# The South African Journal of Sports Medicine

## The Official Publication of the South African Sport Medicine Association

Mobic offers selective COX 2 inhibition for effective inflammatory pain relief.

ONCE-DAILY
mOBIC

Inflammatory pain relief and mobility in any body's language.

meloxicam 7,5 mg/15 mg

S3 Mobic 7,5 mg tablet Each tablet contains 7,5 mg meloxicam. Reg. No. 29/3.1/421.
S3 Mobic Suppositories. Each suppository contains 15 mg meloxicam. Reg No. 29/3.1/425.

Boehringer
Ingelheim    Boehringer Ingelheim (Pty) Ltd. Reg. No. 66/08619/07. 404 Main Avenue, Ferndale, Randburg 2194. Tel. 886-1075.

# EDITORIAL

"This edition of the South African Journal of Sports Medicine, the first of 1998, has a number of changes. First and foremost, there has been a complete restructuring of the management of the Journal which now includes two Senior Associate Editors, who are Prof Maurice Mars and Dr Mike Lambert. I would like to take this opportunity of congratulating Prof Mars and Dr Lambert on their appointment and I look forward to working with them to improve all aspects of the Journal and make it a Journal that the South African Sports Medicine Association can be really proud of. Furthermore, Dr Peter Macfarlane from Cape Town has agreed to serve as a News Editor, a very much needed section within the Journal. I would like to encourage individual members of SASMA to be in contact with Dr Macfarlane to keep him up to date on events that are worthy of publication in this news section.

Finally, I would like to congratulate the new members of the Editorial Board of the South African Journal of Sports Medicine. These members represent Academic Institutions across the country and I look forward to serving the Journal and the Association with their input and expertise.

The contents of this Journal consists of a review article and three original research articles. This ratio of review to original research is what the Journal would strive for over the next 2 years. We recognise that review material is important, but we would first and foremost like to encourage a research culture in the South African Sports Medicine Association. I would therefore encourage researchers in all fields to consider submitting their material to the South African Journal of Sports Medicine, thereby supporting your local Journal. In addition, the Journal would also have ample opportunity for publishing Brief Reports, and Case Reports. Hopefully this will facilitate the busy practitioner who has little time for in-depth clinical research to still submit interesting work in the form of case reports or a brief report. I would like to encourage each of you to consider submitting material in this format for publication. The guidelines for authors wanting to submit either brief reports or case reports has recently been circulated through the SASMA Newsletter. These are also obtainable from the office in Cape Town.

I am indeed excited about the new format of the South African Journal of Sports Medicine, and would encourage you all to support it and our Association.

**Prof MP Schwellnus, MBBCh, MSc (Med), MD, FACSM**
**EDITOR**
**South African Journal of Sports Medicine**

# THE SOUTH AFRICAN JOURNAL OF
# SPORTS MEDICINE

## Editor - in - Chief

# CONTENTS

# Exercise intolerance in patients with chronic heart failure : A Review

KL Derman MSc (Med)
W Derman MBChB PhD FACSM
TD Noakes MBBCh MD FACSM

MRC/UCT Bioenergetics of Exercise Research Unit, Sport Science Institute of South Africa, University of Cape Town Medical School

## ABSTRACT

Exercise intolerance in patients with heart failure is a common but complex phenomenon. Traditionally it has been thought that central cardiorespiratory factors were the important limiting factors of exercise performance in patients with heart failure. However, more recent research suggests that peripheral abnormalities within the skeletal muscle might play a role in the exercise intolerance experienced by these patients. This review encompasses research examining the effects of heart failure on some of the organ systems and physiological response to exercise. Studies of both central (cardiorespiratory) and peripheral (skeletal muscle) factors limiting exercise performance have been reviewed. It is probable that whilst both central and peripheral factors contribute to the exercise intolerance in these patients, patients may be limited more by disease of skeletal muscle than by dyspnoea or central fatigue.

## HISTORICAL PERSPECTIVE OF EXERCISE INTOLERANCE IN HEART FAILURE

Braunwald and Grossman[9] defined heart failure as "the condition in which abnormality of myocardial function is responsible for the ventricles' inability to deliver adequate quantities of blood to the metabolizing tissues at rest or during normal activity". The most common cause of heart failure is loss of cardiac muscle secondary to ischaemia on the basis of coronary artery disease. Viral and other cardiomyopathies are more common in younger patients. Heart failure can also occur in persons with poorly controlled blood pressure or with valvular heart disease.

Of the many symptoms experienced by patients with heart failure, dyspnoea, fatigue and exercise intolerance are amongst the most common. Indeed, the first indication of heart failure is a diminished tolerance to physical exercise.[70] Of such importance are symptoms of dyspnoea that functional capacity in

## CORRESPONDENCE:

Professor Wayne Derman
MRC/UCT Bioenergetics of Exercise Research Unit,
Sport Science Institute of South Africa,
P.O. Box 115
Newlands, 7725
SOUTH AFRICA
Tel    - 27 - 21 - 6867330
Fax    - 27 - 21 - 6867530
email - wderman@sports.uct.ac.za

patients with chronic heart failure was, and still is, measured in terms of the four-tiered classification of breathlessness,[27] which is subjective and only semi-quantitative. Only recently has exercise testing become more popular for the determination of functional capacity in patients with heart failure.[72,146]

Historically it was believed that central cardiorespiratory factors determined submaximal and maximal exercise capacity in patients with chronic heart failure.[10,19,32,111,118,149] As these patients had reduced cardiac output at rest and during exercise, it was thought that the heart was unable to deliver sufficient oxygen to the active skeletal muscles so that an oxygen deficiency in the active muscles caused fatigue. The focus of studies to determine mechanisms of fatigue at rest and during exercise in patients with heart failure was on pulmonary congestion, reduced lung compliance and dyspnoea. Even more recently, the majority of studies have continued to focus on the role of the heart and lungs in the limitation of exercise performance in patients with heart failure. [2,25,59,101,106]

However, since 1960 there has been evidence that signs and symptoms of heart failure could be absent even in the presence of severe heart disease.[41] This led researchers to believe that central cardiovascular function may not necessarily be related to exercise capacity[8,21,44,103] and that chronic heart failure encompasses impairment of multiple organ systems[123] including skeletal muscle.[12,71,76,96,97]

## PHYSIOLOGICAL FACTORS CONTRIBUTING TO EXERCISE INTOLERANCE IN PATIENTS WITH CHRONIC HEART FAILURE

Although exercise intolerance is the most frequent symptom in patients with heart failure, the exact cause of exercise intolerance has yet to be identified.[21] Whilst heart failure is a complex and multisystemic disorder, and whilst the authors appreciate that central and peripheral physiological systems cannot be entirely separated, the limitation of exercise performance in heart failure will be discussed under central (cardiorespiratory) factors and peripheral (blood flow and skeletal muscular) abnormalities which could possibly alter exercise tolerance in patients with chronic heart failure.

**1. Contribution of central cardiorespiratory abnormalities to exercise intolerance in patients with heart failure**

**(i) Left Ventricular Systolic Function**

Left ventricular systolic function is compromised in chronic heart failure. It was thought that this would alter exercise tolerance in patients with heart failure.

However, the poor relationship between maximal exercise capacity and indices of left ventricular (LV) function in patients with heart failure is now an established finding.[6,8,25,44,53,95,105,115,117] There is clearly no correlation between exercise capacity measured on a cycle ergometer or treadmill, and LV ejection fraction, nor any correlation between the degree of ventricular dysfunction and the clinical severity of chronic heart failure.[44,45] A study by Benje et al[8] showed that 50% of patients with an ejection fraction of less than 30% had normal exercise capacity. Reading et al[117] also reported that there was no relationship between resting ejection fraction and peak aerobic power or resistance in the calf vasculature.

Furthermore, it was reported that patients with severe LV dysfunction increased their peak exercise performance after exercise training, whilst they did not show any change in LV ejection fraction or any reduction in wall motion abnormalities.[24,55,60] These findings suggest that factors other than those relating to central cardiovascular function may have determined the beneficial adaptations to training. Regardless of these findings, the ejection fraction continues to be the most common measure of central cardiac performance and predictor of future survival in these patients.[22,53,137,138]

### (ii) Cardiac Output

Cardiac output is reduced at rest and during exercise in patients with chronic heart failure. Furthermore, a lower proportion of total cardiac output is delivered to skeletal muscle during exercise in patients with heart failure.[160] Bain et al[6] demonstrated that exercise time to exhaustion measured during incremental exercise in patients with heart failure, cannot be predicted from resting cardiac output. However, maximal cardiac output and the ability to increase cardiac output during exercise, do correlate with exercise capacity. Muller et al[103] showed that blood flow to the gut, kidneys, and limbs was reduced in patients with chronic heart failure. The lack of any correlation between limb blood flow and cardiac output helps to explain the lack of correlation between cardiac output and symptoms during exercise.

Further evidence that cardiovascular function does not determine exercise capacity in patients with heart failure is the observation that improvement of left ventricular function by acute ingestion of inotropic drugs,[96] vasodilators,[156] or repair of valvular stenosis[84] does not result in an immediate normalization of exercise capacity or of their ventilatory response during exercise, despite marked improvements in cardiac output.[38,85]

### (iii) Blood Pressure

Regulation of blood pressure and temperature are major cardiovascular challenges which arise during muscular work and which may conflict with the need to perfuse the active muscles. Normal persons actively regulate mean arterial blood pressure during exercise by increasing skeletal muscle vascular tone when cardiac output decreases.[75,130] In patients with heart failure, this reflex-mediated peripheral vasoconstriction is thought to maintain blood pressure during dynamic exercise when cardiac output has reached its maximum value.[135] As a result, blood flow to the active muscles is restricted in order to maintain

blood pressure when cardiac output is limited.

Heart rate and mean systemic arterial blood pressure increase less during dynamic exercise in patients with heart failure compared to control subjects and the increase in stroke volume in the patients is also minimal.[155] Despite this abnormal response, mean systemic blood pressure does not relate directly to exercise capacity,[5,53] suggesting that exercise intolerance in these patients is influenced by factors other than peak systolic blood pressure.[43,134]

### (iv) Pulmonary Haemodynamics

It was originally thought that increased pulmonary capillary wedge pressure during exercise caused by left ventricular failure caused dyspnoea, which in turn limited exercise performance.[148] However, the elevated pulmonary capillary wedge pressures at rest in patients with heart failure increase to the same levels during submaximal and maximal exercise,[138] and these elevated levels appear to be well-tolerated.[122] Massie[90] found pulmonary capillary pressures to be no higher in patients with heart failure whose exercise tolerance was limited by dyspnoea compared to patients limited by leg fatigue. These findings indicate that mechanisms other than pulmonary capillary wedge pressure appear to be important in limiting exercise performance in patients with chronic heart failure.[43,134]

### (v) Pulmonary Ventilation

Patients with chronic heart failure have altered ventilatory responses to graded exercise. Hyperventilation is commonly observed during exercise in patients with heart failure and has been attributed to decreased lung compliance, regional ventilation-perfusion mismatch in the lungs as a result of the reduced cardiac output[104,144] and increased dead space volume.[134,148] Despite this abnormal ventilatory response to exercise and decreased maximal ventilation,[123] the normal control of ventilation by $VCO_2$ is present in patients with chronic heart failure.[42,105,134]

Although it is unclear to what extent these ventilatory abnormalities influence exercise performance in patients with chronic heart failure,[105] it has been suggested that leg fatigue causes termination of exercise before there is a substantial encroachment on ventilatory reserve in patients with severe heart failure.[105,123]

It is possible that abnormal respiratory muscle function[164] or skeletal muscle abnormalities[62] may contribute to dyspnoea and ventilatory abnormalities in these patients. Nishimura et al[106] reported that inspiratory muscle strength was impaired in patients with severe congestive heart failure and may limit exercise in these patients. Furthermore, 'periodic breathing' has been described in patients with congestive heart failure at rest and during exercise.[64,119] This has been attributed to unstable ventilatory control caused by poor circulation and to delayed transmission of humoral ventilatory stimuli to the chemoreceptors.[17,66] However, the exact mechanism for the periodic breathing in patients with heart failure is still unknown, although a central (cardiovascular) mechanism is still favoured.[7] Little is known about the time course, exact changes, causative factors, or the mechanisms of these venti-

latory alterations. It is not known whether these alterations in pulmonary ventilation are the cause or effect of the exercise intolerance.

### (vi) Peak Oxygen Consumption (VO₂ peak)

Reduced $VO_2$ peak in patients with heart failure has been repeatedly confirmed.[45,53,100,105,148] However, the exact cause of this finding has not been identified. Decreased oxygen consumption in patients with chronic heart failure implies that either $O_2$ supply or $O_2$ utilization by the active skeletal muscle is altered. Alternatively, the muscle's capacity to perform exercise could be impaired even in the face of an adequate oxygen supply.

Wade and Bishop[145] suggested that the primary limitation of $VO_2$ peak in patients with heart failure is a relatively reduced pulmonary blood flow. However, many investigators have since shown that arterial oxygen tension and systemic arterial $O_2$ saturation remains normal even at peak exercise in patients with chronic heart failure.[43,122,134] This suggests that pulmonary function does not limit $VO_2$ peak in these patients, and that peripheral alterations may indeed influence peak oxygen consumption in these patients. These theories are supported by the following important findings:

(a) Weber et al[148] demonstrated that although peak cardiac output and peak $VO_2$ are linked, as the severity of heart failure worsens, the slope of the cardiac output response with increasing $VO_2$ decreases. This indicates that the normal relationship between cardiac output and oxygen consumption is altered in these patients, possibly due to increased oxygen extraction at the periphery from a reduced (muscle) blood flow.

(b) Thompson et al[139] and Sullivan et al[132,133] reported that femoral venous $PO_2$ does not decrease below the critical level of 10mmHg during exercise in both normal subjects and in patients with chronic heart failure. These findings suggest that muscle fatigue may not be caused by an inadequate oxygen supply to the active muscle.

(c) Failure to observe an improvement in oxygen uptake by skeletal muscle during exercise even when oxygen availability is enhanced by pharmacological intervention in patients with chronic heart failure[89,37] indicates a change in the properties of skeletal muscle itself in these patients.

(d) Moore et al[101] reported that maximal exercise performance on a cycle ergometer was acutely enhanced compared to exercise with room air, when patients with heart failure inspired oxygen at a concentration of 50% $O_2$. This was explained by an increased efficiency during exercise, so that there was a decreased ventilatory and circulatory demand at a given workload, while $O_2$ delivery to muscle was maintained (Moore et al. 1992). However, this does not prove that oxygen supply to the active muscle was limiting during exercise.

(e) Jondeau et al[56] measured $VO_2$ peak during maximal exercise with lower limbs and with lower limbs plus upper limbs in patients with heart failure. Patients were able to increase $VO_2$ peak when exercising with both lower and upper limbs. This finding was not observed in the control group of healthy subjects. This indicates that the exercise intolerance in cardiac patients during lower limb exercise was not due to a central limitation, as $VO_2$ peak increased when additional skeletal muscle was recruited in these patients.

(f) Recent reports indicate that $VO_2$ peak is predicted by muscle strength in patients with chronic disease. Isometric maximal voluntary contraction (MVC) of the quadriceps muscle correlates to peak $VO_2$ during exercise in patients with chronic heart failure.[71] This finding indicates that skeletal muscle function can predict maximal exercise performance. Similarly, isokinetic quadriceps muscle strength correlates to peak $VO_2$ in patients with renal failure.[31] Therefore peak $VO_2$ is probably indicative of peripheral, skeletal muscular rather than central cardiovascular function in this group of patients.[31,61,107]

In light of the above-mentioned findings, the search for mechanisms of exercise intolerance in patients with heart failure has shifted from the central cardiovascular system to the peripheral musculoskeletal system.

### 2. Contribution of peripheral abnormalities to exercise intolerance in patients with heart failure

#### (i) Physiological alterations in the periphery during exercise in patients with chronic heart failure are:

#### (a) Alteration of blood flow to skeletal muscle

Results of studies investigating blood flow to the skeletal muscle during exercise in patients with heart failure have produced conflicting results. It appears that many factors including the mass of skeletal muscle recruited, severity of cardiac failure, exercise protocol used, and the patients' medication contribute to alterations in blood flow during exercise in these patients.

Peripheral blood flow is lower during progressive dynamic and during sustained isometric exercise in patients with chronic heart failure compared to normal controls.[74,154,155,156,161] A lower proportion of total cardiac output is delivered to exercising skeletal muscle in patients with heart failure. This occurs as a compensatory mechanism to prevent hypoperfusion of important non-exercising areas (brain, kidney) or to preserve arterial blood pressure[135,160] or as a result of increased skeletal muscle vascular resistance.[160]

It appears that blood flow to the working skeletal muscle is dependent on the volume of active skeletal muscle mass recruited during exercise.[77] These researchers reported that patients with moderate heart failure can attain a peak skeletal muscle perfusion and leg oxygen consumption comparable to that of healthy control subjects when small amounts of skeletal muscle mass is recruited. However during recruitment of larger volumes of skeletal muscle mass (>4kg), peak blood flow, muscle perfusion and oxygen uptake as well as blood flow to the non-working skeletal muscle is reduced compared to controls.[77]

Recently Yamabe et al[158] reported that blood flow to skeletal muscle during submaximal cycle exercise is higher in NYHA class III patients than to skeletal muscle of patients with less severe heart failure. These investigators suggest that the relatively increased skeletal muscle blood flow in patients with severe heart failure plays a role to compensate for the insufficient cardiac output response in these patients.

Impaired vasodilation during exercise is an important factor responsible for the reduced maximal limb blood flow in patients with heart failure[79,134,152] However, the pathophysiological cause of decreased muscle blood flow during exercise has not been clearly defined. Possible factors limiting blood flow to the skeletal muscle during exercise in patients with heart failure include increased vascular stiffness,[67,102] alterations in neurohumoral activation,[46,77,120] alteration in blood vessel vasodilator capacity[151,156] and alteration in flow-dependent dilation.[68,121,129] These factors may play an important role in the natural history of the disease, but may not be critical to the impaired exercise capacity of these patients.[23]

It is important to note that Wilson et al[151] examined the acute effects of the vasodilators dobutamine, dopamine, and phosphodiesterase on maximal exercise performance in patients with chronic heart failure and found no improvement in exercise performance even when blood flow to the legs was acutely increased by these agents. Nor did increased blood flow to exercising muscle induced by hydralazine appear to reduce femoral venous lactate concentrations or the degree of acidosis or fatigue in patients with heart failure.[154] These results indicate that exercise performance was not dependent on muscle blood flow, as it failed to increase with an acute increase in muscle blood flow.[155]

Drexler et al[34,36] report that chronic administration of angiotensin converting enzyme (ACE) inhibitors can partially reverse mitochondrial abnormalities in patients who have depressed oxidative capacity of skeletal muscle during exercise. Chronic ACE inhibition not only increases skeletal muscle blood flow at maximal exercise,[79] but also improves functional capacity in these patients by acting primarily on the peripheral vasculature without changing intrinsic myocardial function.[92] These results suggest that there is a gradual reduction in structural vascular abnormalities by the chronic effect of ACE inhibition, and that this improves exercise performance.

### (b) Reduced blood flow causing altered skeletal muscle metabolism

It has been suggested that reduced muscle blood flow at rest and during exercise in patients with heart failure[87,88,89,135,150,156,163] might be important in determining the metabolic response to exercise. Reduced muscle blood flow is characterized by increased glycolytic metabolism and decreased oxidative phosphorylation in the skeletal muscles of these patients. However, Weiner et al[150] reported that the reduced blood flow to exercising skeletal muscle was not a consistent finding in forearm muscles where metabolism was altered. Massie et al[90] established that metabolic abnormalities are unrelated to blood flow by comparing the effects ischaemic forearm exercise in patients with heart failure and in normal control subjects. While the brachial artery was temporarily occluded during submaximal finger flexion, patients utilized phosphocreatine (PCr) at a faster rate compared to controls despite performing much less work, and pH levels fell substantially. As blood flow was not different, results suggest that a functional abnormality of the skeletal muscle exists in patients with congestive heart failure. Buller et al[12] measured fatiguability of adductor pollicis muscle during supramaximal repetitive stimulation of

the ulnar nerve during circulatory occlusion in patients with severe heart failure and in normal controls. The effect of ischaemia was greater in patients with severe (Class IV) heart failure than in patients with mild to moderate heart failure or in normals and the investigators attributed this to impaired skeletal muscle metabolism. Marie et al[83] confirmed this when they reported that the rate of PCr resynthesis in calf muscle of patients with congestive heart failure was similar following either aerobic or ischaemic exercise.

