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# THE FLUID REPLACEMENT NEEDS OF YOUNG TENNIS PLAYERS:

## Implications for tennis coaches

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KOSTAS KAVASIS

A Thesis Submitted to the Faculty of Education,

University of the Witwatersrand, Johannesburg,

in fulfilment of the requirements

for the degree of

Master in Education

February 1993

## DECLARATION

I declare that this thesis is my own, unaided work. It is being submitted for the Degree of Master in Education (M.Ed) in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination in any other University.

Kavasis

Kostas Kavasis

On the 26 day of FEB , 1993

To my mother and father

## ABSTRACT

This study aimed to assess selected physiological, osmoregulatory and thermoregulatory responses to tennis play in children. Twenty-four young male tennis players ($\bar{X}$ age: 13.3±1.31 years) were studied while playing for 90 min under warm environmental conditions (WBGT index= 13-19). Ad libitum consumption of pure water was permitted.

Questionnaires were used in order to elicit information regarding the health status, degree of acclimatization and pre-match trait and state anxiety of the subjects. The physiological variables assessed included pre- and post-match rectal temperature ($T_{re}$), heart rate (HR) and oxygen consumption ($\dot{V}O_2$) during the match. The biochemical observations included pre- and post-match blood glucose, haematocrit (Hct), haemoglobin (Hb), plasma electrolyte ($Na^+$, $Cl^-$, $K^+$, $Mg^{++}$) and total plasma protein (TPP) concentrations.

The findings revealed that the young tennis players investigated in this study experienced moderate levels of pre-competitive trait and state anxiety. The estimated mean exercise intensity of 50-55% of $\dot{V}O_2$ max for a duration of 90 min resulted in a $T_{re}$ increase of 0.73 °C. Mean percentage dehydration was 0.80±0.25. Plasma volume (PV) changes were insignificant and were positively related to TPP changes. Mean plasma $Na^+$ and $Cl^-$ concentrations increased by 0.88 mmol.$\ell^{-1}$ and 2.2 mmol.$\ell^{-1}$ respectively. The subjects' mean HR response (145 bpm) of a subset of the sample(n=13) and estimated $\dot{V}O_2$ (21.38 ml.kg$^{-1}$.min$^{-1}$) represented a 55% of the age adjusted HRmax reserve and approximately a 50-55% of $\dot{V}O_2$ max respectively. The time spent within the target heart rate range (60-85% HR max reserve) comprised only 33% of the match duration. Mean energy expenditure was estimated to be 1772 KJ during the 90 min period.

It was calculated that in order to prevent harmful levels of dehydration under warm environmental conditions and at an exercise intensity of tennis play of approximately 55% of the age adjusted HR max reserve or 50-55% of $\dot{V}O_2$ max, consumption of 4.44-6.81 ml.Kg$^{-1}$.hr$^{-1}$ or 200-360 ml.hr$^{-1}$ of water is recommended in young tennis players possessing a body mass of 45-53 Kg. The precise quantity was concluded to be a function of the mass and exercise intensity of the subjects and the environmental heat stress index during play.

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# CHAPTER ONE

# INTRODUCTION

1.1 INTRODUCTION TO THE PROBLEM
1.2 STATEMENT OF THE PROBLEM AND SUBPROBLEMS
1.3 SCOPE OF THE STUDY
1.4 LIMITATIONS OF THE STUDY
1.5 HYPOTHESIS

## 1.1 Introduction to the problem

The recent growth of tennis among young children and adults in South Africa has resulted in an increasing concern among authorities regarding the physiological well-being and potential health risks of young children exercising in the heat.

Tennis is an intermittent activity characterized by short bouts of high exercise intensity interspersed with periods of rest or activity of moderate to low intensity. Children regularly involved in competition can, however, be confronted with strenuous matches, particularly when playing against an opponent of equal ability. These may contain lengthy rallies during which the exercise intensity is high and if taking place in the heat, may result in large degrees of physiological stress.

Concern stems mainly from the laboratory studies done by Bar-Or(1983), Drinkwater et al.(1977), and Haymes et al.(1975), who found that children exercising in warm or hot climatic conditions are less efficient thermoregulators than are adults. They are therefore, according to the American Academy of Paediatrics (AAP) (1982; 1983), at a special risk of becoming dehydrated and acquiring heat-related illnesses, such as heat stroke or heat exhaustion.

The AAP (1982; 1983), suggest the following reasons why children are less efficient thermoregulators than are adults:

(1) Children have a greater BSA/mass ratio than do adults which induces a greater heat transfer between the environment and the body. This implies that when ambient temperature ($T_{air}$) exceeds mean skin temperature ($\bar{T}_{sk}$), the rise in core temperature ($T_c$) at a given heat index will be greater than that of adults.

(2) Children produce more metabolic heat per mass unit than do adults at a given workload.

(3) Children have a lower sweating capacity than do adults, and show a reduced capacity to convey heat by blood from the body core to the skin.

Children are thus reported to possess less heat tolerance, including lower sweat rates and higher $T_{re}$ in comparison to adults at a given work intensity and heat stress index (Bar-Or, 1983, Drinkwater et al., 1977, and Haymes et al., 1975). As the studies on which these assumptions are based are limited, further work is required to confirm these theories. Furthermore, it is significant to determine whether young tennis players are at a risk of dehydration and whether there is the need for fluid replacement in young tennis players during play in hot environmental conditions.

This study thus investigated the fluid losses and thermal responses of young tennis players, aged between 11-12 and 14-15, playing tennis in the heat of the day, during the mid-summer months in South Africa.

## 1.2 Statement of the problem and subproblems

The central problem to be investigated in this study is the identification of the fluid replacement needs of young tennis players (i.e. specific fluid volumes required in a given time period), and the provision of recommendations for school tennis coaches which will be of prophylactic benefit to young tennis players participating in the sport.

With regard to the twenty-four tennis players investigated in this study the following sub-problems were addressed:

1.2.1 The competitive stress levels experienced as reflected in both trait and state anxiety scores.

1.2.2 The metabolic demands of the game in terms of HR, $\dot{V}O_2$ and related energy expenditure.

1.2.3 The existence of tennis-induced hyperthermia in children.

1.2.4 The relationship between post-match $T_{re}$ and hydration level, range of exercise intensity (HR range) and percent body fat.

1.2.5 The potential hemoconcentration or hemodilution response during competitive tennis play in young tennis players.

1.2.6 The plasma electrolyte ($Na^+$, $Cl^-$, $K^+$, $Mg^{++}$), total plasma protein (TPP) and blood glucose response to competitive tennis play.

1.2.7 The incidence of voluntary dehydration (a state of fluid deficit despite the ad libitum provision of fluid) among young competitive tennis players.

## 1.3 Scope of the study

This study was limited to twenty-four male tennis players, aged between 11 to 15 years and implemented during the South African mid-summer (WBGT index= 21) at an altitude of approximately 1800m. Matches were 90 min long and data collected from the subjects were restricted to information obtained from questionnaires, basic anthropometrical measures, telemetry, recordings of $T_{re}$ and the sampling of venous blood and expired air.

## 1.4 Limitations of the study

1.4.1 Due to practical considerations, it was not possible to pre-determine or set the environmental conditions on the days of testing as these sessions had to be planned in advance.

1.4.2 It is well documented that recruiting children as subjects is a difficult procedure, especially when experimental measures include blood sampling. This investigation was thus limited to children who consented to participate in such a procedure, and children motivated and interested to learn more about physiological response to tennis.

1.4.3 The use of two types of Sport Tester HR monitors (Kempele, Finland), namely the PE 300 [measuring heart rate ranges (HRR)] and PE 3000 (measuring continuous HR), limited the continuous monitoring of HR response during tennis play to a subsample of subjects (n=13). Thus, exercise intensity was expressed as time spent in the three HRR, i.e. low exercise intensity (the time spent by the subject playing tennis at <60% of HRmax reserve), medium intensity (the time spent at 60-85% of HRmax reserve), and high intensity (the time spent at > 85% of HRmax reserve), in the complete sample(n=24).

1.4.4 Application of the conclusions reached by this study are limited to the age, gender, physiological and psychological characteristics of the limited sample (n=24) as well as to the environmental conditions in which the data collection took place.

## 1.5 Hypothesis

A number of hypotheses were formulated prior to the commencement of the research. It was postulated that:

1.5.1 The fluid intake recommendation of 300 $ml.hr^{-1}$ (AAP, 1982; 1983) is inappropriately standardized for children.

1.5.2 Children will demonstrate the incidence of voluntary dehydration (Bar-Or et al., 1980; AAP, 1982; 1983) during the ninety minutes of tennis play in hot environmental conditions.

1.5.3 Competitive tennis play will impose strain of moderate intensity on the cardiovascular system of the children.

1.5.4 Blood volume (BV) compartment changes will show great variability among the subjects.

# CHAPTER TWO

## REVIEW OF THE LITERATURE

2.1 INTRODUCTION

2.2 TENNIS

2.3 THERMOREGULATION AND OSMOREGULATION DURING EXERCISE

2.4 PHYSIOLOGICAL AND METABOLIC RESPONSE TO EXERCISE IN CHILDREN

2.5 HEAT TOLERANCE AND HYDRATION IN CHILDREN

2.6 SELECTED INSIGHTS INTO COMPETITIVE STRESS IN THE ATHLETE

2.7 CONCLUSION

### 2.1 INTRODUCTION

This review provides a general introductory overview of literature related to the physiological response to tennis play and discussion of selected principles which underly homeostatic thermoregulation and osmoregulation. This is followed by a brief discussion of petinent issues in pediatric exercise physiology with particular reference to the fluid replacement needs of children and their ability to exercise in the heat. Finally, a brief section is devoted to selected aspects of competitive stress in children and the interelationship between mental stress and physiological response to exercise.

### 2.2 TENNIS

While much research has been conducted on tennis equipment and on the aetiology and anatomical presentation of tennis elbow, the physiological stress imposed on tennis players has received little attention. This particular section will examine existing evidence of the physical demands of this sport and focus on the energy

expenditure, cardiovascular, thermo- and osmoregulatory, and other physiological responses to tennis play. It will also identify the paucity of literature which is available with regard to the physiological response to tennis play in children.

### 2.2.1 HR response to tennis

HR is well documented as an indirect predictor of exercise intensity (Astrand and Saltin, 1961; Astrand, 1967; Ekblom et al., 1972). A number of studies have thus investigated HR response to tennis (Friedman et al., 1984; Elliot et al., 1985; Therminarias et al., 1991). A summary of the results of these studies is presented in Table 2.1.

As opposed to Friedman et al.(1984), Morgans et al.(1987), Eichner(1988) and Elliot et al.(1985), who, using the HR response during tennis play as the criterion, found tennis to be of sufficient exercise intensity to elicit a significant cardiovascular training effect, the findings of Seliger et al.(1973), Docherty(1982), Bergeron et al.(1991) do not confirm this notion. It is possible that this contradiction is due to the method of prediction of percent of maximum HR reserve i.e. that the calculation of a particular fraction of the maximum HR may result in a significant overestimation of exercise intensity when the resting HR (the non-zero value of resting HR) is not taken into account (Davies and Convertino, 1975). Further possible reasons include the variance in the percentage effort of the players (Friedman et al., 1984; Docherty, 1982), the duration of the period during which HR was recorded (Seliger et al., 1973) and the absence of control for age and environmental conditions (Friedman et al., 1984; Seliger et al., 1973).

Morgans et al.(1987) and Eichner(1988) in studies monitoring HR during singles and doubles, concluded that singles competition in tennis meets the maximum HR reserve intensity criteria established by the American College of Sports Medicine (ACSM) for

**Table 2.1: HR response to singles tennis play. A summary of studies: 1973-1991.**

| Study (number of subjects, gender) | Age (yr) | Duration (min) | Environ. Condit. (DB) | Heart rate (bpm/ PMHR) | Comments:- |
|---|---|---|---|---|---|
| Seliger et al.(1973) (16,M) | 25 | 10 | - | 143 | Environmental conditions, may have influenced the HR response (Astrand and Rodahl, 1977) |
| Docherty (1982) (42,M) | 26 | 30 | 21-24 °C | 68-70% | Their mean HRR across all three groups was 133-137bpm |
| Friedman et al.(1984) (28,M) | 45-72 | 60 | 35.1 °C | 60-100% | Four subjects were 20% over-weight and 5 were smokers; high ambient temperature may have influenced the HR response |
| Elliot et al.(1985) (8,M) | 20 | 60 | 23 °C | 153 | The monitoting of HR was completed at selected time intervals |
| Bergeron et al.(1991) (8,M) | 20 | 85 | 17 °C | 145 | Mean percentage HR was 61% calculated from MHRR |
| Therminarias et al.(1991) | | | | | The veterans displayed higher HR during the last 30min of play ($p<0.05$) |
| (9,F) | 21 | 90 | 27 °C | 157 | |
| (10,F) | 46 | 90 | 27 °C | 156 | |

All data are expressed as means; M=Male, F=Female; DB=Dry Bulb Temperature; HR=heart-rate; PMHR=Percentage of Maximum Heart Rate; MHRR=Maximal Heart-Rate Reserve; bpm=beats.min$^{-1}$

developing and maintaining cardiorespiratory fitness (ACSM, 1978). However, doubles competition does not meet those criteria (Morgans et al., 1987; Eichner, 1988) and should not, according to the position statement of the ACSM (1978), be considered as an exercise regimen for developing cardiorespiratory fitness.

### 2.2.3 Work rate and energy expenditure in tennis

Seliger et al.(1973), Docherty(1982), and Bergeron et al.(1991) investigated tennis with respect to energy expenditure and concluded that tennis is a sport which places submaximal load on the cardiovascular and respiratory systems of players. Furthermore, Docherty(1982) also stated that playing tennis is of little value in improving or maintaining fitness levels of individuals who have maximal oxygen consumption ($\dot{V}O_2max$) values greater than 35ml.kg$^{-1}$.min$^{-1}$. To the contrary, a study reported by Friedman et al.(1984) concluded that those who play regular singles' tennis can achieve outstanding fitness levels similar to those of running and swimming training. In the latter study, energy expenditure was, however, predicted from HR during play, and the high mean $T_{air}$ (35,1 °C) may have resulted in an elevation of the mean HR. Furthermore, obesity and habitual cigarette smoking may have influenced the HR response during tennis play and therefore affected the results and conclusions.

In the study of Seliger et al.(1973), players competed in 10 min games wearing Douglas bags. The mean energy expenditure was calculated to be 43.65 kJ.min$^{-1}$ during play. These authors estimated that 88 percent of the energy during tennis play was derived from oxidative metabolism and the remaining 12 percent from oxygen independent metabolism, and that the mean oxygen uptake ($\dot{V}O_2$) during the 10 min period of play was about 50 percent of $\dot{V}O_2$ max.

These findings of Seliger et al.(1973) contrast with those of Fox(1984) who listed the approximate contributions of the energy systems required to play singles tennis as 70 percent from the 'phosphagen' and glycolytic metabolism, 20 percent from the glycolytic and oxidative metabolism and 10 percent from oxidative metabolism alone. Furthermore, the observation of low accumulation of lactate in the study of Kindermann and Keul(1977), which was confirmed by the relatively small size of the "oxygen debt" and the mean blood lactate levels of 3(±1)mM, support the findings of Fox(1984) and Elliot et al.(1985), who observed a marginally higher HR between tennis rallies (X= 153bpm) when compared to the playing periods (X= 150bpm). These authors contend that the significantly higher peak HR recorded during recovery compared to HR recorded during play, suggests that the predominant energy sources being utilized during the rally are the 'phosphagen' energy systems with partial restoration of the ATP-CP and myoglobin stores occurring during the subsequent recovery period (Elliot et al., 1985). These hypotheses do, however, require confirmation. The use of magnetic resonance spectroscopy, rather than reliance on indirect predictions, would provide more reliable information.

### 2.2.4 Other physiological variables examined in tennis

#### 2.2.4.1 Cardiovascular Adaptations

Dentsch et al.(1988) found that trained tennis players respond with a lower cardiac output for a given level of submaximal exercise when compared to untrained individuals.

This finding confirms previous reports (Foster, 1986; Hammond et al., 1984; Mahler et al., 1984). Kuel et al.(1991), when studying professional tennis players of world ranking found that heart-volume (HV) was 20-30% higher than in untrained persons. End-diastolic volume (EDV), left ventricular muscle mass (LVM) and stroke volume

(SV) were elevated while the ratios of SV and of LVM to EDV and HV were within normal limits. These researchers concluded that the changes may be indicative of the possible development of an "athletes heart" in tennis players (Kuel et al., 1991).

Significant increases in systolic and diastolic blood pressure, even in those persons who are normotensive at rest, have been reported during singles tennis play (Jette et al., 1991). It is possible that this increase in blood pressure during tennis play could be related to the adaptive physiological increase in wall thickness reported by Kuel et al.(1991).

Sinoway et al.(1986) studied the maximal blood flow in the dominant forearms of tennis players and reported that the greater peripheral cardiovascular adaptation is a possible aetiological factor resulting in an increased ability of skeletal vasculature of the dominant forearm to dilate during maximal exercise when compared to control subjects.

Furthermore, Vodak et al.(1980) examined high density lipoprotein (HDL)-cholesterol and other plasma lipid and lipoprotein concentrations in middle-aged male and female tennis players and concluded that frequent tennis playing is associated with increased plasma HDL-cholesterol concentration and that this relationship is independent of other factors known to alter plasma HDL-cholesterol concentration.

However, Vodak et al.(1980) and Powers and Walker(1982) questioned their conclusions with regard to the physiological profile of tennis players asking whether regular tennis participation produces above average fitness or whether the physiological profile of the tennis playing population is indicative of physically superior individuals.

### 2.2.4.2 Thermoregulatory Response

Elliot et al.(1985) and Dawson et al.(1985) examined the responses of male college-level players under cool (WBGT index= 21, humidity= 22.7%) and hot (WBGT index= 30.9, humidity= 65%) conditions. Dawson et al.(1985) showed a mean difference of 0.5 °C in $T_{re}$ when the game was played at the same intensity in hot conditions. The mean body mass loss in this study was 2.4% as opposed to the mean body mass loss of 1.3% in the study of Elliot et al.(1985), which took place under cool conditions (mean HR= 152bpm). The average fluid intake during the study of Elliot et al.(1985) was 250ml.$h^{-1}$, representing 27% of the total fluid lost. It was concluded that a greater degree of thermoregulatory and cardiovascular strain is experienced by the players during performance in the hot, when compared to cool, conditions.

### 2.2.4.3 Fluid Balance

The gradual rise in mean HR among female veterans during the last 30 min of the 60 minute-games in the study of Therminarias et al.(1991), was attributed to an 11.9 percent reduction of PV and consequent reduction in cardiac filling. However, mean PV reduction in the study of Mitchell et al.(1992) was 0.90% for the group receiving liquid supplementation containing carbohydrate (7.5 g.100$ml^{-1}$) and 1.05% for the group receiving water alone. Fluid replacement was maintained at a rate of 800ml.$hr^{-1}$ during simulated matches of three hours and under hot environmental conditions (WBGT index= 26.7). Mitchell et al.(1992) concluded that although carbohydrate ingestion is not of benefit to either performance or fluid balance purposes during three hours of tennis match-play, consumption of 800ml.$hr^{-1}$ prevented severe dehydration under the extreme environmental conditions in which the study took place.

#### 2.2.4.4 Metabolic Responses

Copley(1984) in a study comparing professional and amateur tennis players, concluded that strenuous competitive singles tennis playing has an insignificant effect ($p > 0.05$) on blood glucose, blood lactate and plasma electrolyte levels and that there are no differences in these responses in the two categories of players. Low post-exercise blood lactate levels have also been reported by Therminarias et al.(1990) among female veteran and young tennis players.

### 2.2.5 Children and tennis

Birrer et al.(1986) studied correlations of performance variables in 500 preadolescent tennis players. These researchers concluded that track parameters (i.e. running, jumping) were found to be weak predictors of overall tennis performance. Strongly positive correlations were found between tournament play and forehand drive, backhand volley, smash ability and number of leg movements ($r > 0,85$).

An investigation by Mero et al.(1989), which compared an untrained group and trained group of prepubescent tennis players, reveals no differences in hormonal profile (cortisol, testosterone and growth hormone) between these prepubescent groups. There was, however, a significantly lower oxygen consumption of the prepubescent tennis players at anaerobic threshold (Mero et al., 1989). These researchers attributed this difference to the nature of tennis play, assuming that tennis is played at the level of "anaerobic threshold" or slightly below it in children.

### 2.2.6 Conclusion

A review of the existing literature examining the physiological response to tennis play thus reveals that very little is known of the response of children to tennis play. This is

an area which requires further investigation. For the purpose of this thesis, primary focus will be placed on the thermoregulatory and osmoregulatory control and adjustment in children during tennis play.

## 2.3 THERMOREGULATION AND WATER BALANCE DURING EXERCISE

### 2.3.1 Introduction

Small fluctuations in osmotic pressure and temperature can disrupt biochemical activities within the cells (Guyton, 1986; Senay, 1979). Osmoregulation, the process by which the osmotic pressure of the blood and tissue fluids is kept constant, is necessary not only with respect to water content, but also with respect to the concentrations, relative and absolute, of the various solutes normally present in the body fluids. It is also well accepted that irrespective of fluctuations in the environmental temperature, the body seeks to maintain a $T_c$ of approximately 36.7 °C and that disturbance of thermal balance can result in cardiorespiratory and central nervous system disturbances (Hubbard and Armstrong, 1988).

### 2.3.2 Thermoregulatory control and adjustments to heat exposure

Both during exercise and rest, heat is continually being produced by the body as a by-product of metabolism and the body attempts to remain in heat balance by loosing heat to the surroundings (Canong, 1989). It is when the rate of heat production is equal to the rate of heat loss, that the person is said to be in heat balance (Guyton, 1986). To maintain thermal equilibrium the body must quantitatively balance the thermoregulatory mechanisms in relation to metabolic heat production and environmental stressors (Canong, 1989).

The temperature gradient between the blood and the surrounding tissues and the skin and the surrounding environment is a major factor influencing maintance of heat balance (Mitchell, 1977). With an elevation of core body temperature, the thermostatic system employs two primary mechanisms to reduce body heat. Firstly, vasodilation of the cutaneous blood vessels results in greater heat flow via the blood from the core to the skin, which in turn activates sweating. The consequent evaporation of sweat reduces body heat storage. Secondly, decreased heat production also results from inhibition of chemical thermogenesis, which is in turn, brought about by decreased sympathetic stimulation or circulating norepinephrine and epinephrine in the blood (Guyton, 1986). These hormones have the ability to increase the rate of cellular metabolism by uncoupling oxidative phosphorylation for the oxidation of foodstuffs to produce the energy required of the body (Guyton, 1986).

More specifically, Brengelmann(1977) and Gisolfi and Wenger(1984) described that skin blood flow (SBF) and sweating response operate according to the disturbances in $T_c$ and $\bar{T}_{sk}$ temperature. Figure 2.1 illustrates how sweating and SBF responses (R), depend on changes in the weighted sum of afferent thermal information from $T_c$ and $\bar{T}_{sk}$ temperature ($T_{ws}$) (Gisolfi and Wenger, 1984).

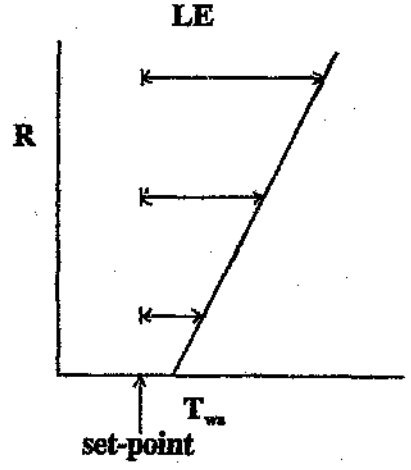



**Fig. 2.1.** ***Set point and threshold $T_{ws}$ that elicits an effector response, R. R changes linearly with the load error (LE), or the difference between the set-point and $T_{ws}$. Reprinted with permission from Gisolfi and Wenger(1984).***

This approach to thermoregulatory response suggests that a perturbation of the $T_{ws}$ away from the set point ($T_{set}$) and, at some threshold will initiate an effector response i.e. SBF, sweating (Brengelmann, 1977; Gisolfi and Wenger, 1984). Elevation of $T_{ws}$ ($T_c$ and $\bar{T}_{sk}$) produces a load error of the thermoregulatory system, which is defined by the quantity $T_{set}$-$T_{ws}$, and works as a conventional proportional control system (Brengelmann, 1977). Thus, each effector response is recruited when the load error reaches a value characteristic for that response, and each response has a characteristic $T_{ws}$ threshold, which may be offset from $T_{set}$ (Brengelmann, 1977; Gisolfi and Wenger, 1984). For example, with exercise $T_c$ rises until a signal develops to call for effect or activity adequate to dissipate the large amount of heat produced and bring about thermal balance by acting on elevated $T_c$. When environmental changes occur, only small changes in $T_c$ and $\bar{T}_{sk}$ are to be expected since the system presumably has high gain and will correct for the change with only a small change in $T_c$ (Gisolfi and Wenger, 1984).

Different effectors have different thresholds but if all effector thresholds $T_{ws}$ for initiating all heat-dissipating responses are shifted in the same direction by an intervention (as occurs in heat acclimatization and endurance training, hyperosmolar state, fever and time of the day), the set-point of the controller is considered to have shifted in that direction (Gisolfi and Wenger, 1984). If the slope of the R-$T_{ws}$ relation is changed, the thermosensitivity (slope or gain) of the response is said to have altered (Gisolfi and Wenger, 1984). This is in accordance with the principles of homeostasis and the potential adaptation of the thermoregulatory system when stresses of exercise and acclimatization are imposed on the human body.

It has been estimated that $T_c$ exerts nine times the influence of $\bar{T}_{sk}$ over sweating or SBF (Nadel et al., 1971), but that the relative importance of $\bar{T}_{sk}$ is greater in smaller species (Roberts et al., 1974; Gisolfi and Wenger, 1984). Sweating response is described to be relatively independent of cardiovascular control (Solack et al., 1985) and dependent on thermal inputs ($T_{re}$, $\bar{T}_{sk}$), and other nonthermal inputs

(interventions) i.e. exercise, heat acclimatization, dehydration, and plasma osmolality (Fortney et al., 1984; Nadel et al., 1974). More specifically, Fortney et al.(1984) describe that plasma hypertonicity influences SR threshold rather than SR sensitivity and that hypovolemia reduces SR sensitivity, but not the SR threshold (Fortney et al., 1981). Control of sweating has also been reported to be related to the quantity of circulating catecholamines (Robertshaw, 1977) and antidiuretic hormone (ADH) (Senay, 1979).

SBF control is influenced by both thermoregulatory and cardiovascular systems i.e. thermal information from ($T_{re}$, $\bar{T}_{sk}$) other nonthermal inputs or interventions i.e. exercise intensity, upright posture, hypovolemia, hyperosmolality, positive pressure breathing, training and heat acclimatization (Roberts et al., 1977; Wenger et al., 1975; Nadel et al., 1979; Johnson, 1979; Fortney et al., 1981; Fortney et al., 1984).

It can thus be concluded that a change in set point produces a corresponding change in the threshold for each thermoregulatory response. The profuse and faster onset of sweating response observed in a subject after training and heat acclimatization (Nadel et al., 1974), could be explained by an increased responsiveness of the sweating mechanism, a lowered internal temperature threshold for sweating and improvement in the sweating to SBF ratio that accompanies physical training. The greater the degree of training and acclimatization, the greater the sweat response (onset and volume) at a given $T_c$ threshold of the subject (Nadel et al., 1974; Baum et al., 1976).

### 2.3.3 Thermoregulatory tolerance

When $T_{sk}$ exceeds $\bar{T}_{sk}$, the temperature gradient favours heat gain and evaporation of water from the respiratory passages and skin surface of the body contributes

substantially to dissipation of the internal heat. The thermal energy required for the evaporation of 1 liter ($\ell$) of sweat is 2.427 Kilojoules (KJ) of heat (Wenger, 1972). Evaporative heat loss is directly related to the amount of liquid vaporized irrespective of the ambient vapour pressure and solute content (Wenger, 1972). If sweat secretions evaporate, skin temperature drops, which further increases the temperature gradient between the deep tissues of the body and the skin surface (Ganong, 1989). However, if $T_{air}$ and relative humidity are high and wind velocity is low, the body reaches its limit of heat tolerance most rapidly due to inhibition of the evaporative process. Under such conditions radiation, conduction and convection will result in body heat gain from the environment. This implies that the skin surface becomes as warm as the $T_c$ temperature and internal heat can no longer be exchanged via the above mentioned mechanisms and body temperature rises.

During athletic events $T_{re}$ in excess of 40 °C have frequently been reported (Wyndham, 1977; Dill et al., 1977). This state of hyperthermia can result in mild cardiovascular and central nervous system disturbances (hypotension, fainting) or profound cellular damage, involving the brain, kidneys, liver and blood clotting mechanisms (Hubbard and Armstrong, 1988). The pathological condition of "heat stroke", which has been defined by Kew(1976) as a condition of acute thermoregulatory failure which manifests with disturbances of the central nervous system and hyperpyrexia (a $T_{re}$ in excess of 40,5 °C) has been described as a serious potential risk to life from violent exercise (Kew, 1976). The ACSM (1985) has therefore published a position stand on the "Preventation of thermal injuries during distance running" and recommended the use of color-coded flags to indicate the risk of thermal stress. The College stipulates that a WBGT index of 23-28 warrants the use of a "red flag" to alert both race organizers and participants to "high risk" environmental conditions. It recommends that distance races be rescheduled or delayed when WBGT index exceeds 28 (ACSM, 1985).

2.3.4 Disturbance of water balance: dehydration

Water comprises the largest component of the human body. In a man of average mass (80 Kg), it represents approximately 66% of his total body mass, depending on his percentage of body fat (Greenleaf, 1992). The intracellular fluid compartment would contain about 30 $\ell$ (37%) of water, whereas the extracellular fluid compartment would contain about 23 $\ell$ (29%) of water (Greenleaf, 1992).

During exercise, loss of water due to sweating can reach dramatically high levels. A SR of 3.71 $\ell.hr^{-1}$ has been reported in the world class distance runners, Alberto Salazar (after heat acclimatization) (Armstrong et al., 1986). To determine a subject's mass loss, the pre- and post-exercise body mass difference must be calculated. Adding liquid intake and subtracting the volume of urine voided will provide an indication of the total amount of fluid lost (Noakes et al., 1988). This total fluid loss can take the form of sweat losses, urinary water losses and the respiratory tract water losses and includes the loss of metabolic water (Noakes et al., 1988). When the sweat mechanism is activated to aid in the dissipation of heat, it, however, also occurs at the expense of intra and extracellular fluid volumes (Sawka, 1992). Unless a portion of this water loss is replaced, dehydration will result.

Loss of sweat resulting in a decrease of body mass of only 1% is known to initiate impaired exercise thermoregulation (Greenleaf and Harrison, 1986) and performance during high-intensity of exercise (Noakes, 1992). However, for a given water deficit, the magitude of exercise performance decrement may be related to the individual aerobic fitness and heat acclimatization (Buskirk et al., 1984; Caldwell., 1984).

Performance deficiencies that accompany dehydration include a) reduced muscular strength (Bosco et al., 1969), b) reduced endurance time (Claremont et al., 1976), c) reduced mental activity (Leibowitz et al., 1972), d) increased HR and reduced

stroke volume at submaximal exercise intensities (Saltin, 1964) and e) decreased renal blood flow and glomerular filtration rate (Radigan and Robinson, 1950; Smith and Robinson, 1952). Blood flow to visceral regions is reduced proportional to the $\%\dot{V}O_2max$ (Rowell, 1974) and the absolute intensity of exercise (Rowell, 1983). The reduction is greater when exercise is performed in the heat than in a cool environment (Radigan, 1950; Rowell et al., 1965).

The lack of clarity surrounding the factors determining the magnitude of changes in PV during progressive dehydration, could be due to the effect of factors including 1) the state of hydration of subjects at the start of the study (Senay, 1979), 2) different inter-study environmental conditions and modes of exercise, and 3) differences in subject status variables such as physical fitness and heat acclimatization (Harisson, 1985). It is important when referring to PV changes to examine exercise body position, duration of time in the position, specific time of blood sampling, environmental temperature during the blood sampling, subject physical fitness, heat acclimatization and dehydration level (Hagan and Diaz, 1978; Harisson, 1985).

The sweating response during exercise can be interpreted as extremely variable instead of tightly controlled (Nadel and Horvath, 1977). Variability in responsiveness is even more of a factor in studies of the cutaneous circulation, which is dependant on two control mechanisms, namely, the thermoregulatory and circulatory (Nadel and Horvath, 1977). These are all important factors contributing to the variability of individual responses to thermal stress during exercise and may profoundly influence BV responses quantitatively and qualitatively. Fluid shifts occur between the extracellular and intracellular space in an attempt to maintain equal osmolality between these compartments (Ganong, 1989). Since transvascular fluid shifts depends on capillary filtration pressure, oncotic (protein-colloids) and osmotic pressure (crystalloids-electrolyte) (Aukland et al., 1981; Sawka et al., 1984; Harisson, 1985; Nielsen, 1986), the potential of hemodilution or hemoconcentration exists. The degree of possible hemodilution and hemoconcentration resulting from

hemoconcentration resulting from exercise and heat stress would be determined by the establishment of a new equilibrium between the above factors (Aukland et al., 1981).

Nielsen(1986) reports that prolonged exercise leads to "thermal" dehydration which interferes with circulatory as well as thermoregulatory functions, due to the reduction of the BV and the increased osmolality of the body fluids. It has been reported that collapse of the subject is likely at about 7% body mass loss, if combined with exercise and heat stress (Greenleaf and Harrison, 1986). More recent studies (Noakes et al., 1988; 1991b) have shown that final $T_{re}$ in marathon-runners exercised in mild environments (19-22 °C) is more closely related to environmental conditions, metabolic rates sustained during the latter section of the race (Maughan, 1985; Noakes et al., 1991b) and individual susceptibility (Jardon, 1982), than to percentage dehydration.

### 2.3.5 Fluid replacement and glycogen breakdown as contributors to water regulation during exercise.

The replacement of fluids lost during prolonged exercise mainly via the sweating mechanism, is of vital importance as it serves to maintain PV and electrolyte balance (Costill et al., 1970). In determining the fluid replacement needs of an athlete, all possible factors contributing to loss as well to gain of mass during the athletic event, need to be considered. Olsson and Saltin(1970), mentioned that whenever total body water content is determined, the water produced during mitochondrial oxidation (CHO and Fat) and the water released from the glycogen stores of the body should be taken into consideration as this water does not contribute to the hydration status of the body. This is supported by Noakes et al.(1988, 1991b). However, in earlier work determining the fluid replacement needs during marathon running (Wyndham, 1977; Wyndham and Strydom 1969) and prolonged tennis play (Mitchell et al., 1992) this factor was not considered resulting in the overestimation of the fluid replacement

needs of the athletes. Based mainly on the study of Wyndham and Strydom(1969), the ACSM (1985) has suggested fluid replacement volumes of between 0.83 and 1.65 $\ell.h^{-1}$ for slow and fast runners respectively. These recommendations have, however, led to isolated reports of water intoxication (Noakes et al., 1985) and therefore require further consideration.

The problem of water deficit (dehydration) and optimal fluid replacement volumes during prolonged athletic events has been reviewed recently by Pandolph et al.(1988); Greenleaf(1992); Sawka(1992); and Gisolfi and Duchman(1992). However, in the above reviews, it was described that optimal fluid replacement volumes should equalize the sweat loss in a water balanced body. As has previously occurred (ACSM, 1985), the unrelated water released from glycogen breakdown to the body water deficit was not taken into account in the determination of the water deficit and thus optimal fluid replacement volumes.

The problem of fluid replacement becomes more complex when an athlete, a coach or an exercise physiologist has to consider the many physiological factors which influence the passage of ingested fluids through the stomach (digestion) and the small intestine (absorption). Murray(1987), reviewed the factors influencing gastric emptying and consequent fluid delivery to the intestine. Such factors are caloric content, beverage osmolality, volume of the beverage, exercise and environmental conditions, fluid temperature, sodium ($Na^+$) content, PH, fat content, anxiety, emotional distress, the time and contents of the previous meal, the hormonal response produced by the meal, diurnal variations and even the phase of the menstrual cycle (Murray, 1987). Hypohydration during exercise has been reported to decrease gastric emptying rates (Rehrer et al., 1990). In more recent studies, Rehrer et al.(1989) and Noakes et al.(1991a) have emphasized the effect of the volume of fluid in the stomach in regulating gastric emptying and confirmed large individual differences in rates of gastric emptying when ingesting the same concentration of a given CHO solution (Foster, 1990). More specifically, Noakes et al.(1991a), concluded that the delivery

concluded that the delivery of any ingested solution to the intestine, is determined primarily by the degree of gastric distension. They regarded carbohydrate concentration of the ingested fluid as a secondary factor in determining gastric emptying rates (Noakes et al., 1991a).

As it has been shown that approximately 3-4g of water are stored with each gram of glycogen (Olsson and Saltin, 1970), the greater the pre-exercise muscle and liver glycogen levels and glycogen breakdown capacity of a person, the greater the water release from the breakdown of glycogen. The importance of taking the water released from glycogen breakdown into account, is illustrated by the findings of the study of Kozlowskin and Saltin(1964). These researchers found that reduction in PV and extracellular space was more significant after dehydration caused by prolonged exposure to a high room temperature (thermal-induced hypohydration), than after a corresponding degree of dehydration during physical work (exercise-induced hypohydration).

In the study of Pugh et al.(1967); Wyndham and Strydom(1969) and Maron et al.(1975) the notion that individuals who sweat profusely during exercise, invariably fail to voluntarily replace their losses (Adolph, 1947), is confirmed. However, among other factors these findings may be explained by the fact that water released from glycogen stores during prolonged exercise enhances cardiovascular volume, decreases osmolality and plasma vasopressin and renin-angiotensin concentrations (Nose et al., 1988), and thus reduces the desire to drink water.

### 2.3.6 Physical training and heat acclimatization as contributors to water and thermal regulation during exercise and heat stress

Two factors can dramatically modify physiological responses to heat stress and exercise. These constitute heat acclimatization and physical training (Gisolfi and Bruce, 1989).

Heat acclimatization has been defined as the adaptive physiological process followed by an exposure to environmental and metabolic stress, which enhances the ability to function in the heat without distressing symptoms (Gisolfi, 1973). The documented physiological benefits to be obtained after the completion of the acclimatization procedure (7 to 10 days of daily exercise in the heat) can be summarized as follows: lower $T_{re}$ and $\bar{T}_{sk}$ and greater gradient from body core to surface at any given work load (Haymes and Wells, 1986), a greater capacity and sensitivity of the sweating mechanism (Gisolfi and Robinson, 1969; Roberts et al., 1977), diminished concentration of $Na^+$ in the sweat, kidney (Bar-Or, 1983; Allan and Wilson, 1971; Smiles and Ronbinson, 1971) and urine (Guyton, 1986), as the result of increased secretion of aldosterone, greater circulatory stability (increase in stroke volume and reduction in HR) resulted mainly by expansion of PV (Wyndham et al., 1968; Senay et al., 1976), an increased in total circulating protein mass (Senay et al., 1976) and improvement of subjective well-being (Bar-Or, 1980).

Physical training is known to be associated with an overall expansion of BV (Holmgren et al., 1960). Oscai et al.(1968) suggest that hemodilution may occur after prolonged strenuous exercise. In addition, to support the importance of training with respect to thermoregulatory responses, Gisolfi(1973) using $T_{re}$/performance time ratio as an index of heat tolerance, concluded that eight weeks of interval training in a cool environment (21 °C) produced only 50 % of the total adjustments achieved through heat acclimatization. Thus factors associated with exercise training may be important in the total heat acclimation response, but as Wyndham(1973) has noted, these do not account for the entire response.

Although both heat acclimatization and physical training result in an expansion of PV (Convertivo et al., 1983), Senay et al.(1976) regard this hypervolemia as the 'single most important mechanism responsible for acclimatization to heat'.

The plasma protein concentrations in the vascular space depend on protein synthesis and catabolism, their distribution between the vascular and extravascular spaces, and PV (Guyton, 1986). Thus hypervolemia in acclimatization and physical training could be explained by an osmotic expansion which manifests with the increased retention of sodium chloride (Robinson, 1963) as a result of the action of aldosterone (Davies et al., 1981). Another explanation is the increase of intravascular protein content brought about by less protein being lost through the cutaneous capillary, because training and acclimatization induced reduction in SBF (Roberts et al., 1977). Increases in protein synthesis and (or) decreases in protein degradation are further possibilities (Convertino et al., 1980).

The $T_{re}/\dot{V}O_2max$ relationship, has been suggested to predict development of heat stroke (Wyndham et al., 1953) and heat tolerance in hot environmental conditions (Avellini et al., 1982; Armstrong et al., 1987). Wyndham et al.(1953) found that even after a heat acclimatization programme, individuals who had higher probability to respond with heat stroke were the ones with the lower absolute $\dot{V}O_2max$ values and more specifically those with values less than 2 $\ell.min^{-1}$. Furthermore, Wyndham et al.(1953) and Lind(1963) showed that the relationship between $T_{re}$ and metabolic rate holds true only up to certain critical $T_{air}$ which varies according to different metabolic rates i.e. the higher the metabolic rate, the lower the critical $T_{air}$.

From this review of the literature, it would thus appear that distribution of body water and the ability to maintain BV may be the critical factors influencing one's ability to perform exercise in the heat. Homeostatically, it is logical that during prolonged exercise intensities (65-90%of $\dot{V}O_2max$) and under moderate environmental conditions, individuals are capable of regulating adequately their fluid replacement needs via the thirst mechanism and preventing significant dehydration levels.

## 2.4 PHYSIOLOGICAL AND METABOLIC RESPONSE TO EXERCISE IN CHILDREN

### 2.4.1 Introduction

Although a general physiological response to exercise is evidenced at all ages, there are growth or development related differences to exertion at different ages (Eriksson, 1972) and physiologic capacities have long been recognized to be dependent on body and system dimensions (Bar-Or, 1983). Although the factors to which the physiological responses of children to exercise can be attributed are strongly interrelated, this study has attempted to separate them.

### 2.4.2 Oxygen independent metabolism

Much of the training and performance of children involves activities of high intensity and short duration. The ability of children to sustain supra-maximal exercise for 30 seconds is markedly lower than that of young adults (Davies et al., 1972; Bar-Or, 1983). Even when adjusted for body mass, the power produced by an 8-year-old boy is estimated to be only 70% of that generated by 11-year-old boy (Davies et al., 1972).

Eriksson et al.(1973) found that the rate-limiting enzyme of glycolysis, phospofructokinase (PFK), is present in lower concentrations in children than in adults and attributed their lower glycolytic capacity to this finding. Furthermore, Bar-Or(1983) hypothesized that children evidenced less predisposition to resort to "anaerobic" metabolic pathways than adults due to their shorter "$O_2$ transients" i.e. the time needed to reach 50% of steady state $\dot{V}O_2$ (Macek and Vavra, 1980; Cumming, 1978; Bell et al., 1980).

Cumming, 1978) attributes the shorter "$O_2$ transients" of children to their smaller body sizes and the resulting shorter circulation time. Children also have higher muscle blood flow per unit of muscle mass when compared to adults at identical relative work loads (Koch, 1980). This is thought to facilitate the availability of $O_2$ to the working muscles. A shortcoming in the literature is the fact that myoglobin concentration, which, has an important function as an oxygen store during short spells of heavy muscular work (Astrand et al., 1960), has not yet been measured in children.

Lower rates of muscle (Eriksson, 1972) and blood lactate formation (Godfrey, 1974) after maximal work and during single bouts of submaximal exercise at the same relative intensity have been reported in children when compared to adults (Astrand, 1952; Eriksson et al., 1971; Macek and Vavra, 1971). It has been suggested that this can be accounted for by the smaller $O_2$ deficit at the onset of exercise (Eriksson, 1971; Macek and Vavra, 1980) and the lower concentrations of the enzyme, PFK, in the muscle of 11-13 year old boys (Eriksson et al., 1973).

One of the characteristic changes during child development is a decrease in the proportion of water in the fat-free body tissue and an increase in the solid matter (Diem and Lentner, 1975; Rudolph, 1991). The proportionate reduction in extracellular fluid space exceeds the increase in intracellular fluid space during growth (Diem and Lentner, 1975; Rudolph, 1991). Eriksson(1972) thus hypothesized that the lower concentration of muscle and blood lactate reported in children during maximal and submaximal exercise (Eriksson, 1972; Godfrey, 1974), is due to a greater level of dilution. In addition, these authors observed higher muscle, but lower blood lactate concentrations during heavy exercise following physical training in children. Eriksson et al.(1973) question whether a greater extraction of lactate by other tissues or different rates of production and utilization of lactate by the different fiber types in the working muscles may be responsible for this.

Since children are habitually active, the low blood and muscle lactate can, however, also be hypothesized to be a physiological adaptation to the intermittent nature of their daily activities. Further research is thus required to clarify the mechanisms.

### 2.4.3 Cardiovascular adaptation to exercise

Since $\dot{V}O_2$max is the product of maximal cardiac output and maximal arterial mixed venous oxygen difference (Astrand and Rodahl, 1977), these variables may limit $\dot{V}O_2$ max.

In untrained children, maximal HR is higher than that of untrained adults, but hemodynamic responses change with age and size and if HR is expressed as a function of BSA, relative HR is the same at peak exercise from 4-21 years of age (Riopel et al., 1979). Absolute stroke volume has been found to be lower in untrained children than in adults (Drinkwater et al., 1977; Bar-Or, 1983). However, Eriksson(1972) states that stroke volume must be considered relative to body dimension, and if stroke volume is expressed on the basis of height cubed, this index is higher in trained children than in untrained adults (Eriksson, 1972). Maximal arteriovenous $O_2$ difference $(a\text{-}v)O_2$ is similar in adults and children (Eriksson, 1972). These findings have thus led to the contention (Rowland, 1985) that the $\dot{V}O_2$max values of children are not different to those of adults when expressed in relative units.

Eriksson(1972) and Eriksson et al.(1973) found that the entire increase in $\dot{V}O_2$ max after training in 11-13 year old boys was due to the increase in maximal oxygen transport which they attributed to an increase in stroke volume due to increased contractility or filling of the left ventricle rather than to differences in heart size. These authors reported the failure of maximal $(a\text{-}v)O_2$ difference to contribute to the increase in $\dot{V}O_2$ max (Eriksson, 1972; and Eriksson et al., 1973). This was confirmed by Gunningham et al.(1984). However, in this study, the increase in $\dot{V}O_2$ was

attributed primarily to changes in stroke volume and secondarily to increases in the (a-v)$O_2$ difference. The insignificant increase in the (a-v)$O_2$ difference during exercise found in the study of Eriksson et al.(1973) was explained by the higher degree of physical fitness in the subjects of the study i.e. the fact that children are habitually more active than adults. Eriksson(1972) postulated that if physical training is begun as early as in prepuberty, a larger stroke volume (heart volume) may be attained in adulthood, than if training were initiated later in life.

Klissouras et al.(1973) studied interpair differences between twin pairs and monozygotic twins, aged 9-52 years and found that regardless of age, existing individual differences in the "functional adaptability" of men can be attributed to heredity. These researchers confirmed the previous findings (Klissouras, 1971; 1972) that $\dot{V}O_2max$ ($ml.Kg^{-1}.min^{-1}$) demonstrates interpair variability which is mainly explained by heredity.

There is an indication that PV expands during exercise of low intensity in children in contrast to the PV reduction observed in adults (Macek et al., 1976; Drinkwater et al., 1977). A possible factor to which the PV expansion observed ($\pm$ 5%) during the initial phase of exercise intensity at 40% of $\dot{V}O_2max$ in children (in the absence of fluid replacement) was attributed, was the low muscle lactate concentration in children (Macek et al., 1976). Support for this hypothesis is found in the studies of Jacobsson and Kjellmer(1964) and Bergström et al.(1971) who demonstrated an increase in extracellular and intracellular water content of muscles after heavy exercise due to the increased muscle lactate concentration. The hemodilution observed at moderate work loads, is hypothesized to be of benefit to the exercising child. Dilution of the by-products of metabolism, enhanced convective dissipation of metabolic heat, and less subjective and somatic circulatory strain, will aid the exercising child. However, further studies are needed to confirm this response. In particular, studies involving higher exercise intensities, are required.

Research dealing with cardiovascular and pulmonary factors, blood serum enzyme activities and other proteins levels has indicated that children can tolerate prolonged work as well as adults (Liesen et al., 1974; Macek et al., 1976; Haralambie et al., 1977). Interestingly, the adaptation of prepubertal boys to prolonged submaximal exercise at intensities which do not exceed 60% of $\dot{V}O_2$ max, is greater than that of both highly trained adults and untrained subjects (Macek et al., 1976). Corresponding to the above findings were the metabolic adaptations observed in children after six weeks of training on a bicycle ergometer which manifested with an increase of 30% in succinate dehehydrogenase (SDH) and an increase of 83% in PFK activities (Eriksson et al., 1973). Fiber distribution remained unchanged, but the oxidative capacity of both fiber types appeared to increase (Eriksson et al., 1973).

Koch(1974) calculated that local blood flow in the working muscle is 30% higher in children when compared to adults. Koch and Fransson(1980), however, showed a continuous tendency toward lower values in muscle blood flow with increasing age. In this longitudinal study, the higher muscle blood flow per unit of tissue, together with the lower cardiac output and slightly higher $(a\text{-}v)O_2$ difference in children aged 12 and 13 years has been attributed to children possessing a different blood flow distribution pattern to that of adults (Koch and Fransson, 1980). A similar theory has been suggested by Eriksson(1972). After the age of 15 years, when muscle mass had markedly increased in all the subjects, relative muscle blood flow tended to be even lower in children when compared to young men with average physical activity and fitness levels (Clausen and Lassen, 1971).

Factors to which the observed higher muscle blood flow in children as compared to adults (Allen, 1971; Koch, 1974; Koch, 1980) exercising at identical work loads have been attributed, include the higher habitual activity of children, the existence of less sympathetic stimulation and concomitantly lower peripheral resistance, the relatively larger diameter of large vessels in relation to the heart size (Brock, 1954) and the shorter circulation time due to the smaller bodies of children (Cumming, 1978). It

is well described that regular activity results in physiological adjustments which include an increased density and number of mitochondria (Howald, 1976), increased oxidative enzyme capacity (Koch, 1978) and decreased blood flow required by the skeletal muscle during exercise after training (Steinberg, 1971).

Children's higher muscle blood flow could be regarded as a metabolic advantage during exercise. The restoration of the internal equilibrium [carbon dioxide ($CO_2$), water, heat, oxygen] necessitates an adequate blood supply. An impaired blood flow limits the $O_2$ supply and the removal of heat and metabolites (Astrand and Rodahl, 1977). Furthermore, the ability of the muscle fibers to maintain a high degree of tension and an individual's subjective feeling of fatigue are highly dependent on the blood flow through the working muscle beds (Astrand and Rodahl, 1977).

Although children possess lower blood Hb concentration (Eriksson et al., 1971), Eriksson(1972) found children's cardiac output to be 1-2 $\ell$ less than that of young men, during submaximal work and during the same $\dot{V}O_2$ level (Ekblom et al., 1968). This may imply that a more important role is played by the $(a\text{-}v)O_2$ difference in children and its dependant mechanisms. The better utilization of the oxygen transported by the blood and consumption of any tissue is reached principally by a) the blood flow being better redistributed in skeletal muscle blood flow (Guyton, 1986; Astrand and Rodahl, 1977), b) its concentration affecting the quantity of $O_2$ transported in each 100ml of blood (Guyton, 1986), and c) the shift of the oxygen dissociation curve to enable the reduction of more oxyhemoglobin at a given pressure of oxygen i.e. a smaller percentage of saturation (Astrand and Rodahl, 1977). The shift in the dissociation curve in turn, is the result of the heat production by the working cell and the formation of $CO_2$ and lactate during exercise (Astrand and Rodahl, 1977).

Eriksson(1971) attributed the lower cardiac output values reported in children to a more efficient distribution of cardiac output to the working muscles in children, due

to a different pattern of blood flow distribution in which the proportion of blood flow directed to non-working organs is smaller in children, resulting in a lower cardiac output as compared to adults (Eriksson et al., 1973). This can also explain the greater $(a\text{-}v)O_2$ difference in children when compared to adults during submaximal exercise, in spite of lower Hb content (Eriksson, 1972). During maximal exercise however, Eriksson(1972) found $(a\text{-}v)O_2$ difference to be comparable to adult values and this was explained by the lower Hb concentration and accordingly lower maximal oxygen binding capacity. This was again confirmed by Eriksson and Koch(1973).

Whether the lower cardiac output and higher blood flow to the working muscles, compensates for the lower Hb concentration and greater $(a\text{-}v)O_2$ difference or for some other underlying mechanisms in children, is yet to be examined. What is, however, known is that, as is commonly reported in trained adults (Eichner, 1992), children's Hb concentration does not increase with conditioning, but total Hb rises (Bar-Or, 1983). The potential to assist the development of the cardiovascular system through training thus appears to exist in children.

### 2.4.4 Heart rate as an indicator of exercise intensity

Rowland(1985) supports the contention that training programs directed towards improving endurance capacity in children should apply the criteria of exercise intensity and duration which have been set for adults. The same suggestion was made by Ross and Gilbert(1985), and Mc Keag(1991) who supported that the position statement of the ACSM (1978) with respect to the development of fitness in adults, should apply to children. The following criteria are set in these position statements (ACSM, 1978):

1. Frequency of training: 3-5 days per week
2. Intensity of training: 60%-90% of max HR reserve or 50%-85% of $\dot{V}O_2max$

3. Duration of training 15-60min of continuous aerobic activity. Lower intensity for longer duration and high intensity for shorter duration.
4. Mode: activity which involves large muscle groups

The Committee of Sports Medicine(1988) classified tennis as a strenuous sport for children.

Research conducted by Drinkwater et al.(1977), has shown that the HR of trained child athletes are lower at rest and during all levels of exercise, and that trained children show a faster post-exertional recovery when compared to unconditioned children.

When HR is used to estimate exercise intensity, it should be kept in mind that the relation between HR and absolute $\dot{V}O_2$ depends, inter alia, on the muscle mass involved in the exercise. Thus, at a given submaximal $\dot{V}O_2$, the HR during arm exercise may be 10-50 bpm higher than during leg exercise. This difference increases as the $\dot{V}O_2$ increases (Asmussen and Hemingsen, 1958; Clausen et al., 1973). Furthermore, if HR is expressed in relative terms (per BSA), it reaches the same values for peak exercise in children ranging between the ages of 4-21 years (Riopel et al., 1979).

Exercise mode will also influence the relationship between HR and $\dot{V}O_2$. For example, during an identical time period in which the same amount of work is performed, the mean HR during intermittent exercise will be higher than during continuous exercise (Astrand et al., 1960). Environmental temperature has indirect effect on HR due to the increase body temperature and consequent "excitability" of the heart muscle (Guyton, 1986). Emotional stress can also effect the HR response to exercise (Saris, 1986) resulting in a greater rise in children than adults. Interestingly, it has been found that HR response to psychological challenge is higher in children displaying lower physical activity (Solcova et al., 1990).

### 2.4.5 Conclusions

It is apparent that from physiological and medical points of view, children are different from adults and from their peers in their response and tolerance to exercise due to the great range of variability in growth rates, anthropometric indices, gender and state of health.

Underling physiological mechanisms to exercise in children adhere to the same homeostatic regulatory responses as adults and are in some instances better [e.g. lower cardiac output and greater $(a\text{-}v)O_2$ difference in spite of lower Hb content in a given submaximal work (Eriksson et al., 1971; Eriksson, 1972; Ekblom et al., 1968), higher muscle blood flow (Allen, 1971; Koch, 1974), PV expansion response to exercise (Macek et al., 1976; Drinkwater et al., 1977)]. The effect of training status on exercise response, growth, body dimensions and health is, however, substantial in children (Bar-Or, 1983).

Children are capable of maximal and submaximal type of exercise. However, they are more suited to intermittent activities and less to "anaerobic" and strength training. Whether these characteristics can be explained as adjustments brought by the type of daily activities, or neuromuscular immaturity, or both, it is difficult to determine. A child's preference for activities of short duration could be explained by the lower absolute energy capacities, shorter attention span, the need for recreational stimuli, and lower socially induced motivation for long term exercise. It appears that prolonged exercise is monotonous and motivation to pursue this is poor in children.

One should, however, bear in mind that children are habitually more active than adults and may thus present with physiological adjustments that can lead to misinterpreted responses and comparisons between truly "inactive" untrained children and adults.

## 2.5 HEAT TOLERANCE AND HYDRATION IN CHILDREN

### 2.5.1 The statement

The AAP (1982; 1983), based on studies conducted by Astrand(1952), Wagner et al.(1972), Haymes et al.(1975; 1975), Drinkwater et al.(1977), and Drinkwater and Horvath(1979) suggest the following reasons, why children are less efficient thermoregulators than adults:

(1) Children have a greater BSA/mass ratio than do adults which induces a greater heat transfer between the environment and the body. This implies that when $T_{air}$ exceeds $\bar{T}_{sk}$, the rise in $T_{re}$ at a given heat index will be greater in children than in adults (Drinkwater et al., 1977; Wagner et al., 1972; Haymes et al., 1974).

(2) Children produce more metabolic heat per mass unit than do adults at a given workload (Asrand, P.O., 1952).

(3) Children have a lower sweating capacity than do adults, and show a reduced capacity to convey heat by blood from the body core to the skin (Haymes et al., 1975; Drinkwater et al., 1977; Wagner et al., 1972).

Children have thus been reported to possess less heat tolerance, lower SR and higher $T_{re}$ at a given work load when compared to adults. Based on this finding, the AAP (1982; 1983) supports that for a given level of dehydration, children are subject to a greater increase in $T_c$, even though sweat losses are not as great as in adults. Furthermore, children do not instinctively replenish the fluid lost during prolonged exercise (Bar-Or et al., 1980), and therefore, they are reported to be especially prone to dehydration (AAP, 1982; 1983).

More recent studies (Davies, 1981; Docherty et al., 1986; Mackova et al., 1984) have, however, reported heat tolerance and heat dissipation capacities in children which compare well with those of adults. Bar-Or(1989) attributes these contrasting findings to the varied environmental heat stress in the above studies (e.g., an ambient temperature of greater than 42 °C is needed for reduced heat tolerance response in children).

In addition, the following conditions have been associated with greater predisposition to heat illness in children: obesity, chronic heart failure, caloric malnutrition, febrile state, cystic fibrosis, gastrointestinal infection, anorexia nervosa, diabetes insipidus, diabetes mellitus, sweat insufficiency syndrome and mental deficiency (AAP, 1982). Furthermore, hypohydrated or insufficiently acclimatized children with a low fitness level, or patients with diarrhoea, fever, or children that have recently experienced heat illness and vomiting are also prone to heat illness (Bar-Or, 1983).

### 2.5.2 Morphologic and physiological characteristics of the exercising child as related to thermoregulation

#### 2.5.2.1 Metabolic heat production

Children produce more metabolic heat per mass-unit than adults when walking and running (Astrand, 1952). Furthermore, the total heat production of obese boys during exercise at different environmental temperatures, has been reported to be 40% greater than the heat production of lean boys at any environmental temperature (Haymes et al., 1975).

In addition, the greater metabolic heat produced by children is in accordance with the higher $O_2$ consumption per mass unit in children as compared to adults at a given speed of walking or running (Waters et al., 1983; Cavagna et al., 1983). This finding

has been attributed to childrens' uneconomical locomotor pattern and the neuromuscular immaturity of children as compared to adults (Astrand, 1952; Mac Dougall et al., 1979; Connoly, 1970). The reputed greater metabolic heat generated by children is a potential handicap when the climate is warm or hot and the exertion level is high (Bar-Or, 1983).

#### 2.5.2.2 BSA/mass ratio

Children have a greater BSA/mass ratio than adults, which induces a greater physical heat transfer to and from the environment. This exchange is lower for the obese child. The increased subcutaneous fat modifies the body contour of obese children, resulting in a decrease of the BSA/mass ratio in the obese child as compared to the lean child (Haymes et al., 1974).

The functional implication of the higher BSA/mass ratio of children is that, for any given temperature gradient between the skin and the ambient environment, heat transfer per mass unit through radiation and convection is greater in the lean child than it is in the adult (Bar-Or, 1980). This may be an advantage to the child whenever the ambient temperature is warm ($T_{air}$ not exceeding $\bar{T}_{sk}$) or whenever a high level of exercise is performed in a cold environment, since a child would be able to lose more heat per mass unit through evaporation, radiation and convection than in adult. However, when the $T_{air}$ is higher than the $\bar{T}_{sk}$, more heat would be lost through evaporation, but more heat gained through radiation and convection by a child than by an adult.

#### 2.5.2.3 Heat tolerance of the obese child

Obese children are at a disadvantage in the heat as compared to lean children for the following reasons:

(1) Fat is hydrophobic and hence the obese child has a lower total body water content per unit mass than the lean child. This implies that for a given percentage of dehydration, relative water loss in an obese child will be greater than in the lean child (Bar-Or, 1983).

(2) Due to higher body fat percentage, the obese child's relative aerobic capacity is usually low (Moccelin and Rutenfranz, 1968). Since the rise in $T_c$ during exercise is proportional to the relative metabolic rate (i.e. percentage of $\dot{V}O_2$ max), at a given metabolic rate, the obese child is exercising at a relatively higher percentage of his maximum $\dot{V}O_2$ as compared to the lean child (Haymes et al., 1975). Consequently the obese child will fatique earlier.

(3) The specific heat of fat tissue is lower than that of fat-free tissue with high water content (Bar-Or, 1983). Thus, an equal heat load per mass unit, will induce a greater temperature increase in the obese child as compared to the lean child (Bar-Or, 1983). This may explain why obese boys have the same heat storage as lean boys, but show a greater rise in $T_{re}$ (Haymes et al., 1975).

Bar-Or et al.(1969) and Haymes et al.(1974) have recently examined heat tolerance in obese women and children respectively. In the study of Haymes et al.(1974) heat tolerance tests were performed on exercising lean and heavy pre-pubertal girls and comparisons were made between these groups and compared to the adult female subjects examined in the study of Bar-Or et al.(1969). The results indicated that small differences exist between heavy and lean prepubertal girls. However, when these groups were compared to woman, the girls appeared to display more heat transfer through radiation and convection; more evaporative heat loss; and a higher heat production per unit mass at 21.1, 26.7 and 29.4 °C. However, pre-pubertal girls have a lower tolerance to exercise in the heat at 32 °C than women (Haymes et al., 1974).

A further investigation by Haymes et al.(1975) using obese and lean children as subjects, found that physiological responses (HR, $T_c$, $\bar{T}_{sk}$, $\dot{V}O_2$) of obese boys, in various environmental conditions, were similar to those of heavy prepubertal girls (Haymes et al., 1974), with the exception that the boys were more tolerant at 32 °C than the girls. When comparing the lean and obese prepubertal boys, obese boys showed greater increases in $T_{re}$, and tended to exhibit more physiological strain when exercising in the heat (Haymes et al., 1975).

Bar-Or(1983), found that the obese child, in spite of a lower density of heat activated sweat glands, sweats more than the non-obese child. In addition, at a given level of dehydration, obese children display a greater rise in $T_{re}$ than do lean children.

It would thus appear that these functional and morphological characteristics of exercising obese children are an important consideration during conditions of heat stress.

### 2.5.3 Heat tolerance in children

It is frequently discussed in the literature that due to the higher BSA/mass ratio, children have more efficient heat dissipation mechanisms when compared to adults (Bar-Or, 1983). