification indicating their diabetic condition and should exercise with a knowledgeable partner in case of hypoglycemia including loss of consciousness.
- When possible exercise should be performed at the same convenient time with similar intensity and duration.

- Because of the pro insulin effect of exercise, insulin dependent diabetics should reduce insulin doses by 20% or adequately increase food intake upon initiating an exercise program.
- To avoid hypoglycemia a small carbohydrate snack should be eaten 30 minutes prior to exercise. During more prolonged activity a 10 g carbohydrate snack (fruit, fruit juice, or soft drink) should be ingested for each 30 minutes of exercise.
- Pay careful attention to the feet of the exercising diabetic patient. Loss of sensation due to neuropathy and/or impaired peripheral circulation increases the risk of injuries. Good footwear and careful foot hygiene are essential to avoid injuries like calluses, corns and blisters that may lead to serious complications.
- Warm up and cool down periods should be an integral part of an exercise program.

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IOC OLYMPIC
PRIZE
ENDOWED BY
PARKE-DAVIS

The IOC Olympic Prize

by
Benno M Nigg

THE IDEA OF THE IOC OLYMPIC PRIZE

Movement and mobility are among the most precious aspects of human life. Imagine a person who cannot move! A person who cannot play golf with friends, cannot go for a hike with family or friends, or cannot visit the next-door neighbours for a chat. Mobility and movement are extremely important for the quality of life of humans. Without the ability to move, life can be very difficult.



Movement is important in all situations of life, from childhood to old age to high performance athletics.

Mobility and movement are equally important for children, adolescents, athletes and the elderly. A young child learns to move and engraves movement patterns into the motor control system. If these patterns are correct, early degenerative disease such as osteoarthritis may be avoided.

Adolescent girls and boys strengthen their muscles, bones, ligaments and tendons by providing the necessary stimuli during movement, exercise and sport. Bone formation in girls, for instance, is maximal during this time and a 'bone bank' may be established due to appropriate sport activities, reducing the risk of osteoporosis.

Athletes expose their bodies to high and repetitive loading situations. This loading may cause damage to the body. However damage can be avoided if training and equipment are well controlled.

Elderly people want to enjoy their retirement age by being physically active, playing games, and spending

time with their grandchildren. To do so, they need to be mobile.

Consequently, understanding the factors influencing mobility and movement and the resultant loading of the human body is important. Scientific research studying movement, exercise and sport can contribute substantially to the improved understanding of mobility and movement. With the increased life expectancy of humans, such research is growing in importance.

The activities of the International Olympic Committee (IOC) centre around all aspects of movement, exercise and sport. The IOC is interested in high performance sport, physical activities for children, adolescents, adults and the elderly, exercise and sport in developing countries, history of exercise and sport, health and well-being due to physical activity and sport, and many other facets of movement, exercise and sport.

The IOC Medical Commission was and is often in the limelight because of athlete doping cases. However, the IOC Medical Commission has many other less well-known activities. Thanks to the initiative of its Chairman, Prince Alexandre de Merode, the IOC Medical Commission has many groups that concentrate on the positive aspects of movement, exercise and sport. In 1987, one such group, the Subcommittee for Biomechanics and Physiology, wanted to somehow acknowledge the importance of science related to movement, exercise and sport.



Prince Alexandre de Merode,
Chairman of the
IOC Medical Commission

The discussions of the Subcommittee for Biomechanics and Physiology resulted in two major project proposals. First, to establish an IOC World Congress for sciences related to movement, exercise, and sport; and second, to establish a highly prestigious prize for science related to movement, exercise and sport, namely the IOC OLYMPIC PRIZE.

Under the leadership and guidance of Prince Alexandre de Merode, chairman of the IOC Medical Commission, selected members of this Subcommittee went to work to develop the two ideas. Dr Charles Dillman provided leadership for the IOC World Congress and acts as scientific chair of all IOC World Congresses. Dr Benno M Nigg provided leadership for the IOC Olympic Prize and acts as the chair of the Selection Committee for the IOC Olympic Prize.

The first IOC World Congress was organised by Dr Charles Dillman in 1989 in Colorado Springs. The development of the IOC Olympic Prize took longer to arrange, since a sponsor had to be found. The initial contact with Parke-Davis, a Warner-Lambert division,

was established in 1992 following unsuccessful initial contacts and discussions with several other world-leading companies. After several meetings between representatives of Parke-Davis (Mr Wayne Dickerson) and the IOC Medical Commission (Drs Patrick Schamasch, Richard Nelson, Charles Dillman, and Benno M Nigg), an agreement between Parke-Davis (Lodewijk de Vink, president and COO of Warner-Lambert) and the IOC Medical Commission (Prince Alexandre de Merode) was signed and announced during the 1994 Olympic Winter Games in Lillehammer. Currently, Parke-Davis is the exclusive sponsor of the IOC Olympic Prize and all other functions of the IOC Medical Commission which relate to movement, exercise and sport sciences (MESS). The IOC Olympic Prize is awarded each Olympic Game year.

The IOC Olympic Prize is an exciting development for the field of sciences related to movement, exercise and sport (MESS) and for anyone who loves and supports mobility and longevity.

THE IOC OLYMPIC PRIZE

The IOC Olympic Prize honours important findings resulting from outstanding basic and/or applied research related to human movement, exercise and/or sport. These findings must represent a significant innovation, contribute to the betterment of humankind, and have significant impact upon science, health and/or society.



The IOC Olympic Prize consists of

- a gold medal
- a diploma of excellence
- a cash award of \$US 500 000

The IOC Olympic Prize is awarded for work in four areas of science: medical, biological, physical, and psychological. Examples of research to be considered include: (a) the understanding of the healthy development of the human body and its main components, (b) the effect of exercise on health, wellness and quality of life, (c) the prevention of injuries in movement, exercise and sport, and (d) the improvement and optimisation of physical performance through enhanced understanding of the functioning of the human body in all age groups.

The winner is announced during a banquet in New York approximately 6 to 8 weeks before the start of the Olympic Summer or Winter Games. The gold medal is awarded during the Opening Ceremony of the IOC Session before the Games.

EFFECTS OF THE IOC OLYMPIC PRIZE

The IOC Olympic Prize was initially (for 1996 and 1998) US\$ 250 000, but will be US\$ 500 000 for the year 2000. This substantial sum, the public announcement of the winner, and the award ceremony have substantial influence on the development of sciences dealing with movement, exercise and sport. Specifically, the IOC Olympic Prize:

- improves the recognition for research on movement, exercise and sport
- attracts established scientists to study these important questions.
- attracts brilliant young scientists into the study of movement and mobility.

THE FIRST WINNERS

The first prize, the 1996 IOC Olympic Prize, was awarded to:

*Dr Jeremy N Morris and
Dr Ralph S Paffenbarger Jr*

for their pioneering studies demonstrating how exercise reduces the risk of heart disease. The research findings of Drs Morris and Paffenbarger changed the practice of medicine and inspired the fitness revolution. The ground-breaking work of these two leading epidemiologists has brought respect to research in the area of health and fitness and inspired additional studies that have contributed enormously to establishing the relationship between physical activity and a reduction in the incidence of coronary heart disease.

Dr Morris was the first epidemiologist to offer scientific support for the (at that time revolutionary) hypothesis that regular physical activity reduces the risk of coronary heart disease.

Dr Paffenbarger was the first to study risk factors and life habits of male graduates of the University of Pennsylvania and Harvard University. He found that physical activity is a relevant factor in reducing the risk of hypertension, non-insulin-dependent diabetes, some forms of cancer, and premature death in general.



The winners of the first IOC Olympic Prize, Drs Morris (left) and Paffenbarger (middle), with the winner of the second IOC-Olympic Prize, Dr Woo (right).

The second prize, the 1998 IOC Olympic Prize, was awarded to:

Savio LY Woo, PhD

for his pioneering contributions to the understanding of the properties of connective tissues, the effects of exercise on tissue properties, and the possibilities for repair of injured tissues. His work had a significant effect on basic research in this area as well as on the medical treatment of ligament injuries, injuries that occur frequently in physical activities. A large number of individuals benefited directly from his research.

A more detailed description of the research work of these prize winners will be presented in one of the next publications on the IOC Olympic Prize.

TIMETABLE FOR THE IOC OLYMPIC PRIZE 2000

- December 1998
Meeting: Selection Committee in Lausanne.
Finalising the format for nominations for the 2000 Prize.
- March to June 1999
Information on nomination procedure published in scientific journals and newsletters.
- September 1, 1999
Deadline for submission of nomination packages to the headquarters of the IOC Medical Commission in Lausanne, Switzerland.
- August 2000
Announcement of winner during a special IOC Olympic Prize Function in New York.
- September 2000
Medal Ceremony during the Opening Ceremony of the IOC Session in Sydney, Australia.

ADDITIONAL INFORMATION

Additional information concerning the IOC Olympic Prize can be found at the IOC Olympic Prize web site http://www.olympic.org/FAMILY/ioc/medical/olyprize1_e.html

The Parke-Davis IOC Olympic Prize web site http://www.parke-davis.com/version_4/iocprize.html

Further information can be received from:

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IOC World Congress on Sport Sciences

by
Charles J Dillman

In 1988, the IOC Medical Commission decided to develop a series of World Congresses on the scientific aspects of sport that would further the growth of this young scientific field. The First Congress was conducted in Colorado Springs in 1989, with subsequent programmes being organised in Barcelona (2) 1991, Atlanta (3) 1995, and Monaco (4) 1997.

The purpose of the World Congress is to provide a forum for leading scientists to exchange ideas about current research and to disseminate new information about human performance to practitioners who are involved in developing athletes for international and Olympic competitions. The field of sport sciences is segmented into four subdisciplines, namely medical, biological, physical and psychological sciences.

The next programme, the Fifth IOC World Congress for the Science of Movement, Exercise, and Sport, will be held in Sydney, Australia, from October 31 to November 5 in 1999.



FIFTH IOC WORLD
CONGRESS
ENDOWED BY
 PARKE-DAVIS

FIFTH IOC WORLD CONGRESS FOR THE SCIENCE OF MOVEMENT, EXERCISE, AND SPORT

Endowed by Parke-Davis
In conjunction with the Australian Conference
of Science and Medicine in Sport

31 October - 5 November, 1999

Sydney Convention and Exhibition Centre,
Sydney, Australia

The IOC World Congress will combine the very best in science and medicine related to movement, exercise and sport with the warmth and hospitality of Sydney — the host city of the 2000 Olympic and Paralympic Games.

The International Olympic Committee's Medical Commission, the Sydney Organising Committee for the Olympic Games, and Sports Medicine Australia invite you to attend this special Congress.

Under the theme of 'The Science and Medicine of Skilled Performance: Optimisation, Injury Prevention and Rehabilitation', the world's leading exercise and sport scientists and practitioners will present their research — theoretical, applied and clinical — in a spectacular setting, namely the Sydney Convention Centre in beautiful Darling Harbour. Further highlights include the Opening Ceremony/Cocktail Party, Congress Dinner, Olympic Venue Tour, Sports Afternoon, and optional tours in and around Sydney.

The Congress also offers a unique opportunity for sport and team healthcare professionals to meet representatives of SOCOG's Medical and Doping Control Programmes to discuss planning for the Sydney 2000 Olympic and Paralympic Games.

PROGRAMME HIGHLIGHTS

Professor Savio Woo, PhD, from the Muscle Research Centre, University of Pittsburgh and winner of the prestigious IOC Olympic Prize, will give the opening presentation at the Congress.

Keynote and Invited Speakers

Professor Ed Coyle, USA
Professor Bente Pedersen, Denmark
Professor Cy Frank, Canada
Professor Richard Lieber, USA
Professor Joachim Mester, Germany
Dr Jos de Koning, Netherlands
Professor Simon Gandevia, Australia
Professor Lew Hardy, UK

Symposia

Articular cartilage repair
Keeping people physically active (1): Motivation through the lifespan
Strategies to enhance fatigue resistance

Research on muscle mechanics
Ethics in sport
Women in the Olympic Games
Clinical and physiotherapy symposia

Workshops

Application of muscle mechanics in sport
Keeping people physically active (2): Public health programmes
Supplements to enhance performance: The evidence?
Biomechanics feedback for the elite athlete
Workshops in sports medicine, sports physiotherapy, sports podiatry and sports dietetics.

Free Papers

Oral, video and poster presentations in the disciplines of medical, biological, physical & behavioural sport sciences

Parke-Davis Symposium

The Cardiovascular Dysmetabolic Syndrome: Diabetes/Insulin Resistance, Hypertension and Hyperlipidaemia taking place on Sunday 31 October will be of particular interest to internists and general practitioners.

Important Dates

Abstract application forms & abstracts due by 15 May 1999
Early Bird registration closes 30 June 1999
Accommodation Bookings by 17 Sept 1999

Further Information on Congress

Congress Secretariat,
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Visit the official Sydney 2000 Olympic Games web site:
<http://www.sydney.olympic.org/>