### (c) Oxygen extraction and utilization

Despite decreased blood flow during submaximal exercise, it has been demonstrated that the skeletal muscles of patients with chronic heart failure can compensate by increasing oxygen extraction, hence increasing the arterio-venous oxygen (A-VO$_2$) difference.[155] However, Roubin et al[122] observed that although the A-VO$_2$ difference across the leg during exercise was greater in patients with heart failure than in normal persons, VO$_2$ peak was 40% lower, suggesting that the greater A-VO$_2$ difference does not compensate completely for the reduction in skeletal muscle blood flow. Alternatively, skeletal muscle weakness may prevent patients with heart failure from reaching high levels of exercise at which higher VO$_2$ values would be measured. Wilson et al[156] showed that maximal leg blood flow and maximal leg oxygen uptake in patients with chronic heart failure were markedly reduced in patients with the poorest exercise tolerance. These findings suggest that either the skeletal muscle cannot fully utilize the delivered oxygen or that oxygen availability or diffusion to the skeletal muscles is impaired in these patients. However, these studies do not exclude the possibility that muscle weakness limits exercise tolerance before an oxygen limitation develops.

### (d) Blood lactate concentrations

It is usually argued that skeletal muscle hypoperfusion during exercise contributes to the early onset of skeletal muscle lactate production[1,146,147,150,156] and that increasing exercise intolerance is associated with progressively earlier increases in mixed venous blood lactate concentrations in patients with chronic heart failure.[146,148,156] Furthermore, it has been demonstrated that venous blood lactate accumulation patterns correlate with severity of circulatory failure at rest[94] and with severity of heart failure during exercise.[146] Increased lactate concentration is thought to be a contributing factor to fatigue and dyspnoea experienced during exercise in patients with chronic heart failure.[50]

However, more recent studies suggest that blood lactate concentrations are indeed much lower at peak and submaximal exercise in patients with heart failure compared with controls and therefore increased blood lactate production does not contribute to the exercise intolerance experience by the patients with heart failure.[29,108,126]

### (ii) Skeletal muscle abnormalities

### (a) Histological and biochemical abnormalities in skeletal muscle biopsies of patients with chronic heart failure

Recent studies report that patients with heart failure identify leg fatigue as the limiting factor during exer-

cise[7,20,62,105,122] and that the stride length of walking is reduced in patients with chronic heart failure.[28] The study of skeletal muscle as the possible cause of exercise intolerance in these patients has gained popular interest.

A review of histological and biochemical findings of previous studies of skeletal muscle samples from patients with chronic heart failure is presented in Table 1. Abnormalities of skeletal muscle fibre morphometry, cellular organelles, capillary structure and biochemical alterations have been described in these patients (Figure a + b).
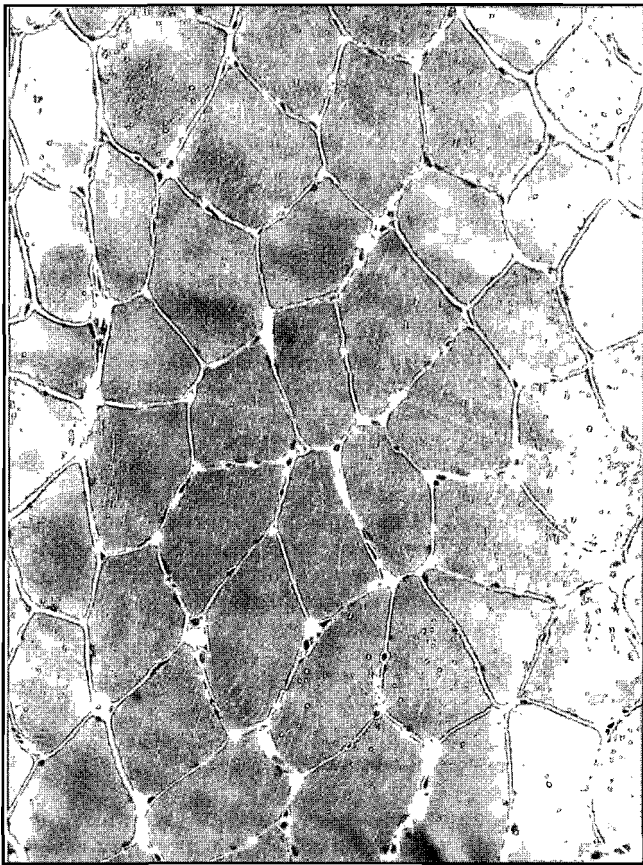


Fig 1a. *A light photomicrograph showing normal skeletal muscle from a control subject (H&E x 200).*

Recent work by Lipkin et al,[71] Mancini et al[80] and Minotti et al[98] suggests that skeletal muscle atrophy occurs early in the course of congestive heart failure and might play a role in the reduction of functional capacity in patients with chronic heart failure. Compared to normal muscle, atrophied muscle is subjected to a greater workload per remaining fiber when faced with a given external load and therefore develops greater reductions in PCr and intracellular pH.[82] Mancini et al[82] recently tested this hypothesis by correlating calf muscle volume with the metabolic response of the calf muscle to exercise during supine plantar flexion against increasing force. They discovered that skeletal muscle atrophy contributed significantly to the abnormal metabolic response to exercise, but that the correlation was relatively weak, suggesting that increases in muscle mass would produce only modest improvements in exercise capacity, and that the primary factor causing reduced pH and PCr during exercise is not skeletal muscle atrophy.



Fig 1b. *A light photomicrograph showing prominent atrophy, hypertrophy and split fibres. There are occasional central nuclei and capillaries with very thick walls can be seen. (H&E x 200)*

A possible cause of skeletal muscle atrophy in patients with chronic heart failure is chronic malnutrition and deconditioning due to reduced levels of habitual activity. Carr et al[15] reported that 50% of 48 patients in severe heart failure were malnourished, defined by a decrease in percent body fat, a decreased weight to height index, or reduced serum albumin concentrations. However, they did not measure skeletal muscle volume. Mancini et al[82] showed that muscle atrophy was not usually associated with signs of severe malnutrition and that protein synthetic function was maintained in heart failure. They also reported that anorexia was not the sole aetiological factor for the muscle atrophy; other factors include inactivity, an increased catabolic state due to heightened sympathetic stimulation and increased serum concentrations of cortisol, ACTH, and tumor necrosis factor.[46,69,112]

Diaphragmatic muscle atrophy has been described in patients who are chronically ill and in patients with chronic obstructive lung disease who have lost weight.[4,139] Indeed, diaphragmatic muscle changes occur in patients with heart failure and could contribute to the generation of dyspneoa during exercise,[52] and indeed to Cheyne-Stokes breathing. Other researchers have recently reported a shift of fast to slow myosin heavy chain isoforms with an increase in oxidative capacity and a decrease in glycolytic capacity in biopsies of the diaphragm muscle of patients with cardiac failure.[141] These changes mirror those noted following endurance training in limb musculature of normal individuals and might be due to increased limb

muscle ergoreflex activity from damaged peripheral skeletal muscle in patients with heart failure, which in turn leads to increased ventilation.[18]

Gibson et al[48] reported that immobility in patients with heart failure is associated with a 25% fall in protein synthesis in quadriceps muscle. However, as skeletal muscle regains normal protein synthesis after heart transplantation,[102] even though patients remain relatively immobile after heart transplantation, immobility cannot be the sole cause of this skeletal muscle abnormality.

Although skeletal muscle atrophy may be caused by inactivity and bedrest, Poole-Wilson et al[114] reported that histological abnormalities seen in patients with chronic heart failure are not typical of the changes seen as a result of either prolonged bedrest or of reversible ischaemia caused by peripheral vascular disease.

Increased subsarcolemmal mitochondrial aggregates have been reported in children with cardiomyopathy and chronic heart failure.[40] Conversely, cardiac conduction defects have been described in many patients with mitochondrial myopathy,[57,109] suggesting that the myopathy is generalized.[11]

### (b) Capillary abnormalities

Longhurst et al[73] studied biopsies from pronator teres muscle and found capillary basement membrane thickening in patients with heart failure compared to a control group. Wroblewski et al[157] documented increased capillary basement membrane thickening in the skin in patients with chronic heart failure and suggested that increased venous pressure due to heart failure and abnormal baroreceptor-mediated arteriolar vasodilation[58] raised capillary pressures in these patients. This enhanced stress could eventually lead to increased thickness of these basement membranes. Some studies suggest that basement membrane thickening is a result of repeated episodes of cell death and cell regeneration and may decrease diffusion across the capillary.[142,143] However, Alpert et al[3] studied increased basement membrane thickness in diabetes and suggested that the increased thickness facilitates rather than retards oxygen diffusion.

### (c) Glycolytic and mitochondrial enzyme activities

Whilst the skeletal muscle glycolytic enzyme activities are not reduced in CHF[78,133] reduced activities of the enzymes involved in aerobic metabolism (succinate dehydrogenase and cytochrome oxidase) is a common finding in these patients.[14,34,81,88,89,116,150] Some researchers have found respiratory exchange ratios to be lower at maximal and at submaximal exercise in patients with chronic heart failure compared to controls.[105,120] The decreased values in the patients could be explained by an increased reliance on fat, as opposed to carbohydrate metabolism in these patients in an effort to preserve skeletal muscle glycogen stores.

### (d) Oxidative enzyme activity

Reduced oxidative capacity of skeletal muscle enzymes involved in aerobic metabolism, including succinate dehydrogenase and cytochrome oxidase, is a common finding in patients with heart failure.[38,81,88,89,116,150,154,155,159] Drexler et al[35] reported a close

relationship between the oxidative capacity of skeletal muscle and $VO_2$ peak during exercise in patients with chronic heart failure, and suggested that the extent of alteration of skeletal muscle metabolism was related to exercise capacity in these patients. Furthermore, Stratton et al[131] reported that training improved oxidative capacity of forearm skeletal muscle, indicating that the impaired oxidative capacity may be due to inactivity and responds to a period of training. However, Lipkin et al[71] reported that skeletal muscle oxidative enzyme activity was within the normal range in patients with severe chronic heart failure. This finding is supported by Minotti et al[97,99] who reported that since oxygen extraction by skeletal muscle is near complete in these patients, skeletal muscle metabolic disorders are not likely to substantially limit maximal exercise performance.

### (e) Increased blood concentrations of tumor necrosis factor

Increased circulating concentrations of tumor necrosis factor (TNF) in skeletal muscle of patients with heart failure have been measured. However, this cytokine is not present in all patients with cardiac abnormalities.[69,93] Furthermore, Drexler et al[34] recently reported that circulating concentrations of tumor necrosis factor do not correlate with the extent of skeletal muscle damage.

In summary, few studies have examined skeletal muscle abnormalities and whilst some studies have documented gross ultrastructural changes in the skeletal muscles of patients with chronic heart failure,[39,71] few studies have yet provided a detailed description of these changes or have provided adequate control groups to establish the specificity of these changes to heart failure.

## SKELETAL MUSCLE FUNCTION IN PATIENTS WITH CHRONIC HEART FAILURE

### (i) Isometric skeletal muscle function

Studies of isometric skeletal muscle function have produced conflicting results. This is possibly due to differences in exercise protocol used in various studies, size of the muscle group examined and correction for lean muscle mass or cross sectional area.

Several studies have reported that skeletal muscle function is impaired in patients with heart failure, even when the heart failure is mild.[12,71,96] Lipkin et al[71] documented that isometric maximal voluntary contraction of quadriceps muscles of heart failure patients was 55% of the predicted strength for an age-, mass- and sex-matched population even after taking into account the decrease in cross-sectional area of the muscle, but no control group was actually tested.

Muscle cross-sectional area in healthy subjects is directly related to force development[91,125] and it has been assumed that decreased cross-sectional area of skeletal muscle causes decreased skeletal muscle strength in patients with heart failure.[76]

Buller et al[12] reported that supramaximal repetitive stimulation of a large muscle mass (quadriceps) caused rapid onset of fatigue and reduced isometric force production in patients with severe heart failure compared to patients with mild heart failure and controls, whereas force production and fatigue of adductor pollicis was not different between groups.

However, the maximal isometric force production per unit muscle cross-sectional area was within the normal range (according to norms of Chapman et al[16]). This led to the conclusion that the reduction in force for the larger muscle mass was not due to impaired force production by the myofibril. Minotti et al[97] reported that maximal voluntary contraction was not different in patients compared to control subjects even though maximal cross-sectional area of the knee extensors was significantly smaller in the patients. They also found a strong correlation between isometric strength and maximal cross-sectional area of the thigh muscles. In addition, Magnusson et al[76] found that although the isometric strength of the quadriceps femoris muscle of these patients was 14% less than age-matched controls, the tension per unit of muscle cross-sectional area was similar between groups, which is in accordance with the report of Buller et al.[12]

More recently Harridge et al[51] studied contractile characteristics and resistance to fatigue in male patients with chronic heart failure. These researchers found that maximal voluntary isometric contraction was similar for patients and controls, however, a faster isometric twitch time course was observed in the patients with heart failure compared with controls. The poor resistance to fatigue was confirmed in this study by using twitch interpolation. This was shown not to be due to poor skeletal muscle activation. These findings indicate that independant of muscle strength, patients with heart failure have impaired resistance to muscle fatigue. However, the increased fatigablility seems not to be due to poor muscle activation.[51]

### (ii) Isokinetic skeletal muscle function

Minotti et al[97] measured peak torque and endurance during isokinetic knee extensions in these patients and found that dynamic muscle endurance was significantly lower in patients compared to controls. Furthermore there was only a weak correlation between dynamic endurance and knee extensor cross-sectional area in these patients.[99] Also, Magnusson et al[76] reported a markedly lower dynamic endurance capacity of the quadriceps femoris muscles in patients with chronic congestive heart failure compared to age-matched controls, but there was no difference in peak tension per unit of cross-sectional area of the skeletal muscle. Thus it appears that diminished skeletal muscle endurance capacity during repetitive exercise is the most consistent finding in these patients, and that a reduction in muscle strength is not always a consistent finding, especially when the cross sectional muscle area is taken into account.[12,97-99]

### (iii) Skeletal muscle recruitment

It has been suggested that central nervous system inhibition of skeletal muscle afferents may limit exercise during hypoxia or hypoperfusion in normal subjects.[47,49,54] It is not known whether this is due to a feedforward or feedback mechanism from damaged or hypoxic skeletal muscle, or whether this mechanism could affect exercise tolerance in patients with chronic heart failure.

Minotti et al[98] measured amplitude and area of the M wave (compound muscle action potential) decline during progressive, fatiguing exercise in an attempt to determine whether the accelerated fatigue in these patients is due to impaired muscle activation as a result of inadequate central motor drive or neuromuscular transmission, or by a change in muscle itself. Their findings indicate that the more rapid onset of muscle fatigue in patients with congestive heart failure is not caused by impaired central motor drive or neuromuscular junction transmission. Rather, they suggest that this fatigue is caused by an abnormality in the muscle itself.

### (iv) Additional factors and their possible effect on exercise performance in patients with chronic heart failure

It is possible that the medication ingested by patients with chronic heart failure, chronic deconditioning due to heart failure, or the age of the patient might also contribute to the impaired exercise performance of these patients.

### (a) Effects of medication on exercise performance in patients with heart failure

Ingestion of medication plays a key role in the management of symptoms in patients with chronic heart failure and the effect of these agents on exercise performance requires consideration. A recent significant development in medical management of heart failure is the introduction of angiotensin converting enzyme (ACE) inhibitors. Chronic ingestion of either captopril and enalapril not only improves signs and symptoms of heart failure and increase exercise performance, but reduces mortality in patients with chronic heart failure.[113,128]

The negative inotropic effect of beta blockers causes haemodynamic and clinical deterioration in patients with heart failure,[65] and may also cause an increased rate of perceived exertion during submaximal exercise effecting either the central nervous system or skeletal muscle in these patients.[30] Diuretics are effective in reducing total body sodium and water content, and thus relieving the symptoms due to fluid retention in patients with heart failure, but there are no data to suggest that they improve quality of life or exercise tolerance in these patients. Digitalis does appear to be effective in improving prognosis in patients with heart failure,[110] but the benefits are confined to those patients who have markedly dilated left ventricles, with markedly impaired systolic function.

### (b) Physical deconditioning

Patients with heart failure are less physically active than normal healthy controls. The pattern of skeletal muscle changes seen in patients with chronic heart failure is consistent with the effects of chronic exercise deconditioning, but the magnitude of these changes exceeds those which occur after exercise deconditioning in normal subjects.[26] In both states there is exercise intolerance, sympathetic activation, increased resting heart rate, reduced heart rate variability, atrophied skeletal muscle and reduced activity of skeletal muscle oxidative enzymes.[1,37,124,136] Deconditioning appears to be one factor involved in the development of these potentially reversible skeletal muscle alterations[39] and for the impaired peripheral vasodilation during exercise in patients with chronic heart failure.[127]

## (c) Contribution of age to exercise intolerance in patients with heart failure

As various physiological changes occur with increasing age, it is important that these changes are considered when the physiological response to exercise in patients with heart failure is gauged. Although it has been reported that oxygen consumption, cardiac output, stroke volume, ejection fraction, heart rate and Frank-Starling response at maximal exercise are significantly reduced in normal elderly subjects compared to younger subjects,[63] the muscle strength of patients with chronic heart failure[12] is significantly less than age-matched, sedentary controls.[96] Hence, while cardiovascular function is attenuated in both healthy elderly persons and elderly persons with chronic heart failure, skeletal muscle strength appears to be affected to a greater extent in patients with chronic heart failure than in age-matched sedentary controls.

## SUMMARY

Exercise performance is markedly reduced in patients with heart failure but the the mechanism explaining the exercise intolerance has not been precisely defined. Previously, central cardiorespiratory factors were the main focus of researchers, who measured mainly central physiological variables during exercise to determine the limitation of exercise in these patients. The many inconsistencies discussed in this review indicate that other factors could be responsible for exercise intolerance in these patients. More recently exercise intolerance in patients with heart failure has been explained by abnormalities in peripheral blood flow, skeletal muscle structure, and in skeletal mucscle metabolism. It is probable that whilst both central and peripheral factors contribute to the exercise intolerance in these patients, patients may be limited more by disease of skeletal muscle than by dyspnoea or central fatigue.

**Table 1. Review of histological and biochemical findings in studies of skeletal muscle biopsies in patients with chronic heart failure.**

| Structure | Finding | Study |
|---|---|---|
| **Fibre Morphometry** | | |
| Type I CSA | increased | Sullivan et al. 1990. |
| | decreased | Lipkin et al. 1988; Poole-Wilson et al. 1988. |
| Type II CSA | decreased | Dunnigan et al. 1987; Lipkin et al. 1988; Wilson et al. 1992; Poole-Wilson et al. 1988; Bussieres et al. 1997. |
| | increased | Mancini et al. 1989. |
| Type I % | decreased | Mancini et al. 1989; Sullivan et al. 1990; Drexler et al. 1992. |
| Type II % | increased | Mancini et al. 1989; Sullivan et al. 1990; Bussieres et al. 1997. |
| **Cellular organelles** | | |
| Mitochondrial volume density | decreased | Drexler et al. 1992. |
| Surface area of mitochondrial cristae | decreased | Drexler et al. 1992. |
| Subsarcolemmal mitochondrial aggregates | increased | Dunnigan et al. 1987; Smith et al. 1976. |
| Interstitial cellularity | increased | Lipkin et al. 1988. |
| **Capillaries** | | |
| Capillary density | normal | Lipkin et al. 1988; Mancini et al. 1989. |
| Capillary density | decreased | Sullivan et al. 1990; Drexler et al. 1992. |
| Basement membranes | thickened | Longhurst et al. 1975. |
| Intracellular lipid content | normal | Drexler et al. 1992. |
| | increased | Dunnigan et al. 1987; Lipkin et al. 1988; Smith et al. 1976. |
| Intracellular glycogen content | normal | Drexler et al. 1992. |
| | decreased | Sullivan et al. 1990. |
| Glycolytic enzyme activity | normal | Mancini et al. 1989; Sullivan et al. 1990. |
| **Oxidative enzyme activity** | | |
| Cytochrome oxidase | decreased | Drexler et al. 1992; Ralston et al. 1991 |
| | normal | Lipkin et al. 1988. |
| Citrate synthase | decreased | Mancini et al. 1989; Sullivan et al. 1990; Ralston et al. 1991; Wilson et al. 1992; Bussieres et al. 1997. |
| Succinate dehydrogenase | decreased | Ralston et al. 1991; Mancini et al. 1989; Sullivan et al. 1990; |
| β-hydroxyacyl CoA dehydrogenase | decreased | Bussieres et al. 1997 |
| Succinate cytochrome reductase | normal | Lipkin et al. 1988. |

Abbreviations: CSA, cross sectional area.

# References

1. Adamopoulos S, Coats AJS. Peripheral abnormalities in chronic heart failure. Postgrad Med J 1991;67:S74-S80

2. Agostoni P Wasserman K, Perego GB, Marenzi GC, Guazzi M, Assanelli E. Lauri G, Guazzi MD. Am J Cardiol 1997: 79 (8): 1120-1124.

3. Alpert JS, Coffman JD. Baldimos MC, et al. Capillary permeability and blood flow in skeletal muscle of patients with diabetes mellitus and genetic prediabetes. N Engl J Med 1972;286:454-469.

4. Arora N. Rochester D. Effect of body weight and muscularity on human diaphragm muscle mass, thickness, and area. J Appl Physiol 1982;52:64-70.

5. Bain R, Bun Tan L, Murray RG. The correlation of cardiac power output to exercise capacity in chronic heart failure. Eur J Appl Physiol 1990;61:112-118.

6. Baker JB. Wilen MM, Body CM. Dinh H. Franciosa JA. Relation of right ventricular ejection fraction to exercise capacity in chronic left ventricular failure. Am J Cardiol 1984;54:596-599.

7. Ben Dov 1, Sietsema KE, Casaburi R. Wasserman K. Evidence that circulatory oscillations accompany ventilatory oscillations during exercise in patients with heart failure. Am Rev Respir Dis 1992;145:776-781.

8. Benje W, Litchfield RL. Marcus ML. Exercise capacity in patients with severe left ventricular dysfunction. Circulation 1980;61:955-959.

9. Braunwald E and Grossman W. Heart Disease. A textbook of cardiovascular medicine. Fourth Ed. WB Saunders Company. Harcourt Brace Jovanovich, Inc. 1992: p444.

10. Bruce RA. Exercise testing for evaluation of ventricular function. N Engl J Med 1977;296:671-675.

11. Brussieres LM, Pflugfelder PW, Guiraudon C, Brown WE, Munoz DG, Taylor AW, Kostuk WJ. Exercise responses after cardiac transplantation in mitochondrial myopathy. Am J Cardiol 1993;71:1003-1006.

12 Buller NP, Jones D, Poole-Wilson PA. Direct measurement of skeletal muscle fatigue in patients with chronic heart failure. Br Heart J 1991;65:20-24.

13. Buller NP, Jones D. Poole-Wilson PA. Evidence for an intrinsic defect of skeletal muscle metabolism in patients with chronic heart failure. Clin Sci 1987;72:60P

14. Bussieres LM, Pflugfelder PW Taylor AW Noble EG. Kostuk WJ. Changes in skeletal muscle morphology and biochemistry after cardiac transplantation. Am J Cardiol 1997: 79 (5): 630-634.

15. Carr J, Stevenson L, Walden J, Heber D. Prevalence and hemodynamic correlates of malnutrition in severe congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol 1989;63:709-713.

16. Chapman SJ, Grindrod SR, Jones DA. Cross-sectional area and force production of the quadriceps muscle. J Physiol (Lond) 1984;353:53P.

17. Cherniack NS, Longobardo GS. Cheyne-Stokes breathing, an instability in physiological control. N Engl J Med 1973;288:952-7.

18. Clark AL, Poole-wilson PA, Coats AJ. Exercise limitation in chronic heart failure: central role of the periphery. J Am Coll Cardiol 1996; 28 (5): 1092-1102.

19. Clausen JP. Circulatory adjustments to dynamic exercise and effect of physical training in normal subjects and in patients with coronary artery disease. Prog Cardiovasc Dis 1976;28:459-495.

20. Coats AF. The "muscle hypothesis" of chronic heart failure. J Mol Cell Cardiol 1996, 28 (11): 2255-2262.

21. Coats AJ. Origins of symptoms in heart failure. Cardiovasc Drugs Ther 1997, May; 11 Suppl 1: 205-272.

22. Cohn JN, Archibald DG, Francis GS, Ziesche S, Franciosa JA, Harston WE, et al: Veterans Administration Cooperative Study on vasodilator therapy of heart failure: influence of prerandomization variables on the reduction of mortality by treatment with hydralazine and isosorbide dinitrate. Circulation 1987;75:1V49-1V54.

23. Cohn JN. Role of neurohumoral control of the circulation in determining exercise capacity in patients with heart failure. Chest 1992:101:347S-349S.

24. Conn EH, Whitom ORS, Wails GA. Exercise responses before and after physical conditioning in patients with severely depressed left ventricular function. Am J Cardiol 1982;49:296-300.

25. Cowley AJ, Fullwood LI. Muller AF. Stainer K. Skene AM, Hampton JR. Exercise capability in heart failure: is cardiac output important after all? Lancet 1991;337:771-773.

26. Coyle EF, Martin WH. Sinacore DR, Joyner MJ. Hagberg JM. Holloszy JO. Time course of loss of adaptations after stopping prolonged intense endurance training. J Appl Physiol 1984;57:1857-1864.

27. Criteria Committee. New York Heart Association. Diseases of the heart and blood vessels. Nomenclature and criteria for diagnosis 1964;6th Ed.

28. Davies SW, Greig CA. Jordan SL. Lipkin DP. Short-stepping gait in severe heart failure. Br Heart J 1992;55:469-472.

29. Derman EW. Selley KL, Emms M, Sinclair-Smith C, Brink J & Noakes TD. Structural and functional abnormalities before and after orthotopic cardiac transplantation. Med Sci Sports Exerc 1992: 24:5 S512 .

30. Derman EW. Sims R. Noakes TD. The effects of antihypertensive medications on the physiological response to maximal exercise testing. J Cardiovasc Pharmacol 1991:18: S122-S127.

31. Diesel W. Noakes TD, Swanepoel C. Lambert M. Isokinetic muscle strength predicts maximal exercise tolerance in renal patients on chronic hemodialysis. Am J Kidney Dis 1990;XVI:109-114.

32. Donald KW. Gloster J, Harris EA. Reeves J. Morris P. The production of lactic acid during exercise in normal subjects and patients with rheumatic heart disease. Am Heart J 1961;72:494-510.

33. Drexler H, Faude F, Hoing S. Blood flow distribution within skeletal muscle during exercise in the presence of chronic heart failure: effect of milrinone. Circulation 1984;76: 1344-1352.

34. Drexler H, Munzel T. Riede U. Just H. Adaptive changes in the periphery and their therapeutic consequences. Am J Cardiol 1991b;67:29C-35C.

35. Drexler H, Riede U, Hiroi M, Munzel T. Holubarsch C. Meinertz T. Ultrastructural analysis of skeletal muscle in chronic heart failure: relation to exercise capacity and indices of LV-dysfunction. Circulation 1988:78:107.

36. Drexler H, Riede U. Munzel T. Konig H. Funke E. Just H. Alterations of skele-

37. Drexler H, Riede U, Schafer H-E. Reduced oxidative capacity of skeletal muscles in patients with severe heart failure. Circulation 1987b;76:178

38. Drexler H. Reduced exercise tolerance in chronic heart failure and its relationship to neurohumoral factors. Eur Heart J 1991a:12:21-28.

39. Drexler H. Skeletal muscle failure in heart failure. Circulation 1992:85:1621-1623.

40. Dunnigan A, Staley NA, Smith SA, Pierpont ME, Judd D, Benditt DG, Benson DW. Cardiac and skeletal muscle abnormalities in cardiomyopathy: comparison of patients with ventricular tachycardia or congestive heart failure. J Am Coll Cardiol 1987;10:608-618.

41. Eichna LW. Circulatory congestion and heart failure. Circulation 1960;22:864-886.

42. Fink LI, Wilson JR. Ferraro N. Exercise ventilation and pulmonary artery wedge pressure in chronic stable congestive heart failure. Am J Cardiol 1986;57:249-53.

43. Franciosa JA, Leddy CL. Wilen M. Schwartz D. Relation between haemodynamic and ventilatory response in determining exercise capacity in severe CHF. Am J Cardiol 1984;53:127-34.

44. Franciosa JA, Park M, Levine B. Lack of correlation between exercise capacity and indexes of resting left ventricular performance in heart failure. Am J Cardiol 1981:47:33-39.

45. Franciosa JA, Ziesche S, Wilen M. Functional capacity of patients with chronic left ventricular failure. Relationship of bicycle exercise performance to clinical and haemodynamic characterization. Am J Med 1979;67:460-466.

46. Francis GS, Benedict C, Johnstone D, Kirlin P. Nicklas J, Liang C, Kubs S, Rudin-Toretsky E, Yusuf Y Comparison of neuroendocrine activation in patients with left ventricular dysfunction with and without congestive heart failure. A substudy of the Studies of Left Ventricular Dysfunction (SOLVD). Circulation 1990:82:1724-1729.

47. Garner SH, Sutton JR, Burse RL, McComas AJ, Cymerman A, Houston CS. Operation Everest 11: Neuromuscular performance under conditions of extreme simulated altitude. J Appl Physiol 1990:1167-1172.

48. Gibson JNA, Morrison WL, Halliday D, et al. Decrease in human quadriceps muscle protein turnover consequent upon leg mobilisation. Clin Sci 1987;72:503-509.

49. Green HJ. Manifestations and sites of neuromuscular fatigue. Biochem Exerc 1990;21:13-35.

50. Hanson P. Exercise testing and training in patients with chronic heart failure. Med Sci Sports Exerc 1994;26:527-537.

51. Harridge SD. Magnusson G, Gordon A. Skeletal muscle contractile characteristics and fatigue resistance in patients with chronic heart failure. Eur Heart J 1996, 17 (6): 896-901.

52. Harrrington D, Clark AL. Chua TP Anker SD, Poole-Wilson PA, Coats AJ. Effect of reduced muscle bulk on the ventilatory repsonse to exercise in chronic congestive heart failure secondary to idiopathic dilated and ischemic cardiomyopathy. Am J Cardiol 1997; 80 (1): 90-93.

53. Higginbotham MB, Morris KG, Conn EH. Coleman RE. Cobb FR. Determinants of variable exercise performance among patients with severe left ventricular dysfunction. Am J Cardiol 1983;51:52-60 .

54. Hochachka PW. Dunn JF. Metabolic arrest: The most effective means of protecting tissues against hypoxia. in Sutton JR, Houston CS. Jones NL (eds): Hypoxia. Exercise, and Altitude. New York, Alan R Liss. 1983: 297-309.

55. Hoffman A, Duba J, Lenyyel M. Majer K. The effect of training on physical working capacity of MI patients with left ventricular dysfunction. Eur Heart J 1987;88:43-49.

56. Jondeau G. Katz SD, Zohman L, Goldberger M, McCarthy M, Boudarias JP. LeJemtel TH. Active skeletal muscle mass and cardiopulmonary reserve. Failure to attain peak aerobic capacity during maximal bicycle exercise in patients with severe congestive heart failure. Circulation 1992;86:1351-1356.

57. Karpati G, Carpenter S, Labrisseau A, Lafontaine R. The Kearns-Shy syndrome. A multidiseuse with mitochondrial abnormality demonstrated in skeletal muscle and skin. J Neurosci 1973:19:133-151.

58. Kassis E. Baroreflex control of circulation in patients with congestive heart failure. Thesis. Dan Med Bull 1989:36:195-211.

59. Katz S, Wahl J, Troha J. Specific phosphodiesterase inhibition and maximal and submaximal exercise performance in patients with congestive heart failure. J Cardiov Phar 1989:14:S45-S48.

60. Kellerman JJ, Ben-Ari E, Fisman E, Hayet M, Drory Y, Haimovitz D. Physical training in patients with ventricular impairment. Adv Cardiol 1986:34:131-147.

61. Kempeneers G, Myburgh KH, Wiggins T, Adams B, Van Zyl-Smit R, Noakes TD. Skeletal muscle factors limiting exercise tolerance of renal transplant recipients: Effect of a graded exercise training programme. Am J Kidney Dis 1990;14:57-65.

62. Kitzman DW Higginbotham MB, Cobb FR, Sheikh KH, Sullivan MJ. Exercise intolerance in patients with heart failure and preserved left ventricular systolic function: failure of the Frank-Starling mechanism. J Am Coll Cardiol 1991;17:1065-1072.

63. Kitzman DW. Sullivan MJ. Cobb FR. Higginbotham MB. Exercise cardiac output declines with advancing age in normal subjects. J Am Coll Cardiol 1989:13:241A.

64. Kremser CB. O'Toole ME, Leff AR. Oscillatory hyperventilation in severe heart failure secondary to idiopathic dilated cardiomyopathy or ischemic cardiomyopathy. Am J Cardiol 1987;59:900-905.

65. Kupper W. Interrupting the adaptive changes in congestive heart failure. Am J Cardiol 1991:67:20C-22C.

66. Lange RL, Hecht HH. The mechanisms of Cheyne-Stokes respiration. J Clin Invest 1962;41:42-52.

67. LeJemtel TH, Maskin CS. Lucido D. Chadwisk CJ. Failure to augment maximal blood flow in response to one-leg versus two-leg exercise in patients with severe heart failure. Circulation 1986:74:245-251.

68. Lerman A, Sandok EK, Hildebrand FL. Burnett JC Jr. Inhibition of endotheli-

um-derived relaxing factor enhances endothelin-mediated vasoconstriction. Circulation 1992;85:1894-1898.

69. Levine B, Kalman J, Mayer L, Fillit H, Packer M. Elevated Circulationulating levels of tumor necrosis factor in severe chronic heart failure. N Engl J Med 1990;323:236-241.

70. Lewis T. Discussion of the heart. London: MacMillan 1933:1-2.

71. Lipkin DP, Jones DA, Round JM, Poole-Wilson P Abnormalities of skeletal muscle in patients with chronic heart failure. Int J Cardiol 1988;18:187-195.

72. Lipkin DP, Perrins J, Poole-Wilson PA. Respiratory gas exchange: the assessment of patients with impaired ventricular function. Br Heart J 1985;l54:321-328.

73. Longhurst J, Capone R, Zelis R. Evaluation of skeletal muscle capillary basement membrane thickness in congestive heart failure. Chest 1975;67:195-198.

74. Longhurst J, Gifford W Zelis R. Impaired forearm oxygen consumption during static exercise in patients with congestive heart failure. Circulation 1976;54:477-480.

75. Mack G, Nose H, Nadel ER. Role of cardiopulmonary baroreflexes during dynamic exercise. J Appl Physiol 1988;65:1827-1832.

76. Magnusson G, Isberg B, Karlberg K, Sylven C. Skeletal muscle strength and endurance in chronic congestive heart failure secondary to idiopathic dilated cardiomyopathy. Am J Cardiol 1994;73;307-309.

77. Magnusson G, Kaijser L, Sylven C, Karlberg KE, Isberg B, Saltin B. Peak skeletal muscle perfusion is maintained in patients with chronic heart failure when only a small muscle mass is exercised. Cardiovasc Res 1997; 33 (2): 297-306.

78. Mancini D, Coyle E, Ferraro N. Skeletal muscle metabolic abnormalities in heart failure are in part due to intrinsic skeletal muscle changes. J Am Coll Cardiol 1989;13:39A.

79. Mancini DM, Davis L, Wexler JP, Chadwick B, LeJemtel TH. Dependence of enhanced maximal exercise performance in increased peak skeletal muscle perfusion during long term captopril in heart failure. J Am Coll Cardiol 1987;10:845-850.

80. Mancini DM, Eisen H, Kussmaul W, Mull R, Edmunds LH, Wilson JR. Value of peak oxygen consumption for optimal timing of cardiac transplantation in ambulatory patients with heart failure. Circulation 1991:83:778-786.

81. Mancini DM, Ferraro N, Tuchler M, Chance B. Wilson JR. Detection of abnormal calf muscle metabolism in patients with heart failure using phosphorus-31 nuclear magnetic resonance. Am J Cardiol 1988;62:1234-1240.

82. Mancini DM, Walter G, Reichek N, Lenkinski R, McCully KK, Mullen JL, Wilson JR. Contribution of skeletal muscle atrophy to exercise intolerance and altered muscle metabolism in heart failure. Circulation 1992;85:1364-1373.

83. Marie PY, Escanye JM. Brunotte F, Robin B, Walker P, Zannad F Robert J, Gilgenkrantz JM. Skeletal muscle metabolism in the leg during exercise in patients with congestive heart failure. Clin Sci 1990;78:515-519.

84. Marzo KP, Herrmann IIA. Rein A, Mancini D. Acute effect of balloon mitral valvuloplasty on exercise capacity, ventilation and skeletal muscle oxygenation. Circulation 1991;84:II-72.

85. Marzo KP, Wilson JR, Mancini DM. Effects of cardiac transplantation on ventilatory response to exercise. Am J Cardiol 1992;69:547-53.

86. Maskin CS, Forman R, Sonnenblick EH, Frishman WH, LeJemtel TH. Failure of dobutamine to increase exercise capacity despite haemodynamic improvement in severe chronic heart failure. Am J Cardiol 1983;51:177-182.

87. Massie BM, Conway M, Yonge R, Frostick S, Ledingham J, Sleight P, Radda G, Rajagopalan B. Skeletal muscle metabolism during exercise under ischemic conditions in congestive heart failure. Circulation 1988;78:320-326.

88. Massie BM, Conway M, Yonge R, Frostick S, Ledingham J, Sleight P, Radda G, Rajagoplan B. Skeletal muscle metabolism in patients with congestive heart failure: relation to clinical severity and blood flow. Circulation 1987a;76:1009-1019.

89. Massie BM, Conway M, Yonge R, Frostick S, Sleight P Ledingham J, Radda G, Rajagoplan B. 31-P nuclear magnetic resonance evidence of abnormal skeletal muscle metabolism in patients with congestive heart failure. Am J Cardiol 1987b;60:309-315.

90. Massie BM. Exercise tolerance in congestive heart failure: Role of cardiac function, peripheral blood flow, and muscle metabolism and effect of treatment. Am J Med 1988;84:75-82.

91. Maughan RJ. Relationship between muscle strength and muscle cross-sectional area. Sports Med 1984;1:263-269.

92. McGrath BP, Arnolda L, Matthews PG, Jackson B, Jennings G, Kiat H, Johnston CI. Controlled trial of enalapril in congestive heart failure. Br Heart J 1985;54:405-414.

93. McMurray J, Abdullah I, Dargie HJ, Shapiro D. Increased concentrations of tumour necrosis factor and "cachectic" patients with severe chronic heart failure. Br Heart J 1991;66:356-358.

94. Meakins J, Long CNH. Oxygen consumption, oxygen debt and lactic acid in circulatory failure. J Clin Invest 1927;4:273-293.

95. Meiler SE. Ashton JJ, Moeschberger ML, Unverferth DV, Leier CV. An analysis of the determinants of exercise performance in congestive heart failure. Am Heart J 1987;113:1207-1217.

96. Minotti JR, Christoph I, Oka RK. Weiner MW, Wells L, Massie BM. Impaired skeletal muscle function in patients with congestive heart failure: relationship to systemic exercise performance. J Clin Invest 1991a;88:2077-2082.

97. Minotti JR, Christoph I, Oka RK. Weiner MW, Wells L, Massie BM. Significance of muscle atrophy in heart failure. Circulation 1991b;84:597.

98. Minotti JR, Pillay P, Chang L, Wells L, Massie BM. Neurophysiological assessment of skeletal muscle fatigue in patients with congestive heart failure. Circulation 1992;86:903-908.

99. Minotti JR. Pillay P, Oka R, Wells L, Christoph I, Massie BM. Skeletal muscle size: relationship to muscle function in heart failure. J Appl Physiol 1993;75:373-381.

100. Miyagi K, Asanoi H, Ishizaka S, Kameyama T. Sasayama S. Limited value of anaerobic threshold for assessing functional capacity in patients with heart failure. Clin Cardiol 1993:16:133-137.

101. Moore DP, Weston AR, Hughes JMB, Oakley CM, Cleland JGF. Effects of increased inspired oxygen concentrations on exercise performance in chronic heart failure. Lancet 1992:339:850-953.

102. Morrison WL. Gibson JNA. Johnston RN, Yacoub M, Rennie MJ. Improved whole body protein turnover following heart and lung transplantation. Lancet 1988;ii:853-854.

103. Muller AF, Batin P, Evans S, Hawkins M, Cowley AJ. Regional blood flow in chronic heart failure: the reason for the lack of correlation between patients' exercise tolerance and cardiac output? Br Heart J 1992;67:478-481.

104. Myers J, Froeicher VF. Hemodynamic determinants of exercise capacity in chronic heart failure. Ann Internal Med 1991;115:377-386.

105. Myers J, Salleh A, Buchanan N, Smith D, Neutel J, Bowes E, Froelicher VF. Ventilatory mechanisms of exercise intolerance in chronic heart failure. Am Heart J 1992;124:710.

106. Nishimura Y, Maeda H, Tanaka K, Nakamura H, Hashimoto Y, Yokoyama M. Respiratory muscle strength and haemodynamics in chronic heart failure. Chest 1994;105:355-359.

107. Noakes TD. Implications of exercise testing for prediction of athletic performance: a contemporary perspective. Med Sci Sports Exerc 1988;20:319-330.

108. Ogino K, Osaki S, Kitamura H, Noyuchi N, Hisatome I, Matsumoto T, Omodani H, Kato M, Kinugawa T, Miyakoda H, Kotake H, Mashiba H. Ammonia response to exercise in patients with congestive heart failure. Heart 1996;75(4):343-348.

109. Olson W, Engel WK, Walsh GO, Einaugler R. Oculacraniosomatic neuromuscular disease with 'ragged red' fibres. Arch Neurol Exp Neurol 1972;26:193-211.

110. Packer M. Treatment of chronic heart failure. Lancet 1992b;340:91-95.

111. Patterson JA, Naughton J, Peitras RM. Treadmill exercise in assessment of the functional capacity of patients with cardiac disease. Am J Cardiol 1972;30:757-762.

112. Peterson H, Rothschild M, Weinberg C, Fell R, McLeisch K, PfeiferM. Body fat and the activity of the autonomous nervous system. N Eng J Med 1988:318:1077-1083.

113. Pfeffer MA, Braunwald E, Moyle LA. et al. Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction: results of the Survival ad vetricular Enlargement Trial. N Engl J Med 1992;327;669-677.

114. Poole-Wilson PA, Nigel PB, Lipkin DP Regional blood flow. muscle strength and skeletal muscle histology in severe congestive heart failure. Am J Cardiol 1988;62: 49E-52E.

115. Port S, McEwan P, Cobb FR, Jones RH. Influence of right ventricular function on left ventricular response to exercise in patients with coronary artery disease. Circulation 1981: 63:856-863.

116. Ralston MA, Merola AJ, Leier CV. Depressed aerobic enzyme activity of skeletal muscle in severe chronic heart failure. J Lab Clin Med 1991;117:370-372.

117. Reading JL; Goodman JM; Plyley MJ; Floras JS; Liu PP; McLaughlin PR; Shephard RJ. Vascular conductance and aerobic power in sedentary and active subjects and heart failure patients. J Appl Physiol. 1993 Feb, 74:2; 567-73

118. Reddy HK, Weber KT, Janicki JS. McElroy PA. Light isometric exercise in patients with chronic heart failure. J Am Coll Cardiol 1988;12:353-358.

119. Ribiero JP, Knutzen A, Rocco MB, Hartley LH, Colucci WS. Periodic breathing during exercise in severe heart failure. Chest 1987;92:555-556.

120. Riley M. Elborn JS, Bell N. Substrate utilization during exercise in chronic cardiac failure. Clin Sci 1990:79:89-95.

121. Rodeheffer R. Lerman A, Heublein DH, Burnett JC Jr. Plasma endothelin concentrations are increased in humans with congestive heart failure. Mayo Clin Proc 1992;67:719-724.

122. Roubin GS. Anderson SD, Shen WF. Choong CY, Alwyn M, Hillery S. Harris P, Kelly DT. Haemodynamic and metabolic basis of impaired exercise tolerance in systemic LV dysfunction. J Am Coll Cardiol 1990;15:986-994.

123. Rubin S. Swan HJC. Response to exercise of patients with severe chronic heart failure. Biblthca Cardiol 1986;40:52-60.

124. Saltin B. Gollnick PD. Skeletal muscle adaptability: Significance for metabolism and performance, in Handbook of Skeletal Muscle. Bethesda. Md. American Physiological Society 1988;589-596.

125. Schantz P, Randall-Fox E. Hutchinson W, Tyden A, Astrand PO. Muscle fibre type distribution, muscle cross sectional area and maximal voluntary strength in humans. Acta Physiol Scand 1983:117:219-226.

126. Schaufelberger M, Erikson BO. Held P, Swedberg K. Skeletal muscle metabolism during exercise in patients with chronic heart failure. Heart 1996; 76 (1): 29-34.

127. Sinoway LI. Minotti JR. Dwight D. Pencock JL, Burg JE, Musch TI, Zelis R. Delayed reversal of impaired vasodilation in congestive heart failure after heart transplantation. Am J Cardiol 1988;61;1076-1079.

128. SOLVD Investigators. Effect of enalapril on mortality and the development of heart failure in asymptomatic patients with reduced left ventricular ejection fractions. N Engl J Med 1992:327:685-691.

129. Stingo A, Clavell A, Aarhus L, Burnett JC Jr. Cardiovascular and renal actions of C-type natriuretic peptide. Am J Physiol 1992:262:11308-H312.

130. Strandell T, Shepherd JT. The effect in humans of increased sympathetic activity on the blood flow to active muscles. Acta Med Scand Suppl 1967;472:146-167.

131. Stratton JR, Dunn JF, Adamopoulos S. Kemp GJ, Coats AJS, Rajagopalan B. Training partially reverses skeletal muscle metabolic abnormalities during exercise in heart failure. J Appl Physiol 1994;76(4):1575-1582.

132. Sullivan J. Saltin B. Klitgaard H. Cobb FR. Altered expression of myosin heavy chain isoforms in skeletal muscle in patients with chronic heart failure. Circulation (abstr) 1990b;82:4.

133. Sullivan MJ. Green HJ. Cobb FR. Skeletal muscle biochemistry and histology

# Performance indicators of elite beach volleyball players

SEH Davies * MA
MF Coetsee * PhD
M Siroky MBBCh MSc (Med)
L Leach ** MA
A Travill ** MA
* Department of Human Movement Science, University of Zululand
** Department of Human Movement Science, University of the Western Cape

## ABSTRACT

**Objectives:** This study analyzed heart rate, blood lactate and motion patterns of South African (SA) and Overseas (OS) professional beach volleyball players. **Methods:** Thirty two subjects participating in the World Beach Series, Cape Town, 1995, volunteered with written informed consent (SA=20; OS=12). Anthropometric measures included stature, mass and percentage body fat. Physiological parameters included heart rate monitoring during match-play recorded by the Polar Heart Watch Sports Tester. Blood lactate was measured after match-play using the Acusport Blood Lactate Analyzer. Motion analysis was recorded during match-play and included; jump serve, serve, block, dig, volley, run and dive. This information was synchronised with the heart rate data. **Results:** Results indicated that OS players were significantly (p<0.05) older, taller, heavier and lower in percentage body fat, compared to SA players. Mean heart rate (beats.min⁻¹) during match-play was not significantly different (SA=157.94 vs OS=155.66) neither was maximal heart rate (SA=182.20 vs OS=180.66). No significant difference was observed in post-match blood lactate concentrations (mmol⁻¹) for both groups (SA=3.83 vs OS=2.85). The frequency of motion technique utilization was similar for SA and OS players with the exception of service and volley technique (p<0.05). Thus, SA players used the standing serve and volley significantly more often than OS players, while conversely the OS players used the jump serve more frequently than SA players. The playing surface of beach volleyball (sand) and a "2-man team" appeared to increase the energy cost of play relative to the indoor code (hard surface) and "6-man team". Rallies were relatively short 5.39 secs alternating with non-rally (rest) periods of 19.08 secs. These temporal characteristics suggest that beach volleyball makes predominant demands on the ATP-CP and glycolytic energy systems during rally-play, while the aerobic system sustains low intensity motion during non-rally play and concomitantly replenishes ATP-CP stores. **Conclusions:** The results from this study indicate that

## CORRESPONDENCE:

Mr SEH Davies
Dept of Human Movement Science
University of Zululand
Kwa Dlangezwa, 3886
South Africa
Tel/Fax - 27 - 351 - 93916
email - sdavies@pan.uzulu.ac.za

OS players were significantly older, taller, heavier and lower in % body fat, and utilized the jump serve more than SA players. Indicators of physical exertion, namely mean and maximal heart rates were similar for SA and OS players, indicative of comparable energy expenditure during match-play. Mean rally length (5.39 secs) and non-rally (19.08 secs) were respectively shorter and longer in duration than the values reported during indoor volleyball match-play.

## INTRODUCTION

There appears to be a paucity of research regarding the sport of "2 Man" beach volleyball. This study sought to redress this shortcoming by examining heart rate, blood lactate and motion analysis of participants at the World Series Beach Volleyball Championships, Cape Town 1995.

Although beach volleyball may be construed to be an emerging sport in comparison to other sports, including "6 versus 6" indoor volleyball, it has however gained increasing importance at both recreational and professional levels. This is perhaps best reflected by the inclusion of beach volleyball at the 1996 Olympic Games, and the '1996 World Series' offering 4.5 million US Dollars in prize money.

Beach volleyball would appear to be a sport characterised by short periods of intense physical activity, followed by longer periods of less intense activity. Motion analysis of patterns of play for beach volleyball appear to be lacking, although this has been well researched for the indoor code,[1,2,3,4] along with other sporting codes such as rugby,[5] soccer,[6] handball,[7] waterpolo,[8] and ice-hockey goal tending.[9]

Physiological studies of the indoor code have identified the predominant energy requirements during match-play. Smith et al.[10] consider the indoor game to be aerobic in nature, combined with a high anaerobic component. Conlee et al.[11] investigated glycogen depletion by muscle fibre staining as a result of volleyball match-play. They reported after examining pre- and post-match muscle staining of slow and fast twitch muscle fibres, the mean percentage depletion of glycogen in slow twitch fibres was 36% as compared to 6.5% in fast twitch fibres. This may suggest that the increased glycogen depletion of slow twitch fibres as compared to fast twitch fibres in this study illustrates a significant involvement of the aerobic energy pathways during volleyball match-play.[11] Kunstlinger et al.[12] reported elevated concentrations of free fatty acids after volleyball match-play, implying an increased contribution by the oxidative metabolism in well-trained players. Indications of energy production by the anaerobic pathways during the dynamic high intensity movements including spikes and blocks were shown by

changes in electrolyte and catecholamine levels.[12]

Smith et al.[10] considered that during international indoor volleyball matches, fitness, physical characteristics and skill are the key factors determining performance. This may also be the case for beach volleyball for there is little doubt that the underlying principles of match-play for the two volleyball codes are similar. However the two codes are distinct from each other with regard to the following factors. Beach volleyball is a 2-aside game, while indoor volleyball is 6-aside, but both are played on a court 9m by 18m. The playing surface of beach volleyball is sand, a displaceable/compliant substrate, which raises the metabolic cost of motion by as much as 100% compared to a hard surface.[13]

Climatic and geographic factors are also likely to affect performance. This is because the major beach volleyball competitions in the world, including the World Beach Volleyball Series take place almost exclusively during the summer months. The combination of several strenuous games in a day with relatively high temperatures and humidity, in energy sapping sand is likely to raise the metabolic cost of playing, and increase the risk of dehydration.[14,15] Murray makes the observation that when a competitive psyche from a cool, temperate climate meets a warm and humid environment without considering acclimatization, the results can be disastrous for both performance and health.[16]

The World Series Beach Volleyball Tour was staged in Cape Town, 1995, attracting many of the best players from both South Africa and Overseas. Participants were studied to identify anthropometric characteristics and motion-patterns. Physiological demand on the players was assessed using continuous heart rate monitoring and postmatch blood lactate concentrations. The organisation of the World Series allows the host nation to enter significantly more teams (n=15) than would normally occur. This meant that subject numbers allowed for a comparison between South African (SA) and Overseas (OS) players. The aim of this study is to identify anthropometric and physiological characteristics of successful players, as well as their preferred patterns of play.

## METHODS

20 South African (SA) and 12 Overseas (OS) professional beach volleyball players participated in this study with written informed consent.

Climatic parameters included dry and wet bulb temperature readings (°C) along with wind speed measures (m.sec⁻¹). A slingpsychrometer was rotated three minutes prior to each match in a shaded area for one minute whereupon dry and wet bulb temperature readings were recorded. Relative humidity was derived from a psychometric chart conversion of dry and wet bulb readings.[17] Wind speed data was collected by a TurboMeter™ wind speed indicator, Davis Instruments, Hayward, California, USA, sampling wind speed (m.sec⁻¹) every ten seconds for two minutes prior to the match with the mean value being recorded. Matches were played at either Camps Bay beach or on the man-made courts at the Waterfront in Cape Town, site of the World Series.

*Anthropometric measures* included stature and body mass as described by Tanner.[18] Percentage body fat was calculated from skinfold measures of four sites, namely biceps, triceps, supra iliac and subscapular

according to the method of Durnin and Womersley.[19]

*Physiological measures* involved the recording of heart rate during competitive matchplay and blood lactate sampling immediately after the game. Heart rate was measured by the telemetric Polar Heart Watch 'Sports Tester', a portable heart-rate monitor set at 5 second record intervals. The transmitter and electrode strap was placed around the subject's chest at the level of the inferior border of the pectoralis muscles. The 'watch'/receiver was located on the back of the subject, attached to the electrode strap between the scapulae. The reason for not wearing the 'watch'/receiver on the wrist, was to avoid high impact with the ball during spiking, blocking, digging and diving actions. It was anticipated that this high impact might damage the 'watch'/receiver itself and distort the telemetric transmission. After the game the heart rate data was downloaded onto a computer where it was analyzed via the Polar Heart Rate Analysis Software.[20] An example of the graphic representation of heart rate response during a game can be observed in Figure 1.

Blood lactate was analyzed using the Acusport Blood Lactate Analyzer from Bohringer. Within 2 minutes of the conclusion of a match the finger was pricked with a lancet, the suspended globule of blood was allowed to drop onto the test strip, which was then inserted into the Acusport for analysis.[21]

*Motion analysis* was recorded during actual play. A recorder observed one player during a game and noted down on a running spread sheet every major motion that the player performed. The different patterns of play were synchronised with heart rate data in order to study the physical strain associated with each major motion. Eight major motions were noted, namely: Jump Serve, a serve whereby the player jumps up in
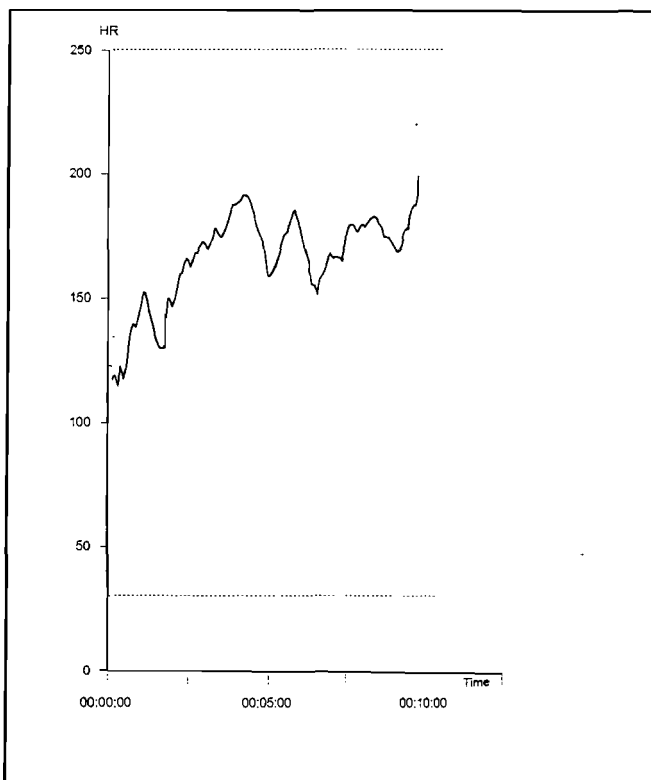


Figure 1: *An example of a smoothed heart rate plot during match-play*

the air and hits the ball hard with heavy top spin; Serve, a regular tennis style action where the player stands and strikes the ball above his head; Block, a defensive action where a player jumps at the net and attempts to stop an offensive action (hit/spike) by presenting a barrier with both arms out stretched above the net band; Dig, a passing technique where the player plays the ball with clasped hands and straight arms off the forearms; Volley, where the ball is played above the head with the fingers of both hands in a clean manner; Hit/Spike, an attacking shot where the player jumps as high as possible at the net, enabling him to hit/spike the ball down into the opponent's court; Run, when a player runs more than 3m to chase or retrieve a ball; Dive, when a player in order to play the ball is forced to throw himself or sprawl in the sand. Video recordings were taken of match-play as a back up to the insitu motion analysis recording.

*Statistical analysis* was performed by the Statgraphics software package, Version 5.[32] Summary statistical analysis included means and standard deviations, along with One-Way Analysis of Variance (ANOVA) at $p < 0.05$.

## RESULTS

*Anthropometric data* of both SA and OS players are shown in Table 1. It was noteworthy that the OS players were significantly ($p < 0.05$) taller, heavier, lower in percentage body fat and older when compared to SA players.

*Physiological parameters* in Table II show the means and standard deviations of total, maximal and mean heart rates during match play. It was interesting to note that there is little difference between the maximal and mean heart rates of SA and OS players.

There was a significant difference between the two groups with regard to total heart rate, and this was directly related to the difference in the mean length of the games of the two groups analyzed (SA=25.18 mins and OS=37.45 mins). Blood lactate concentrations 2 minutes after the game showed no significant difference between SA and OS players (Table II).

*Climatic conditions* were ascertained by measurement of wet and dry bulb temperature, wind speed and relative humidity (Table III). The climatic conditions experienced by both SA and OS players during match-play were not significantly different.

*Motion analysis* data can be observed in Table IV which indicates the mean time-lapse between specific motions. This is a way of presenting the frequency at which each motion technique was utilized. SA and OS players had similar frequency rates for the block, dig, hit/spike, run and dive. The two groups differed with regard to the type of serve used and to a lesser extent the volley technique. Table IV also shows the mean time lapse (secs) between all motion techniques (SA=20.58 secs vs OS=20.78 secs), as well as between high intensity motions (SA=41.41 secs vs OS=38.22 secs), which includes jump serve, block, hit/spike, run and dive. The time-lapse between all monitored motions and high intensity motions were comparable for SA and OS players.

TABLE I: Means and Standard Deviations anthropometric data of South African (SA) and Overseas (OS) players.

|  | SA (n = 20) | OS (n = 12) |  |
|---|---|---|---|
| Stature (cm) | 184.94 (5.9) | 190.75 (5.49) | * |
| Mass (kg) | 77.67 (8.14) | 88.66 (3.47) | * |
| % Body Fat | 15.95 (3.09) | 12.38 (4.55) | * |

* indicates a statistical difference at $p < 0.05$

TABLE II: Means and Standard Deviations for total (beats), maximal and mean heart rate (beats.min[-1]), and blood lactate (mmol.l[-1]) of South African (SA) and Overseas (OS) players during match play.

|  | SA (n = 20) | OS (n = 12) |
|---|---|---|
| Total HR | 4135.47 (1190.82) | 6193.17 (1188.41)* |
| Max HR | 182.20 (12.41) | 180.66 (8.60) |
| Mean HR | 157.94 (12.68) | 155.66 (13.58) |
| Lactate | 3.83 (2.48) | 2.85 (1.60) |

* indicates a statistical difference at $p < 0.05$

TABLE III: Means and Standard Deviations for wet bulb, dry bulb (°C) and wind speed (m.sec[-1]), along with relative humidity (%) encountered by South African (SA) and Overseas (OS) players during match play.

|  | SA (n = 20) | OS (n = 12) |
|---|---|---|
| W. Bulb | 20.21 (3.01) | 20.75 (3.81) |
| D. Bulb | 26.73 (6.15) | 27.50 (6.31) |
| W. Speed | 1.76 (1.01) | 1.41 (1.22) |
| Humidity | 50 | 50 |

* indicates a significant difference at $p < 0.05$

TABLE IV: Mean time-lapse between individual motion (mins) and combined motion frequency (secs) of South African (SA) and Overseas (OS) players during match-play.

|  | SA (n=20) | OS (n=12) |  |
|---|---|---|---|
| Jump serve (mins) | 4.24 | 2.51 | * |
| Serve (mins) | 3.08 | 5.19 | * |
| Block (mins) | 3.03 | 2.40 | |
| Dig (mins) | 1.21 | 1.24 | |
| Volley (mins) | 2.37 | 3.37 | * |
| Hit/spike (mins) | 1.42 | 1.44 | |
| Run (mins) | 3.07 | 3.25 | |
| Dive (mins) | 6.27 | 6.28 | |
| All Motion (secs) | 20.97 | 20.78 | |
| High intensity motion (secs) | 41.69 | 38.22 | |

* indicates a significant difference at $p < 0.05$

Table V shows previous studies of temporal analyses during indoor 6 aside match-play and the present study of beach volleyball. The rallies during beach volleyball matchplay are generally shorter (5.39 secs), while rest periods (non-rally match play) are generally longer (19.08 secs) than for the indoor code.

Table V: Temporal analyses of indoor volleyball (1979 - 1987) and beach volleyball (present study) with regard to mean rally length, rest duration (secs) and rally-rest ratio.

|  | Lecompte & Rivet (1979) | Fiedler (1979) | Dyba (1982) | Viitasalo et al (1987) | Present study |
|---|---|---|---|---|---|
| Mean duration of rally | 9.7 | 9.0 | 7.0 | 6.6 | 5.39 |
| Mean duration of rest | 11.7 | 12.0 | 13.3 | 14.2 | 19.08 |
| Rally : rest ratio | 1:1.2 | 1:1.3 | 1:1.2 | 1:2.2 | 1:3.5 |

## DISCUSSION

It has been observed in previous studies that temperature and relative humidity can have a significant effect on selected physiological responses including heart rate and levels of blood lactate during exercise.[14,15] It was clear that temperature and relative humidity experienced by the two groups namely SA and OS beach volleyball players was very similar, and therefore did not confound or skew the data (Table III).

SA and OS players were significantly ($p<0.05$) different in terms of stature, body mass and percentage body fat, with the OS players being taller, heavier and leaner. Previous studies of the indoor code suggest that elite players have greater stature, higher body mass and lower percentage body fat, when compared to lower standard performers.[10] In order to hit/spike and block effectively it is necessary to be able to play the ball above the net, therefore greater stature is advantageous. Jumping ability also plays an important part when attempting to hit/spike or block the ball. The OS players were in most cases experienced members of the World Series tour and were more likely to be full-time professionals, while the SA players were in most cases semi-professional, whose experience of pro-beach volleyball was largely restricted to domestic competitions. Greater frequency of high level tournaments at international level of the OS players has probably resulted in greater concern about dietary intake and training preparation with regard to optimising performance, and could account for the lower percentage body fat of these players when compared to SA players.

The maximal heart rate (beats.min$^{-1}$) for the two groups was highly comparable (SA=182.2 vs OS=180.66) along with mean heart rate (SA=157.9 vs OS=155.6). This would appear to imply that SA and OS players have similar peak and mean levels of exertion during match-play, as indicated by heart rate response. Previous studies of heart rate during indoor volleyball match play show it to be less strenuous than the findings in this study, with the following average heart rates (beats. min$^{-1}$) reported: 110-125,[23] 155,[24] 139,[25] 144,[3] and 127.[4]

The only major divergence in heart rate analysis concerned total heart rate per game, and this is linked unquestionably to game length. The OS players spent a significantly ($p<0.05$) longer period on court than their SA counterparts, thus their greater total heart rate (SA=4135.47 beats.match$^{-1}$ vs OS=6139.17 beats. match$^{-1}$).

The post match concentrations of blood lactate for SA and OS players (3.83 and 2.85 mmol.l$^{-1}$, respectively) were relatively low and concur with similar findings reported for indoor volleyball of 2.57 mmol.l$^{-1}$ and 0.85 mmol.l$^{-1}$.[11,12] These observations indicate that during the rally-phases of both beach and indoor volleyball, energy is predominantly supplied by phosphogen breakdown (ATP/CP) for the first few (2-3) seconds of work, coupled by an increasing contribution by glycolysis as the exercise duration lengthens. Thus, there is an interplay between all three energy systems (ATP/CP, glycolysis and oxidative metabolism) to provide energy for work. During less intense phases of play, namely non-rally phases the oxidative metabolism replenishes depleted ATP/CP stores. This contention appears to be supported by the findings of Essen and Kaijser[27] who reported that during intermittent exercise of 15 seconds work and 15 seconds rest, the ATP/CP concentrations were largely replenished during the recovery phase. This oscillating demand made upon the energy systems of the body is a reflection of the intermittent nature of beach volleyball. The work to rest ratios in this study were 1:3.5, comprising on average a rally-phase of 5.39 seconds and a non-rally-phase (recovery/partial recovery) of 19.08 seconds. The non-rally phases would also appear to facilitate the removal of lactate which may have accumulated during prolonged rally-phases where one would assume that an increased demand had been made upon glycolysis.[26]

Motion analysis (Table IV) focused on the mean frequency at which each motion was performed by identifying mean time-lapse (minutes/seconds) between each defined motion. The time-lapse between all of the identified motions by SA and OS players was relatively comparable with the exception of the service and volley technique. Thus, SA players performed a jump serve on average every 4.24 minutes, while OS players performed the same motion significantly ($p<0.05$) more often every 2.51 minutes. Conversely SA players performed the standing serve more frequently, once every 3.08 minutes, while OS players did so every 5.19 minutes. The SA players used the volley technique every 2.37 minutes, where as the OS did so only every 3.37 minutes, this was significantly different ($p<0.05$). The average frequency at which all motions were performed in relation to game length was once every 20.97 seconds for SA players and once every 20.78 seconds for OS players (Table IV). High intensity motion techniques such as the jump serve, block, hit/spike, run and dive occurred on average less frequently; SA players, performed one every 41.69 seconds and OS players one every 38.22 seconds. This would indicate that the pace at which elite beach volleyball was played, was similar for both SA and OS players and provides an explanation for the parity of mean and maximal

heart rate values exhibited by both groups.

The average length of a rally was 5.39 seconds (SA and OS players)(Table V). This appears to be considerably shorter than reported mean rally length for the indoor code, characterised by average rally lengths of 9.7 secs,[1] 9.0 secs,[2] 7.0 secs[3] and 6.6 secs.[4] Conversely the average duration of the non-rally phase was longer for beach volleyball at 19.08 seconds, when compared to the indoor code, where rest times of 11.7 secs,[1] 12.0 secs[2] 13.3 secs[3] and 14.2 secs[4] have been reported (Table V). This indicates that beach volleyball has shorter rallies and longer rest periods (work to rest ratio = 1:3.5), as compared to the indoor game where (work to rest ratio varied between = 1:1.2,[1] 1:1.3,[2] 1:1.2[3] and 1:2.2,[4]) even though the general physiological exertion as indicated by heart rate was higher. An explanation of the substantially longer non-rally periods can be attributed to more permissable time outs (4 vs 2 per team compared to the indoor code) and end changes after every 5 points, which is not a feature of indoor volleyball and involves both teams having to walk and occupy 'opposing' ends. An explanation for the higher mean heart rate levels was probably due to the fact that only 2 players form a beach volleyball team as opposed to 6 players for the indoor code on a court the same size. Also the sand would appear to 'slow' down the speed of players around the court, as well as inducing increased energy cost.[13] These factors probably diminish the likelihood of repeated exchanges (rallies) between teams, and result in shorter rallies than those reported for indoor volleyball. Another factor concerns the increased difficulty involved when defending against a typical power offence (hit/spike) in beach volleyball when compared to the indoor code. It is clear that a team with six players defending against one person hitting/spiking, typified by a two man block and four other players covering the rest of the court, are more likely to be successful than two players attempting the same task. Thus the numerical advantage that a 6-aside team has over a 2-aside beach volleyball team, improves their defensive capabilities and contributes to longer rallies.

## CONCLUSION

The data from this study indicate that beach volleyball is made up of short intense sequences of motion (rally-phases) that are on average 5.39 seconds in duration indicative of a predominant provision of energy by the ATP/CP systems for the initial phase of high intensity activity (2-3 seconds), along with glycolysis and the oxidative metabolism. These 'rally-phases' alternated with recovery 'non-rally-phases' which were on average 19.08 seconds in duration, and allowed for the replenishment of ATP/CP by the aerobic metabolism. Relatively low concentrations of blood lactate (3.34 mmol.l[-1]) found after match-play are probably due to the intermittent nature of beach volleyball. Thus, non-rally-phases which were on average 19.08 seconds provided sufficient time for the removal of any lactate that may have accumulated during rally phases. The aerobic energy system was utilised extensively, especially during non-rally-phases, when the players performed low and medium intensity motion on sand such as standing, walking and jogging. The aerobic energy system concomitantly replenished ATP/CP stores depleted during intense phases of match-play. The overall increased energy cost of beach volleyball was reflected in higher mean (159.5 beats.min[-1]) and maximal (181.43 beats.min[-1]) heart rate values relative to those found for the indoor code.

Motion analysis of the major techniques utilised in beach volleyball indicated that both SA and OS utilized similar motion techniques at comparable frequencies, with the notable exception of service and volley technique. The difference in the use of the volley pass, which is usually employed to set the ball up for an attack may be explained by the fact that some of the OS players were previously specialist international indoor volleyball players, whose roles rarely required them to volley. The beach volleyball rules on volleying are strict, and it is likely that the OS players in this study used the volley only when they had to, so as to avoid being penalized for handling errors. Apart from the type of service employed and the volley technique the patterns and frequency of play of the SA and OS players were highly comparable. This resulted in similar exertional profiles as indicated by heart rate response and blood lactate levels.

The results of this study should provide coaches and players of beach volleyball with scientifically derived information which can assist in preparation and match-play. Motion analysis highlighted desirable motion patterns of elite professional beach volleyball players, along with an insight into the exertional demands of the game. These factors should serve to improve the standard of performance, particularly for those players who are involved at competitive levels.

## REFERENCES

1. Lecompte JC and Rivet D (1979). Tabulated data on the duration of exchanges and stops in a volleyball game. Volleyball Technical Journal, 4(3):87-91.
2. Fiedler M (1979). Volleyball. Ost, Berlin.
3. Dyba W (1982). Physiological and activity characteristics of volleyball. Volleyball Technical Journal, 6(3):33-51.
4. Viitasalo J, Rusko H, Pajala O, Rahkila P, Ahila M, Mononen H (1987). Endurance requirements in volleyball. Canadian Journal of Applied Sports Science, 12(4):194-201.
5. Docherty D, Wenger HA, Neary P (1988). Time-motion analysis related to the physiological demands of rugby. Journal of Human Movement Studies, 14:269-277.
6. Mayhew SR and Wenger HA (1985). Time-motion analysis of professional soccer. Journal of Human Movement Studies, 11:49-52.
7. Alexander MJL and Boreskie SL (1989). An analysis of fitness and time-motion characteristics of handball. The American Journal of Sports Medicine, 17(1):76-82.
8. Smith H (1991). Time-motion analysis of water polo goal tenders. In: Proceedings of the 24th Annual Meeting of the Canadian Association of Sport Sciences. Kingston, Ontario: Canadian Association of Sport Sciences.
9. Richard JF, Gionet N, Nowlan J (1995). Time-motion analysis of ice-hockey goal tending. Journal of Human Movement Studies, 29:115-131.
10. Smith DG, Roberts D, Watson B (1992). Physical, physiological and performance differences between Canadian National team and Universiade volleyball players. Journal of Sports Sciences, 10:131-138.
11. Conlee RK, McGown CM, Fisher AG, Daisky GP, Robinson KC (1982). Physiological effects of power volleyball. The Physician and Sports medicine, 10:93-97.
12. Kunstlinger U, Ludwig HG, Stegemann J (1987). Metabolic changes during volleyball matches. International Journal of Sports Medicine, 8:315-322.
13. Zamparo P, Perini R, Orizio C, Sacher M, Ferretti G (1992). The energy cost of walking or running on sand. European Journal of Applied Physiology, 65:183-187.
14. McArdle WD, Katch FI and Katch VL (1991). Exercise Physiology: Energy, Nutrition and Human Performance.

# The heart rate response of cricket umpires to on-field events

RA Stretch * D Phil
J Tyler** PhD
S Bassett*** PhD

\* Sport Bureau, University of Port Elizabeth, South Africa.
\** Department of Statistics, University of Fort Hare, South Africa.
\*** Department of Human Movement Studies, University of Fort Hare, South Africa.

## ABSTRACT

**Objectives:** This study assessed the heart rate (HR) responses recorded every 15 seconds, of ten umpires (six provincial and four international umpires) to on-field events during limited-overs cricket matches.
**Methods:** Specific on-field events, including appeals, the umpires resulting decisions and any other tasks the umpire had to make such as calling a no-ball or a wide-ball, were recorded.
**Results:** The results showed that the umpires' ratings of the on-field decisions varied from "below average" to "stressful" with regard to the amount of perceived stress they felt. The international umpires' ratings indicated less perceived stress than the provincial umpires, who showed a far greater variance with regard to LBW, catch, run-out and wide-ball decisions. The mean HR rate before the commencement of the match ($99\pm11.8$ beats min$^{-1}$) and during the first ($100\pm12.4$ beats min$^{-1}$) and second innings ($100\pm8.6$ beats min$^{-1}$) of the match corresponds to approximately 60% of the umpires' estimated maximal HR. The recorded HR varied from 68 beats min$^{-1}$ to 127 beats min$^{-1}$ at the time of the event, with the highest HR (139 beats min$^{-1}$) recorded 15s post event. Non-significant variations in the HR occurred during the various periods of the match for the whole group. The differences (D=-0.7 beats min$^{-1}$) between the heart rate of the international umpires prior to the match and the first session of the first innings was significantly less ($p<0.05$) than the corresponding difference (D=7.5 beats min$^{-1}$) during the same period of the second innings, as well as between the second and the third sessions of the second innings, illustrating greater variation for the inexperienced umpires. Heart rate data of an umpire during a hat-trick (three wickets in three balls) during an international, showed anticipatory increase in HR for each delivery.
**Conclusions:** It can be concluded that the heart rate response of the umpires to on-field events indicate that they are under psychological pressure, with international umpires, although umpiring at a higher level, better able to handle these pressures.

**CORRESPONDENCE:**

Dr Richard Stretch
Sport Bureau
University of Port Elizabeth
PO Box 1600
Port Elizabeth 6001
Tel    - 27 - 41 - 504 2584
Fax    - 27 - 41 - 53 2605
email - sparas@upe.ac.za

## INTRODUCTION

Due to the increased speed of bowlers and the complexity of cricket, the integral role of umpires within the game has necessitated a rise in standard with a high degree of accuracy and consistency. The introduction of the third or "TV" umpire has relieved the on-field umpires of making certain "line decisions" in internationals and Test matches. This has assisted in making the task of the umpire easier with regard to stumping and run-out decisions, as well as in cases where doubt may exist as to whether the ball had crossed the boundary or not. This has, however, necessitated an increase in the number of TV cameras and slow motion replays available to the third umpire, as well as to the commentators and viewers who are able to view the incidence a number of times before making their "decision". However, the on-field umpires have to make split-second decisions without the luxury of these slow motion television replays from a variety of different angles with any mistakes high-lighted by "trial by television" re-plays. As the pressure increases on the umpire, the more likely he is to make the wrong decision with inaccuracy and inconsistency bringing the umpire under tremendous pressure and criticism.

Besides having to implement the laws of the game, the umpire is also required to arbitrate on issues of gamesmanship, "sledging" and personality differences. Umpires are expected to maintain alertness throughout the duration of the match, which may last up to six hours a day for five consecutive days in a Test match. An umpire who performs well goes largely unnoticed, while an umpire who is deemed to have erred is accused of incompetence and even bias. This constitutes immense psychological pressure.

Umpires, due to the fact that they need to gain experience before becoming eligible to umpire at provincial and international level, tend to belong to an age range that is older than the cricket players themselves. Umpires further differ from the players in that they are not professionals, generally holding full-time employment outside of the game of cricket.

HR monitors have primary being used to evaluate the movement patterns and intensity of playing a variety of sports,[1,2,3,4,5,6,7] with researchers having focused very little attention on the match official. Two Australian football field umpires recorded mean heart rates (HR) of 159 beats min$^{-1}$ and 148 beats min$^{-1}$, increasing to 173 beats min$^{-1}$ when having to intervene in a scuffle between players.[8] The resting HR of soccer referees were high (100 beats min$^{-1}$) before a match, averaging 165 beats min$^{-1}$ (95% of the estimated maximal HR) for the duration of the match.[9] In both studies the mean HR was similar for both halves of the match.[8,9]

The task of cricket umpiring was rated as "psychologically demanding" and in order to achieve and maintain high standards, umpires need to be introduced to the educative and performance enhancing applicability of psychological techniques.[10] These include the setting of realistic performance goals, improving communication skills and the development of techniques to improve attention, concentration and confidence.

Therefore, the main aim of this study was to assess the HR response of cricket umpires to on-field events during provincial and international limited-overs matches. These findings would provide a better understanding of the psychological and physiological demands of umpiring, which in turn could improve the mental and physical preparation of the umpires in order to meet specific on-field demands, thereby raising the standard of umpiring.

## METHODS

The subjects consisted of ten umpires who were on the South African Cricket Umpires Association provincial panel. Four of these umpires had umpired at international level, with the other six umpires having umpired at provincial level. Consent was obtained from the South African Cricket Umpires Association and all subjects following an explanation of the investigation.

The data collection took place during three official 50-over day-night provincial fixtures of the United Cricket Board of South Africa and two official 50-over day-night international fixtures during the quadrangular series between South Africa, New Zealand, Sri Lanka and Pakistan. All these matches were played at Buffalo Park, East London.

### Heart rate monitoring

The HR was recorded for the full duration of each match by means of short-range telemetry using the Polar Sport Tester (Vantage XL, Polar USA, Stanford, Connecticut, USA). Rubber electrodes made contact with the skin and were held in position just beneath the pectoral muscles by an elastic strap. The HR signal was transmitted to a microcomputer receiver worn on the wrist. The receivers were programmed to measure and store the HR readings every 15 seconds. At the end of the match these data were downloaded into an IBM PC using an interface and software purchased from the manufacturers (Polar Computer Interface, Polar USA) for later analysis.

The HR monitor was placed on the subject 20 minutes before the start of the match, with the collection of the HR commencing 15 minutes before the start of play. This allowed the subject time to become familiar with the equipment. The collection of HR continued until 5 minutes after the end of the match. The commencement of the collection of the HR data was synchronised with a stop watch in order to relate the HR to specific on-field events which were recorded. These included appeals and resulting decisions made by the umpire, and any other tasks or decisions that the umpire had to make such as calling a no-ball or a wide-ball.

The HR were expressed as the average HR rate for different periods. These were: (i) for the fifteen minutes before the start of the match, (ii) from the start of the match to the first break, (iii) from the first to the second break, (iv) from the second break to the

end of the innings and (v) for five minutes after the end of the innings. This was repeated for the second innings of the match. Maximal heart rates were estimated using the formula 220-Age.[11]

In order to assess the differences between the on-field responses of the umpires the HR at the time of the occurrence of the umpire's decision, 15s after the occurrence and 30s after the occurrence were analysed. To gain further insight in to the umpire's responses, differences between the HR at the time of the event and 15s post event ($D_1$), as well as the differences 15s and 30s post event ($D_2$) were investigated. It was felt that such differences could be assumed to be normally distributed. The responses of the umpires to select on-field events were divided into the following categories: catch - out; catch - not out; Leg before wicket (LBW)- out; LBW - not out; run-out; no-ball; and wide-ball. However, due to the fact that a number of these categories did not contain enough data it was decided to consider only the catch - out, catch - not out, LBW - out and LBW - not out.

As the HR was collected during the actual match situation it was not possible to control for or take into account factors such as the time of the day of the HR measurements, food or liquid ingested, tobacco or medication used before or during the match, and the posture or change in posture during the recording of the HR, which all have a significant effect on the HR response.

The umpires were required to complete a questionnaire on their perceived rating of how stressful they found making certain on-field decisions. These included the following decisions: LBW, catch close to and behind the wicket, run-out and deciding on a "wide-ball" and a "no-ball".

### Statistical Analysis

Single variable statistics were computed using the SAS Package with the results expressed as means and standard deviation (± SD). A Student's t test was used to determine differences between the provincial and international umpires, with statistical significance accepted for P values <0.05.

| TABLE I: Biographic data and umpiring experience | | | | | | |
|---|---|---|---|---|---|---|
| | All Umpires | | International | | Provincial | |
| | Mean | SD | Mean | SD | Mean | SD |
| Age (years) | 46.3 | 8.4 | 46.8 | 8.3 | 46.0 | 8.5 |
| **Umpiring Experience (years)** | | | | | | |
| Club | 12.1 | 4.5 | 12.8 | 6.1 | 11.7 | 3.7 |
| Provincial | 7.2 | 4.5 | 10.0 | 4.8 | 5.3 | 2.6 |
| International | 2.5 | 3.2 | 4.3 | 2.6 | - | - |
| **Limited-overs matches umpired(#)** | | | | | | |
| Provincial | 22.9 | 22.0 | 36.0 | 30.1 | 14.2 | 11.3 |
| International | 5.2 | 9.3 | 13.0 | 11.2 | - | - |
| **First-class matches umpired(#)** | | | | | | |
| Provincial | 25.3 | 17.3 | 35.0 | 15.8 | 20.5 | 16.3 |
| Test matches | 1.7 | 3.3 | 4.0 | 4.3 | - | - |

## RESULTS

The biographical data (Table I) shows a mean age of 46.3 ± 8.4 years for all the umpires, with similar average ages for those having umpired at international (46.8 ± 8.3 years) and provincial (46.0 ± 8.5 years) level. On average the umpires had experience umpiring at club level for 12.1 ± 4.5 years, at provincial level for 7.2 ± 4.5 years and at international level for 2.5 ± 3.2 years. At provincial level on average the umpires had umpired 22.9 ± 22.0 limited-overs and 25.3 ± 17.3 first class (3 or 4 days) matches, while at international level they had umpired 5.2 ± 9.3 limited-overs and 1.7 ± 3.3 Test (5 days) matches. All the international umpires, with the exception of one, had umpired at Test match level at the time of the study. This umpire has subsequently umpired in a number of Test matches.

The umpires generally rated the on-field decisions from "below average" to "stressful" with regard to

### TABLE II: Umpires ratings (#) of the perceived stressfulness of decisions made

|  | LBW | Catch | Run-out | No-ball | Wide |
|---|---|---|---|---|---|
| Not stressful | 1 | · | 1 | 2 | 3 |
| Below average | 3 | 4 | 3 | 5 | 4 |
| Average | 3 | 2 | 1 | 3 | 2 |
| Stressful | 3 | 3 | 4 | - | 1 |
| Very stressful | - | 1 | 1 | - | - |

the amount of stress they felt (Table II). The umpires who had umpired at international level tended to rate the on-field decisions as "below average" to "average", with the ratings of the umpires who had only umpired at provincial level showing a far greater variance in their ratings. They rated the LBW, catch, run-out and wide decisions being rated as "stressful". One of the provincial umpires rated the catch and run-out decision as "very stressful". All the umpires rated umpiring in first-class and Test matches as more stressful than the limited-overs matches as they had to maintain concentration for a longer period of time and, due to the field placing, there was a greater likelihood of them having to make decisions with regard to catches behind and close to the wicket.

The number of decisions that the umpires had to make in response to appeals are shown in Table III. From this it is evident that the "LBW - not out" decisions was the most common decision made by the umpires. In the international matches, the third umpire made the decisions with regard to run-out and stumping appeals with the use of slow motion TV replays, while during provincial matches the on-field umpire had to make these decisions.

The results showed a mean HR rate for the umpires before and during the match (Table IV) which corresponds to approximately 60% of the umpires' estimated maximal HR. The HR rate of the

### TABLE III: Number of decisions made by the umpires

|  | All Umpires | International | Provincial |
|---|---|---|---|
| **Catch** |  |  |  |
| Out* | 31 | 14 | 17 |
| Not Out | 20 | 11 | 9 |
| **LBW** |  |  |  |
| Out | 8 | - | 8 |
| Not Out | 47 | 21 | 26 |
| **Run-out** |  |  |  |
| Out | 5 | 1 | 4 |
| Not Out | 5 | 1 | 4 |
| **Stumping** |  |  |  |
| Out | 2 | - | 2 |
| Not Out | 1 | - | 1 |
| **Wide-ball** | 23 | 12 | 11 |
| **No-ball** | 22 | 4 | 18 |
| **TOTAL** | 164 | 64 | 100 |

*\* These were all the dismissals where the batsman was out caught. In some cases the umpire was not called on to give a decision as the batsman "walked", other decisions were fairly straight forward decisions, while in some cases it was difficult to determine whether the umpire was called on to give a decision or not.*

### TABLE IV: Mean heart rates (beats min$^{-1}$) during the first and second innings of the match

|  | All Umpires | | | International | | | Provincial | | |
|---|---|---|---|---|---|---|---|---|---|
|  | Mean | SD | Diff | Mean | SD | Diff | Mean | SD | Diff |
| **15 min to start:** |  |  |  |  |  |  |  |  |  |
| 1st Innings | 99 | 11.8 | - | 102 | 13.9 | - | 98 | 11.1 | - |
| 2nd Innings | 94 | 9.6 | - | 95 | 10.7 | - | 93 | 9.6 | - |
| **Start to 1st break (Session 1):** |  |  |  |  |  |  |  |  |  |
| 1st Innings | 102 | 11.1 | 2.4 | 101 | 12.0 | -0.7 | 102 | 11.6 | 4.3 |
| 2nd Innings | 101 | 10.0 | 7.4 | 103 | 9.9 | 7.5** | 100 | 10.8 | 7.3 |
| **1st to 2nd break (Session 2):** |  |  |  |  |  |  |  |  |  |
| 1st Innings | 100 | 14.2 | -1.4 | 102 | 13.7 | 0.3 | 99 | 15.8 | -2.5 |
| 2nd Innings | 100 | 8.1 | -1.6 | 97 | 6.7 | -5.8 | 101 | 9.2 | 1.2 |
| **2nd break to end (Session 3):** |  |  |  |  |  |  |  |  |  |
| 1st Innings | 99 | 12.3 | -1.3 | 101 | 12.8 | -0.8 | 98 | 13.1 | -1.7 |
| 2nd Innings | 96 | 8.7 | -2.7 | 97 | 5.2 | 0.0 | 95* | 11.4 | -6.0 |
| **End plus 5 min:** |  |  |  |  |  |  |  |  |  |
| 1st Innings | 100 | 12.5 | 0.7 | 98 | 13.5 | -3.0 | 101 | 13.0 | 3.2 |
| 2nd Innings | 99 | 7.1 | 3.0 | 100 | 5.5 | 2.5 | 99* | 8.9 | 4.0 |
| **Mean for innings:** |  |  |  |  |  |  |  |  |  |
| 1st Innings | 100 | 12.4 | 0.3 | 102 | 12.5 | 2.5 | 100 | 13.5 | -1.1 |
| 2nd Innings | 100 | 8.6 | 0.6 | 100 | 8.9 | 0.0 | 100 | 10.1 | 1.0 |

*\* Data from one umpire was excluded because the HR monitor malfunctioned during this part of the match*

*Diff The difference between the mean HR for a period and the preceding period, with a - indicating that the second session HR was the greater of the two*

*\*\* Significant difference (p<0.05)*

umpires did not differ significantly during various periods of the match. The slight, non-significant decreases in heart rates during the second innings may be as a result in a decrease in environmental temperature during the evening.[11] However, significant differences ($p < 0.05$) were found for the difference between the HR of the international umpires during the 15 minutes before the start of the match and the first session of the first innings and the corresponding time during the second innings, as well as when the differences between the HR of the second and third sessions of the second innings, were compared. Although these differences are significant this could be as a result of differences in environmental conditions.

The HR response of the umpires at the time of the event, 15s post event and 30s post event are shown in Figure 1 and Figure 2. The HR of the umpires varied from as low as 68 beats $min^{-1}$ at the time of the event to 139 beats $min^{-1}$ at 15s post event. The differences in the heart rate response between the time of the event and 15s post event ($D_1$) and 15s and 30s post-event ($D_2$) were both positive and negative (Fig 3 and Fig 4). In order to obtain more information from the data the positive and negative differences were separated for $D_1$ and $D_2$. However, the small sample size did not reflect any statistical significant differences, but for the "LBW -not out" decision it is clear that the provincial umpires manifested greater negative differences for both $D_1$ and $D_2$.

## DISCUSSION

The principal finding of this study was that the heart rates of the umpires was high before and during the match, with variations in the HR of the umpires occurring as a result of on-field decisions. Cricket umpires do not engage in light physical warm-up activities before going onto the field and the elevated HR could be attributed to the emotional state of the umpires prior to the commencement of the match. This anticipatory increase in HR was evident in all the umpires before the game and concurs with the findings of the study on soccer referees.[9] The elevated HR during the match was similar, although with lower recorded mean HR than those found in Australian football field umpires,[8] association football referees[9] and netball umpires.[1] In these sports the bodies physiological responses to the task of umpiring and refereeing would be greater due to the nature of the task which is more physically demanding than that of cricket umpiring.

The second important finding is the large variations in the umpires' HR at and after the event (Fig 1 and Fig 2) with the HR varying from as low as 68 beats $min^{-1}$ to 139 beats $min^{-1}$. These variations in HR response could be as a result of the psychological or physiological response to the appeal. At the time of the event, an appeal for a catch, the measured HR varied from of 68 beats $min^{-1}$ (Umpire 2) to 127 beats $min^{-1}$ (Umpire 9) - a difference of 59 beats $min^{-1}$. The greatest change in HR occurred when Umpire 9 showed an increase in HR from 121 beats $min^{-1}$ at the time of the event to 139 beats $min^{-1}$ 15s after an appeal for a catch that he gave "not-out". This
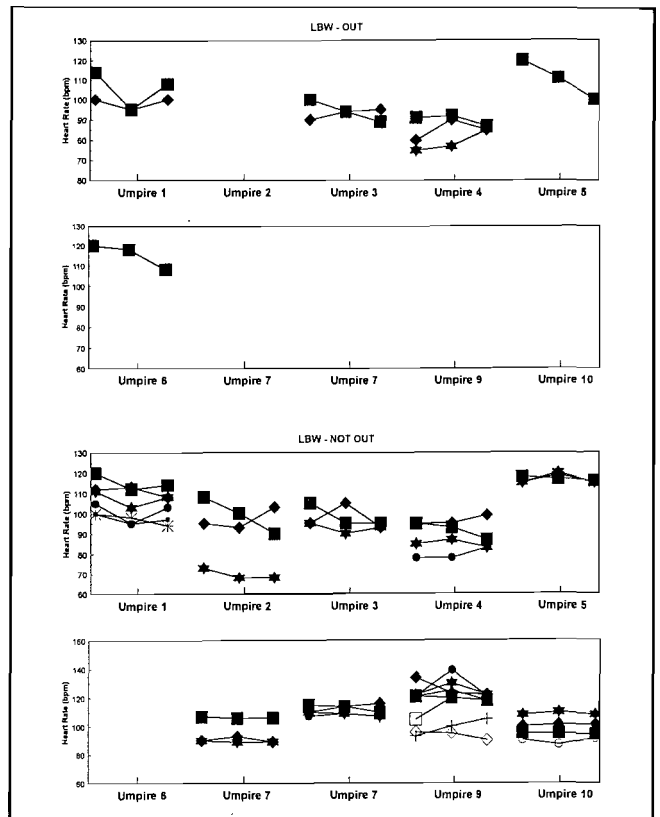


Figure 1: The HR response of the umpires to "LBW - out" and "LBW - not out" decisions at the time of the event, 15s post event and 30s post event.

umpire, an international umpire, who admitted to going through a "relatively bad patch" in his umpiring, also showed the greatest variation in heart rate from 15s to 30s after an appeal. The opposition players obviously attempted to exploit this by pressurising him with a large number of LBW appeals as recorded.

Figure 1 and Figure 2 also show that the provincial umpires (Umpires 1 to 6) generally showed greater differences in HR response than the international umpires (Umpires 7 to 10), with the exception of Umpire 9 who by his own admission had a very poor match. This could be an indication that the international umpires, although umpiring at a higher level with more spectators in attendance, greater media coverage, particularly television coverage, and more at stake for the players, through a combination of their psychological characteristics and years of conditioning as an umpire, are better able to control their emotions on the field of play and not feel pressurised by the players and the importance of the occasion.

The variation in HR response was further evident in the increased HR occurring in the case of one of the umpires (Umpire 4) where the differences ($D_1$) between the event and 15s post-event (Fig 3) were smaller than the differences ($D_2$) between 15s and 30s post-event (Fig4). However, in all cases the change in the HR of the umpires after an appeal could not be attributed solely to anxiety. The umpire would often have to make a decision and then move into position in order to prepare for the next decision, such as an appeal for LBW followed by a attempted

run-out. This physical activity, as well as environmental factors have had an effect on the heart rate.

During one of the internationals Waquar Younis, the Pakistani fast bowler, took a hat-trick (three wickets in consecutive deliveries). Although the umpire was not required to make a decision in any of these dismissals as all three batsmen were bowled, an increase in the heart rate of the umpire occurred at the time of each dismissal (Table V). This could again illustrate the psychological component of HR being so closely involved in an event that occurred very seldom in international cricket.

It is recommended that further studies should focus on the HR response of cricket umpires during the first-class and Test matches which last for between three and five days and that, although it may
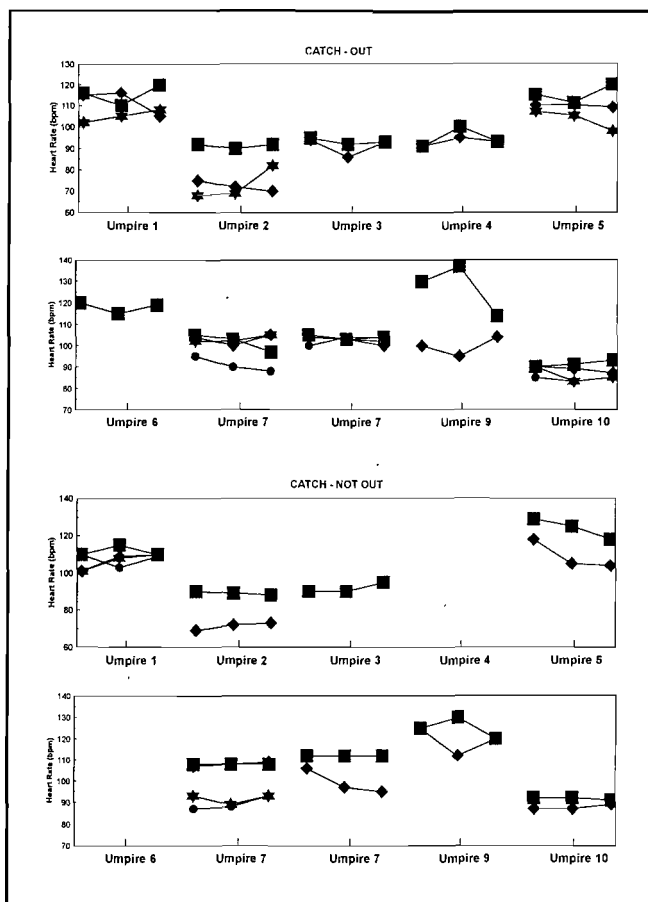


Figure 2: The HR response of the umpires to "Catch -out" and "Catch - not out" decisions at the time of the event, 15s post event and 30s post event.

TABLE V: Heart rate (beats min⁻¹) of umpire during Waquar Younis hat-trick during an International match.

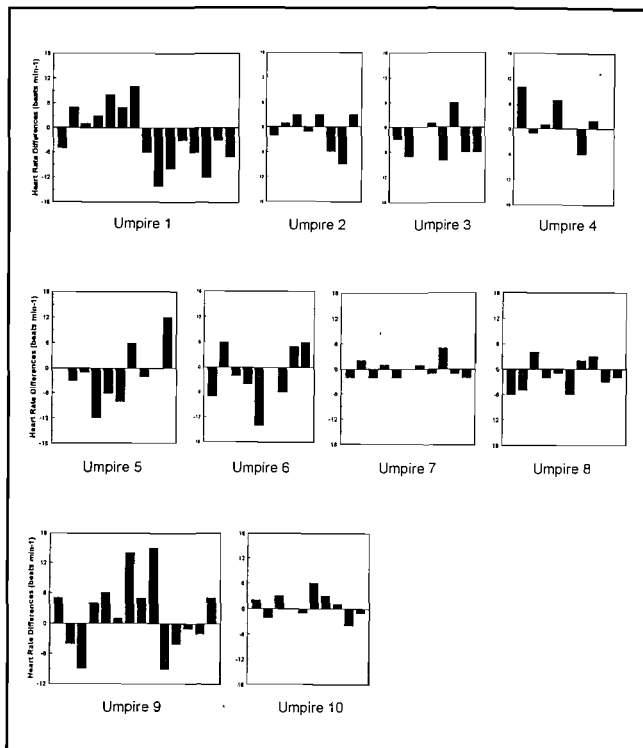| | Dismissal (beats min⁻¹) | 15s post Dismissal (beats min⁻¹) | 30s post Dismissal (beats min⁻¹) |
|---|---|---|---|
| 1st Dismissal | 89 | 92 | 91 |
| 2nd Dismissal | 93 | 88 | 90 |
| 3rd Dismissal | 98 | 103 | 106 |



Figure 3: Differences ($D_1$) between the HR (bpm) at the time of the event and 15s post-event for the umpires



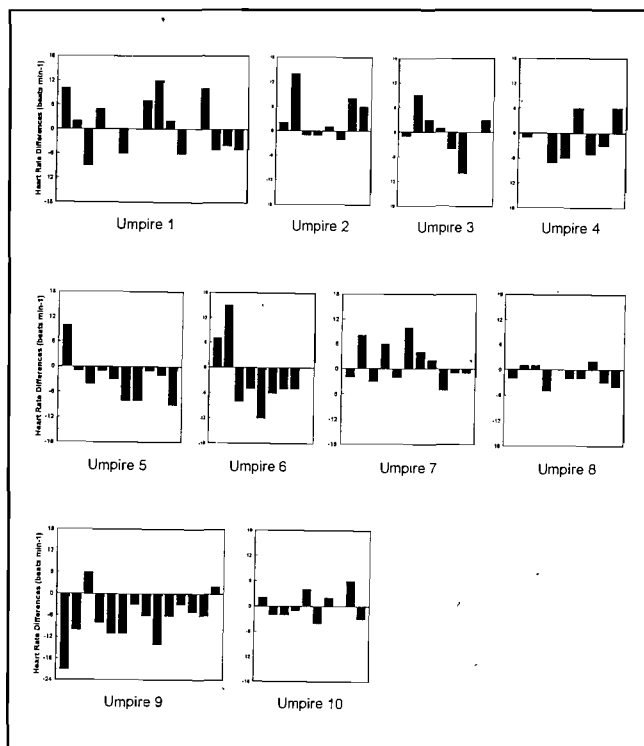Figure 4: Differences ($D_2$) between the HR (bpm) from 15s to 30s post-event for the umpires

be extremely difficult in the match situation, attempts should be made to control for other factors such as the ingestion of food, postural changes, temperature, etc.

In conclusion, it seems that the major finding of this study is that the HR response of the umpires to on-field events indicate that they are under psychological pressure. Stressful perceptions were related to the development of dissatisfaction, burn-out and withdrawal in soccer referees[12,13] and before a similar situation arises in cricket training clinics, which include stress management, should be introduced for cricket umpires at all levels. Good umpires, like good cricketers, are both born and made through their experiences and some umpires are better able to handle the situation due to their psychological characteristics which may be a product of years of conditioning. Accurate and consistent decision-making, though desirable from officials at all times, is in reality difficult to achieve. However, together with regular practice and competition and the correct mental and physical preparation, errors and variability in the performance of the umpires should be reduced.

REFERENCES

1. Otago L, Riley T, Forrest J. Movement patterns and energy requirements of netball umpires. Sports Coach 1994;17(2):10-14.
2. Steele J, Chad K. An analysis of the movement patterns of netball players during match implications for designing training programs. Sports Coach 1992;15(1):21-29.
3. Reilly T, Thomas V. A motion analysis of work-rate in different positional roles in professional football match-play. J Hum Mov Stud 1976;2:87-97.
4. Morgans LF, Scovil JA, Bass KM. Heart rate responses during singles and doubles competition in racquetball. Phys Sportsmed 1984;12(11):64-72.
5. Blanskby BA, Elliott BC, Bloomfield J. Telemetered heart rate responses of middle-aged sedentary males, middle-aged males and "A" grade male squash players. Med J Aust 1973.2:477-481.
6. Montgomery DL, Malcolm V, McDonnell E. A comparison of the intensity of play in squash and running. Phys Sportsmed 1981;9(4):116-119.
7. Kozar AJ, Hunsicker P. A study of telemetered heart rate during sports participation of young adult men. J.Sports Med 1993;3(1):1-5.
8. Pyne D, Ackermann K. Physiological relationships between the training and match performance of Australian field umpires, Sports Coach 1987. 10:2;20-24.
9. Catterall C, Reilly T, Atkinson G, Coldwells. An Analysis of the work rates and heart rates of association football referees. Br J Sports Med 1993;27(3):193-196.
10. Longmore BE. Psychological pressures on cricket umpires. N Z J Sports Med 1992;20(2):5-8.
11. McArdle W, Katch FI, Katch VL. Exercise Physiology - Energy, nutrition and human performance (3rd Edition), Lea & Febiger, London, England, 1991.
12. Taylor A, Daniel J, Leith L, Agnew J. An investigation of the psychological factors preceding the dropout of Ontario soccer officials. University of Toronto School of Physical and Health Education 1988 (ex Evans 1994).
13 Evans R. A psychological profile of top Australian soccer referees. Sports Coach 1994;17(2):17-18.

in ambulatory patients with long-term heart failure. Circulation 1990a; 81:518-27.
134. Sullivan MJ, Higginbotham MB, Cobb FR. Increased exercise ventilation in patients with chronic heart failure: intact ventilatory control despite hemodynamic and pulmonary abnormalities. Circulation 1988b;77:552-559.
135. Sullivan MJ, Knight JD, Higginbotham MB. Cobb FR. Relation between central and peripheral haemodynamics during exercise in patients with chronic heart failure. Muscle blood flow is reduced with maintenance of arterial perfusion pressure. Circulation 1989;80:769-81.
136. Sullivan MJ, Merola AJ, Timmerman AP. Unverferth DV, Leier CV. Drug-induced aerobic enzyme activity of human skeletal muscle during bedrest deconditioning. J Cardiopulm Rehab 1986;6:232-237.
137. Swan HJ. Significance and prognostic value of left ventricular function in ischaemic heart disease and in cardiomyopathy. Adv Cardiol 1986;34:45-57.
138. Szlachcic J, Massie BM. Kramer BL, Topic N, Tubau J. Correlates and prognostic implication of exercise capacity in chronic congestive heart failure. Am J Cardiol 1985:55:1037-42.
139. Thompson JM, Dempsey JA, Chosy LW, Shahidi NT. Reddan WG. Oxygen transport and oxyhaemoglobin dissociation during prolonged muscular work. J Appl Physiol 1974; 37:658-64.
140. Thurlbeck W. Diaphragm and body weight in emphysema. Thorax 1978;33:483-48.
141. Tikunov B, Levine S. Mancini D. Chronic congestive heart failure elicits adaptations of endurance exercise in diaphragmatic muscle. Circulation 1997: 95 (4): 910-916.
142. Toussaint D, Dustin P Electron microscopy of normal and diabetic retinal capillaries. Arch Opthalm 1963;70:96.
143. Vracko R. Skeletal muscle capillaries in diabetics: A quantitative analysis. Circulation 1970:41:271.
144. Wada O, Hidetsugu A, Miyagi K, Ishizaka S. Kameyama T. Seto H, Sasayama S. Importance of abnormal lung perfusion in excessive exercise ventilation in chronic heart failure. Am Heart J 1992:125:790-798.
145. Wade OL, Bishop JM. Cardiac output and regional blood flow. Blackwell, Oxford. 51-72 1962.
146. Weber KT, Janicki JS. Cardiopulmonary exercise testing for evaluation of chronic heart failure. Am J Cardiol 1985a:55:22A-31A.
147. Weber KT, Janicki JS. Lactate production during maximal and submaximal exercise in patients with chronic heart failure. J Am Coll Cardiol 1985b;6:717-724.
148. Weber KT, Kinasewitz GT, Janicki JS, Fishman AP. Oxygen utilization and ventilation during exercise in patients with chronic cardiac failure. Circulation 1982;65:1213-1223.
149. Weiss S. Ellis LB. Oxygen utilization and lactic acid production in the extremities during rest and exercise. Arch Intern Med 1935:55:665-680.
150. Wiener DH, Fink LI, Maris J, Jones RA, Chance B, Wilson JR. Abnormal skeletal muscle bioenergetics during exercise in patients with heart failure: Role of reduced muscle blood flow. Circulation 1986:73:1127-1136.
151. Wilson JR, Fink L, Maris J, Ferraro N, Power-Vanwart J, Eleff S, Chance B. Evaluation of skeletal muscle energy metabolism in patients with heart failure with gated phosphorous-31 nuclear magnetic resonance. Circulation 1985;71:57-62.
152. Wilson JR, Frey MJ, Mancini DM, et al. Sympathetic vasoconstriction during exercise in ambulatory patients with left ventricular failure. Circulation 1989;79:1021-1027.
153. Wilson JR, Mancini DM, Simson M. Detection of skeletal muscle fatigue in patients with heart failure using electromyography. Am J Cardiol 1992;70:488-493.
154. Wilson JR. Martin JL. Ferraro N, Weber KT. Effect of hydralazine on perfusion and metabolism in the leg during upright bicycle exercise in patients with heart failure. Circulation 1983;69:425-32.
155. Wilson JR. Martin JL. Ferraro N. Impaired skeletal muscle nutritive flow during exercise in patients with congestive heart failure: role of cardiac pump dysfunction as determined by the effect of dobutamine. Am J Cardiol 1984a;53:1308-1315.
156. Wilson JR. Martin JL. Schwartz MD, Ferraro N. Exercise intolerance in patients with chronic heart failure: role of impaired nutritive flow to skeletal muscle. Circulation 1984b;69:1079-87.
157. Wroblewski H. Kastrup J, Norgaard T. Mortensen S. Haunso S. Evidence of increased microvascular resistance and arteriolar hyalinosis in skin in congestive heart failure secondary to idiopathic dilated cardiomyopathy. Am J Cardiol 1992;69:769-774.
158. Yamabe H, Itoh K. Yasaka Y, Kakimoto T. Yokoyama M. The distribution of the blood flow during exercise in chronic heart failure - compensatory mechanism to the decreased cardiac output. Japanese Circulation J - Eng Ed. 1992;56:494-499.
159. Yancy CW, Parsons D, Lane L. Carry M, Firth B. Blomquist G. Capillary density. fiber type and enzyme composition of skeletal muscle in congestive heart failure. J Am Coll Cardiol 1989;13:38A.
160. Zelis R, Flaim SF. Alterations in vasomotor tone and congestive heart failure. Prog Cardiovasc Dis 1982:24:437-459.
161. Zelis R, Longhurst J. Capone RJ, Mason DT. A comparison of regional blood flow and oxygen utilization during dynamic forearm exercise in normal subjects and patients with congestive heart failure. Circulation 1974; 50:137-143.
162. Zelis R, Mason DT, Braunwald E. A comparison of the effects of vasodilator stimuli on peripheral resistance vessels in normal subjects and in patients with congestive heart failure. J Clin Invest 1968;47:960-970.
163. Zelis R, Nellis SH, Longhurst J. Lee G. Mason DT. Abnormalities in the regional circulations accompanying congestive heart failure. Prog Cardiovasc Dis 1975;18:181-209.
164. Zelis R. Sinoway L. Musch T. Why do patients with congestive heart failure stop exercising? Ed comment. J Am Coll Cardiol 1988;12:359-361.

# Anterior cruciate ligament surgery - A survey of surgical technique and rehabilitation practice in Southern Africa

A St Clair Gibson MBChB PhD
MI Lambert PhD
J Milligan BSc.Physio BSc.Med(Hons)(Exercise Science)
W Van der Merwe * MBChB BSc.Med(Hons)(Exercise Science)
J Walters * MBChB FCS
TD Noakes MBChB MD FACSM

MRC/UCT Bioenergetics of Exercise Research Unit, Sport Science Institute of South Africa, University of Cape Town Medical School
* Department of Orthopaedic Surgery, University of Cape Town Medical School

## ABSTRACT

**Objectives :** To assess the choice of surgical repair procedures and post-operative management of the ruptured Anterior Cruciate Ligament (ACL) in the South African Orthopaedic community.

**Methods :** A census questionnaire study was conducted on all 475 Orthopaedic surgeons registered with the Orthopaedic Association of South Africa during 1993/1994. The Chi-square test was used to determine statistical significance between different groups of respondents.

**Results :** Seventy seven percent of the questionnaires were returned. Of the respondents, 77% performed ACL reconstructions. The surgeons who described themselves as "general" Orthopaedic surgeons were more likely to perform less than 1 operation per month (p<0.001), to use arthrotomy incisions (p<0.001), to be in hospital practice (p<0.001) and not to use accelerated rehabilitation protocols (p<0.001). The majority of registrar respondents (85%) use arthrotomy procedures for ACL repairs.

**Conclusions :** Procedures used by Orthopaedic surgeons who perform ACL reconstructions infrequently (one or less than one operation per month), particularly those of arthroscopic procedures and post-operative rehabilitation, need to be reviewed. A cause for concern is that the registrar population is using techniques of ACL repair which have been superceded by more modern methods, which are used more frequently in the private sector. This concern can perhaps be resolved by involving the private Orthopaedic surgeons in Orthopaedic registrar training where deficiencies exist.

## INTRODUCTION

Since 1919, when Hey Groves described a reconstruction procedure for the ruptured Anterior Cruciate Ligament (ACL) using tensor fascia lata as a replacement material,[1] there has been continued interest in ACL repair and rehabilitation.[2,3] In 1963, Jones used part of the patellar tendon as a replacement graft.[4] A modified version of his method, popularised by Clancy - the bone-patellar tendon-bone graft - has become the replacement material of choice.[5,6] However, post-surgical problems with the knee extensor mechanism have prompted continued interest in the use of the semitendinosis, gracilis, iliotibial band or vastus lateralis tendons as replacement grafts.[7]

Research in the last decade has also focused on the use of artificial grafts,[8] the efficacy of primary repair of the damaged ACL,[9] and non-surgical management.[10] All these techniques have shown to be less successful than the bone-patellar tendon-bone graft described above.[8,9,10] The advent of arthroscopic procedures reduced intra-operative damage to the knee joint capsule, removed the need for long term casting, and allowed a shorter period of rehabilitation with reduced post-operative knee extensor mechanism weakness.[2,11] Although the greater portion of the literature shows that arthroscopic procedures produce fewer complications than the arthrotomy type ACL procedures,[11,12,13,14,15] studies have reported complications, including tension pneumarthrosis and fistula formation, which may be directly caused by the arthroscopy procedure itself.[16,17]

Rehabilitation of patients after surgical repair of the ACL ligament has also evolved, with studies showing the potentially deleterious effects of immobilisation, both on the graft itself and surrounding musculature.[13] Later studies showed that early mobilisation protocols decreased the side effects of immobilisation,[15] and Shelbourne and Nitz's landmark study on accelerated rehabilitation,[18] though still provoking some controversy,[19] heralded the way for almost immediate mobilisation and full weight bearing after an ACL reconstruction. Their protocol, with some amendments, has become accepted practice for post-surgical rehabilitation of patients undergoing ACL surgery in most developed countries.[12,20]

In 1981, Paulos et al[14] questioned fifty American Orthopaedic surgeons, and discovered that these surgeons were rehabilitating their patients to full activity faster than was considered appropriate at that time. The surgical and rehabilitation practices used by South African Orthopaedic surgeons in repairing the ruptured ACL have not been quantified. Therefore, the aim of this study was to determine, through a questionnaire study, the current ACL reconstruction technique and post-operative rehabilitation protocols used by the majority of Orthopaedic surgeons in South Africa.

**CORRESPONDENCE:**

Dr A St Clair Gibson
MRC/UCT Bioenergetics of Exercise Research Unit
Sport Science Institute of South Africa
Boundary Road
Newlands, 7700
RSA
Tel    - 27 - 21 - 6867330
Fax    - 27 - 21 - 6867530
email - agibson@sports.uct.ac.za

## METHODS

A census-type questionnaire study was undertaken of the Orthopaedic community of Southern Africa during 1993/1994. A draft copy of the questionnaire was sent to an Orthopaedic surgeon, physiotherapist and statistician for their comments. In addition, a small sample (n=8) of hospital Orthopaedic surgeons completed the questionnaire in a pilot study. Problematical questions were identified and adjusted. The corrected questionnaire was translated into Afrikaans and checked by an Afrikaans-speaking Orthopaedic surgeon.

A questionnaire was sent to each Orthopaedic surgeon listed with the South African Orthopaedic Association. A second questionnaire was sent to those Orthopaedic surgeons who did not respond to the first mailing, and a third questionnaire to those that did not respond to either the first or second mailings. This was followed by a short questionnaire which was sent to those that did not respond to any of the three earlier questionnaires, seeking the reasons for their lack of response.

The questionnaire sought answers to the following questions: i) personal data, including the type of practice and sub-specialty interest of the Orthopaedic surgeon, ii) operative details and surgical techniques used by the Orthopaedic surgeon for ACL reconstructions, including the number of operations performed per month, choice of graft, and whether augmentation procedures were used, iii) protocols used for post-operative rehabilitation, including time of post-operative limb immobilisation, whether continuous passive motion and accelerated rehabilitation regimens were prescribed, and when the patients were referred post-operatively to physiotherapists or biokineticists, and iv) the Orthopaedic surgeons perceptions of the use of physiotherapists in the ACL rehabilitation process.

The Chi-square test was used to determine statistical significance between different groups of respondents. Statistical significance was accepted when $p<0.05$.

## RESULTS

### Respondent Characteristics

Four hundred and seventy five questionnaires were distributed using the methods described. Three hundred and sixty eight Orthopaedic surgeons responded to either of the three questionnaires, or the non-response questionnaire, giving a 77% return. Sixty questionnaires (13%) were excluded from analysis due to missing or incorrect data. Of the respondents who answered the questionnaire correctly, 176 answered the first sendout, 38 the second, 42 the third, and 52 the non-return questionnaire. A large portion of non-responders (44%) felt the questionnaire was not relevant to their field of interest. Other reasons given by the non-responders were that they were retired (17%), personal reasons precluded response (8%), or that incorrect grammar (2%) or incorrect use of language (2%) in the questionnaire prevented their response. Fifteen percent of the non-responders indicated that they did not receive any of the three questionnaire mailings.

Of the 256 respondents whose answers were used in the analysis, 40% were in private practice only, 20% in hospital practice only, and 40% in both private and

hospital practices. The highest proportion of the respondents practiced in Southern Gauteng (Transvaal) (31%), followed by the Northern Gauteng (Northern Transvaal) (21%), Western Cape (20%), KwaZulu-Natal (14%), Eastern Cape (7%) and Orange Free State (7%). The largest proportion of the respondents classified themselves as "general" Orthopaedic surgeons (41%). The most common sub-specialties in which the other Orthopaedic surgeons concentrated were the knee (14%), spine (8%), hip (8%) and shoulder (7%). Of the respondents, 14% were registrar members of the Orthopaedic Association of South Africa.

### Surgical Procedures

Seventy seven percent of the 256 respondents performed ACL repairs. Of these, 61% performed one, or less than one, operation per month, 34% performed two to five operations per month, and 5% more than five operations per month. Table I shows the surgical preferences for ACL repair of all respondents. Seventy three percent of respondents used either the Clancy or modified transpatellar arthrotomy incisions, compared to 27% using arthoscopic procedures. The large majority of surgeons used autografts (88%) to reconstruct the ruptured ACL, with the bone-patellar tendon-bone graft being the most popular replacement material used (79%). Primary repair of the ACL was performed by 31% of surgeons. Ninety two per-

---

**TABLE I: Number of Orthopaedic surgeons (n=256) reporting surgical techniques for ACL repair**

| | Percentage |
|---|---|
| **Incision** | |
| Athrotomy - Standard Clancy | 37 |
| Athrotomy - Modified Transpatellar | 36 |
| Athroscopy | 27 |
| | |
| **Primary Repair** | |
| Yes | 31 |
| No | 69 |
| | |
| **Primary Repair Augmentation** | |
| Yes | 12 |
| No | 19 |
| Do Not Perform Primary Repairs | 69 |
| | |
| **Reconstruction Graft Used** | |
| Autograft | 88 |
| Allograft | 1 |
| Artificial Graft | 0 |
| Perform Primary Repair Only | 11 |
| | |
| **Reconstruction Material Used** | |
| Bone-patellar Tendon-Bone Graft | 79 |
| Semitendinosus Tendon | 5 |
| Gracilis Tendon | 1 |
| Iliotibial Band | 3 |
| Other | 1 |
| Perform Primary Repair Only | 11 |
| | |
| **Extra-Articular Graft Augmentation** | |
| Yes | 39 |
| No | 61 |

---

cent of surgeons would manage a ruptured ACL non-surgically in certain situations. The main reasons given for this option were that the age of the patient (70%) or the patient's level of activity (77%) precluded surgery. Twenty percent of these surgeons felt that non-surgical management was the best option routinely, and 6% felt that the patient's gender was important in deciding whether to manage an ACL injury non-surgically.

The respondents who performed one or less than one ACL repair per month performed a significantly higher proportion of arthrotomies than those performing 2 or more ACL repairs per month ($p<0.001$) (Figure 1). Respondents in hospital practice performed a significantly higher proportion of arthrotomies than those in private practice ($p<0.001$ (Figure 2). Similarly, a signifiantly higher proportion of repondents who performed one, or less than one operation per month were in hospital practice compared to those in private practice ($p<0.001$).

A highly significant proportion of "knee specialists" use arthroscopic procedures for ACL repair ($p<0.001$) (Figure 3). Similarly, "knee specialists" were more often in private practice, and a significant proportion performed two or more ACL procedures a month compared to "general" Orthopaedic surgeons ($p<0.001$). Conversely, a significantly higher proportion of "general" Orthopaedic surgeons performed one or less than one ACL repair per month, were in hospital practice and used arthrotomy type procedures for ACL repairs ($p<0.001$). A significant proportion of Orthopaedic surgeons who performed Clancy arthrotomy incisions also performed the insubstance primary repair procedure ($p<0.001$).

A higher proportion of registrar respondents performed less than one operation per month ($p<0.05$) when compared to qualified Orthopaedic surgeons in private and hospital practice. Eighty five percent of these registrars used arthrotomy procedures for ACL repairs.
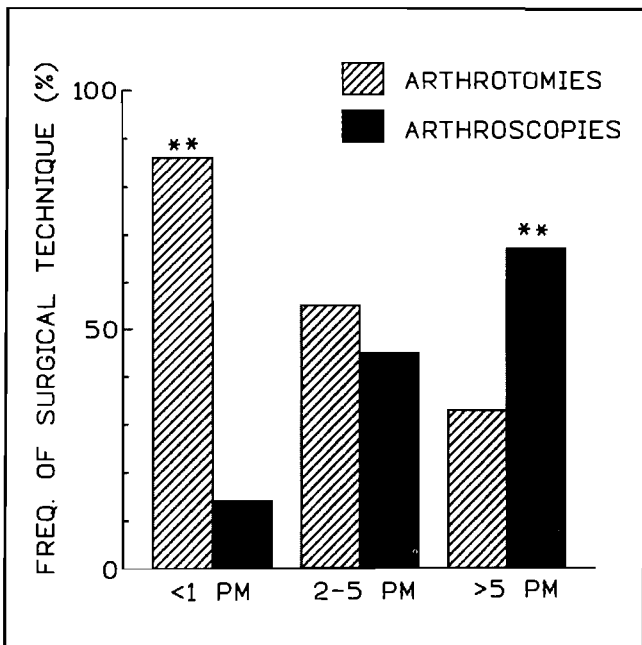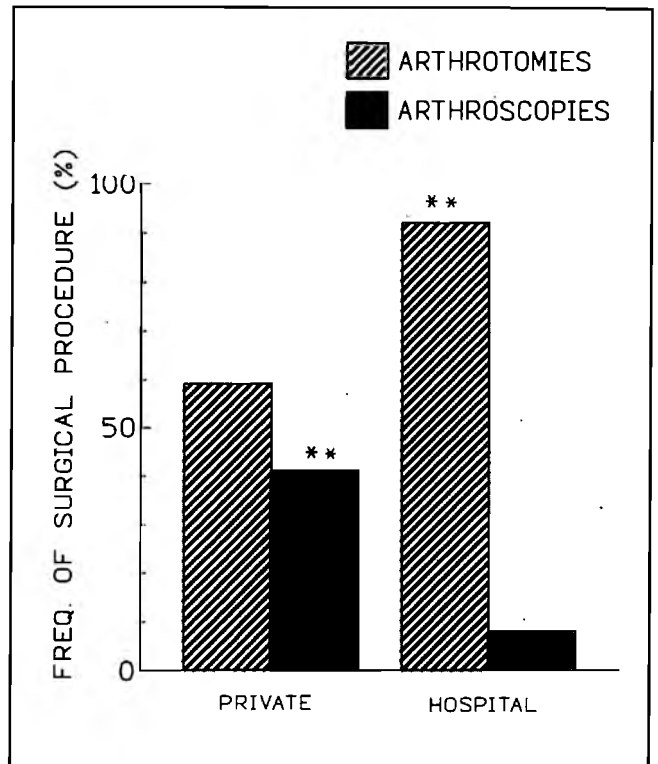


Figure 2. *The relationship between the type of surgical procedures used for ACL repair (Arthroscopy vs Arthrotomy) and the type of practice in which the Orthopaedic Surgeon is involved (hospital vs private).(** -p<0.001)*



Figure 1. *The relationship between the type of surgical procedures used for ACL repair (Arthroscopy vs Arthrotomy) and the number of ACL operations performed by Orthopaedic surgeons per month (PM).(** -p<0.001)*
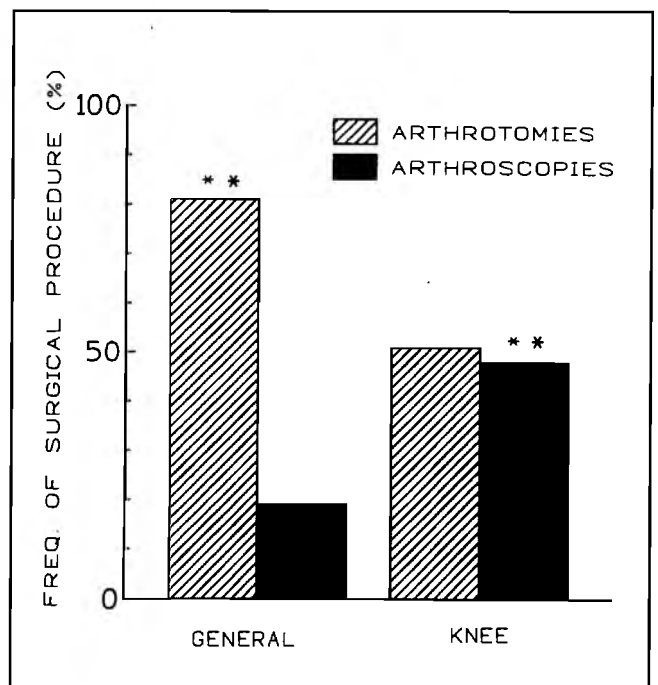


Figure 3. *The relationship between the type of surgical procedure used ACL repair (Arthroscopy vs Arthrotomy) and whether the Orthopaedic Surgeons were "general" Orthopaedic Surgeons or "knee specialists" (see text for definitions).(** -p<0.001)*

## Post-operative ACL Rehabilitation

The majority of respondents (65%) referred their patients to a physiotherapist or biokineticist in the first post-operative week. Sixty five percent of all respondents believed that the lower limb should be immobilised after an ACL repair (Table II). Of these, 29% believe that the limb should be immobilised for 6 weeks or longer.

TABLE II: Number of surgeons who immobilise the involved limb after ACL repair and the duration of immobilisation compared to surgeons who mobilise the limb immediately after the repair (n=256)

| Immobilise 65% | Percent |
|---|---|
| Duration: 1 week | 10 |
| 2 weeks | 10 |
| 3 weeks | 5 |
| 4 weeks | 10 |
| 5 weeks | 1 |
| 6 weeks | 27 |
| 6 weeks-3 months | 2 |
| >3 months | 0 |
| Mobilise 35% | |

Forty nine percent of the respondents allowed their patients to use an accelerated rehabilitation protocol, although only 23% of them allowed full weightbearing on the injured limb in the first week post-operatively. Of the 49% of respondents who used the accelerated rehabilitation protocols, 35% immobilised the limb for 6 weeks or longer. Of the same surgeons who use accelerated rehabilitation, 32% allowed weightbearing on the damaged limb 4 weeks or longer post-operatively.

Approximately half of the respondents (51%) used continuous passive motion post-operatively. The large majority of respondents thought that muscular atrophy (83%) and decreased range of motion (66%) were the most common post-operative complications caused by immobilization.

The surgeons who performed more than five ACL repairs per month used accelerated rehabilitation more frequently than those who performed one or less than one operation per month (p<0.05). Similarly, respondents who performed arthroscopic ACL procedures used accelerated rehabilitation protocols with greater frequency compared to those who performed the Clancy arthrotomy procedure (p<0.05).

Finally, although 96% of surgeons used physiotherapists or biokineticists for post-operative ACL rehabilitation, 13% of all respondents, including the 4% who did not use physiotherapists, did not believe that physiotherapy treatment speeds up recovery. In addition, 45% of all surgeons were not sure, or did not believe, there was adequate communication between Orthopaedic surgeons and physiotherapists during the period of rehabilitation.

## DISCUSSION

The first important finding of this study was that a high proportion of South African hospital Orthopaedic Surgeons use Clancy or modified transpatellar arthrotomy procedures rather than arthroscopic procedures.

This is surprising given that arthroscopic procedures have been shown to have fewer complications than arthrotomy ACL procedures.[2,11,12,15,20] This can perhaps be explained either by the financial constraints in the hospital sector, or because these operative techniques are the first choice procedures of the respondents for reasons not elucidated by the study.

Whichever of these two is the cause, it is clear that the large majority of registrar respondents who replied to the questionnaire are using arthrotomy procedures rather than arthroscopic techniques. It has been suggested by Mackenzie[22] that, as in the New Zealand system, registrars spend six months rotation in accredited private Orthopaedic practices as part of their Orthopaedic training. In this period they would learn more sophisticated surgical techniques such as arthroscopic procedures. Given the findings of this study, his idea would appear to have merit.

When the questionnaires were analyzed according to different sub-groups, some tendencies developed. Firstly, those surgeons who perform one, or less than one, ACL operation per month performed the arthrotomy type procedure with significantly higher frequency than those surgeons who performed the operation more frequently and who were more likely to perform the surgery arthroscopically (Figure 1). Typically, surgeons performing arthrotomies worked in a hospital practice (Figure 2), and defined themselves as "general" Orthopaedic surgeons (Figure 3). They also used accelerated rehabilitation less frequently than did surgeons that performed two or more ACL operations a month.

In contrast, those Orthopaedic surgeons who performed two or more ACL operations per month, and particularly those who performed five or more procedures a month, used arthroscopic procedures significantly more often, and were more often in private practice.

This group also defined themselves as "knee specialists", and prescribed accelerated rehabilitation protocols with greater frequency. This perhaps may indicate that specialization in a certain sub-specialty of Orthopaedic surgery, which allows more frequent use of specific operations by an Orthopaedic surgeon, may be beneficial in maintaining surgical progress.

The majority of Orthopaedic surgeons in this survey still believe that it is beneficial to immobilise the limb for a period post-operatively. A portion (29%) of the respondents in the study immobilise the limb for 6 weeks or longer, which is contrary to the concepts quoted in the literature of the last two decades.[15,16,17,18] A wide variety of accelerated rehabilitation protocols were described by the respondents. Some protocols were far less aggressive than the prescribed regimen with some respondents immobilising the limb for up to six weeks. These findings indicate that the interpretation of accelerated rehabilitation varied among the respondents. This perhaps explains the finding that 13% of respondents do not believe physiotherapy is of benefit post-operatively. In addition, 45% of respondents were not sure, or did not believe, there was adequate communication between Orthopaedic surgeons and physiotherapists. A possible reason for this lack of communication may be that the requests made to physiotherapists by Orthopaedic surgeons for certain rehabilitation protocols are based on incorrect assumptions, and varied interpretation of terminology.

The strengths of this study were that, firstly, a cen-

sus study design was used. This is probably the most representative research design available, because it canvasses an entire population, and thus no selection bias ensues, although a non-response bias is possible because persons self-select as respondents in the census. Secondly, the response rate of the questionnaire was high. Therefore we can accept with a high degree of probability the findings of the study as being representative of South African Orthopaedic surgeons as a whole.

In conclusion, it appears that South African Orthopaedic surgeons use the majority of surgical practices of ACL repair as described in the recent literature. However, the procedures used by Orthopaedic surgeons who perform ACL reconstructions infrequently (one or less than one per month), particularly those of arthroscopic procedures and post-operative rehabilitation, need to be reviewed. A cause for concern is that the registrar population who replied to the questionnaire are using techniques of ACL repair which have been superceded by more modern methods, and which are used more frequently in the private sector. This concern can perhaps be resolved by involving the private Orthopaedic surgeons in Orthopaedic registrar training where deficiencies exist.

## REFERENCES

1. Hey-Groves. The crucial ligaments of the knee joint: their function, rupture, and the operative treatment of the same. Brit J Surg 1919:7(4):505-515
2. Levin S. ACL Reconstruction - The Best Treatment Option? Physician and Sportsmedicine 1992:20(6):151-161
3. Cross MJ, Harris J and Slater HK. Recent Developments in the Treatment and Repair of Anterior Cruciate Ligament Injuries in the Athlete. Sports Med 1990:10(6):349-354
4. Jones KG. Reconstruction of the Anterior Cruciate Ligament. A Technique Using the Central One Third of the Patellar Ligament. J Bone Joint Surg 1963;45-A(5):925-932
5. Clancy WG et al. Anterior and Posterior Cruciate Ligament Reconstruction in Rhesus Monkeys. J Bone Joint Surg 1981:63-A(8):1270-1284
6. Noyes FR et al. Biochemical Analysis of Human Ligament Grafts used in Knee Ligament Repairs and Reconstruction. J Bone Joint Surg 1984;66-A(3):344-352
7. Yasuda K et al. Quantitative Evaluation of Knee Instability and Muscle Strength after Anterior Cruciate Ligament Reconstruction using Patellar and Quadriceps Tendon. Am J Sports Med 1992:20(4):471-475.
8. Paulos LE et al. The Gore-Tex Anterior Cruciate Ligament Prosthesis - A Long Term Follow-up. Am J Sports Med 1992:20(3):246-252
9. Engebretsen L et al. A Prospective, Randomised Study of Three Surgical Techiques for Treatment of Acute Ruptures of the Anterior Cruciate Ligament. Am J Sports Med 1990:18(6):585-590
10. Noyes FR et al. The Symptomatic Anterior Cruciate-Deficient Knee - Part 1: The Long Term Functional Disability in Athletically Active Individuals. J Bone Joint Surg 1983:65-A(2):154-162
11. Hamberg P et al. The Effect of Diagnostic and Operative Arthroscopy and Open Meniscectomy on Muscle Strength in the Thigh. AM J Sports Med 1983:11(5):289-292.
12. Wilk KE. Andrews JR. Curent Concepts in the Treatment of Anterior Cruciate Ligament Repair. JOSPT 1992:15(6):279-293
13. Appell H-J. Muscular Atrophy Following Immobilisation - A Review. Sports Med 1990:10(1):42-58
14. Paulos LE et al. Knee Rehabilitation after Anterior Cruciate Ligament Construction and Repair. Am J Sports Med 1981:9(3):140-149
15. Noyes FR. Mangine RE. Early Knee Motion after Open and Arthroscopic Anterior Cruciate Ligament Operation. Am J Sports Med 1987:15(2):149-160
16. Fernyhough J. Razza BE. Tension Pneumarthrosis Complicating Arthroscopy of the Knee. Am J Sports Med 1992:20(4):479-480
17. Austin KS. Sherman OH. Complications of Arthroscopic Meniscal Repair. Am J Sports Med 1993:21(6):864-9
18. Shelbourne KD. Nitz P. Accelerated Rehabilitation after Anterior Cruciate Ligament Reconstruction. Am J Sports Med 1990:18(3):292-299
19. Fu FH. Woo SL-Y. Irrgang JJ. Current concepts for Rehabilitation following Anterior Ligament Reconstruction. JOSPT 1992:15(6):270-278
20. Paessler HH. Deneke J. Dahners LE. Augmented Repair and Early Mobilization of Anterior Cruciate Ligament Injuries. Am J Sports Med 1992:20(6):667-674
21. South African Orthopaedic Association Mailing List 1993, 1994
22. Mackenzie D.B. Presidential Address of The South African Orthopaedic Association: The Road Ahead. J Bone Joint Surg [BR] 1992:74-B:Supp 11 190-195 □

---

3rd edition. Philadelphia/London. Lea and Ferbiger.
15. Lind AR (1963). A physiological criterion for setting thermal environmental limits on everyday work. Journal of Applied Physiology. 18(1):51-56.
16. Murray R (1995). Fluid needs in hot and cold climates. International Journal of Sports Nutrition. 5:562-573.
17. Chambers AB (1970). A psychometric chart for physiological research. Journal of Applied Physiology. 29(3):406-411.
18. Tanner JM (1964). The Physique of the Olympic Athlete. London: George Allen and Urwin Ltd.
19. Durnin J and Womersley J (1974). Body fat assessed from total body density and its estimation from skinfold thickness. Measurement on 481 men and women aged 16-72 years. British Journal of Nutrition. 32:77-97.
20. Polar heart rate analysis software (1993). Version 4.10 for Windows. Copyright 1992 Polar Electro OY, Professorintie 5. SF-90 440 Kempele. Finland.
21. Gambke B. Berg A. Fabian K. Frankaur M. Lormes W. Haber P. Hartmann U, Kamber M, Rosskopf P, Schwarz L,
Steinacker JM (1994). Multicentre evaluation of a new portable system for determining blood lactate. Poster Presentation at ACSM Annual Meeting, Indianapolis, June 1-4.
22. Statgraphics (1991). Statistical graphics system by Statistical Graphics Corporation. STSC, Inc. 2115 East Jefferson Street, Rockville, Maryland 20852. USA.
23. Selinger V (1968). Energy metabolism in selected physical exercises. Internationale Zeitschrift fur Angewphysiologie Einschl Arbeitsphysiologie. 25:104-120.
24. Walker J (1973). Conditioning requirements for power volleyball. International Volleyball Review. 3:39-40.
25. Fardy PS, Hritz MG. Hellerstein HK (1976). Cardiac responses during women's intercollegiate volleyball and physical fitness changes from a season of competition. Journal of Sports Medicine. 16:291-300.
26. Brooks GA (1988). Blood lactate acid: Sports "bad boy" turns good. Sports Science Exchange: Sports Physiology/Biochemistry. 1(2):April. Gatorade Sports Institute.
27. Essen B and Kaijser L (1978). Regulation of glycolysis in intermittent exercise in man. Journal of Physiology. 281:499-511. □
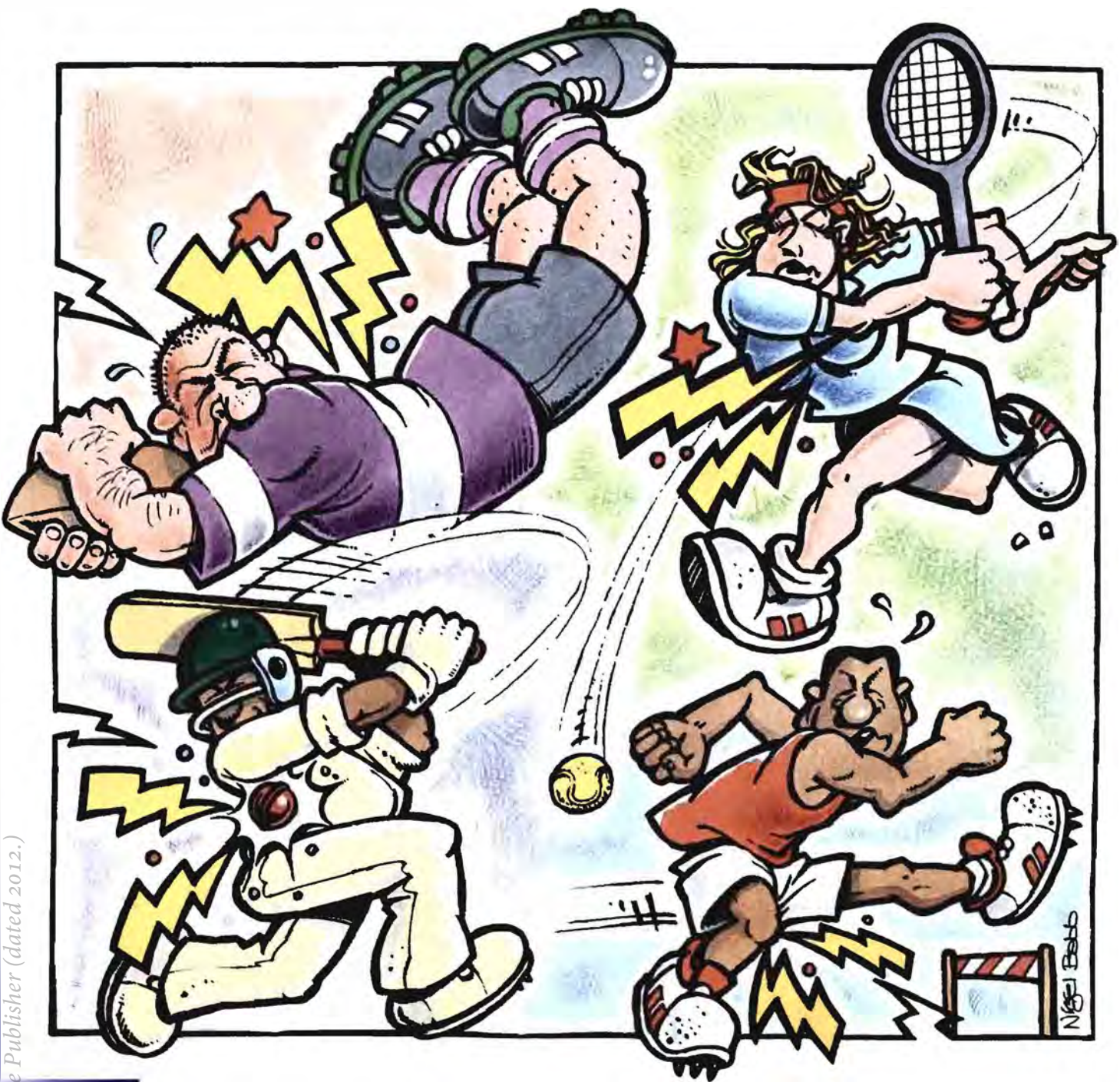
---

# **E***veryone feels pain at some time . . .*

# Froben.100

- Significant analgesic, anti-inflammatory and antipyretic action.
- Rapid therapeutic response ensured[1].
- Action Pack containing 15 tablets.
- Pack of 12 suppositories.

S3 Flurbiprofen 100 mg.

Tablets 100 mg, suppositories 100 mg.

knoll
**BASF** Pharma

# Stick it to acute pain and inflammation.

## TransAct LAT patches for NSAID power
### that goes to work immediately and continues to work for up to 12 hours.

- effective locally acting therapy against pain and inflammation.[1]
- well tolerated.[1]
- convenient non-sticky, non-greasy formulation with no residue.
- cooling sensation on skin, with a pleasant menthol aroma.[1]

# TransAct™ LAT

We've got acute pain and inflammation covered.

BOOTS
HEALTHCARE
40 Electron Avenue, Isando 1600

SS TransAct. Each patch contains 40 mg Flurbiprofen. Reg. No. 28/3.1/0268

Flurbiprofen 40 mg

Reference: 1. Ritchie L, Sugawara S, Ishigami M, Satoh T, Suzuki K. Local action transcutaneous flurbiprofen (flurbiprofen LAT) in the treatment of acute musculoskeletal conditions: Review of European and Japanese experience.

TRA1195G041

5000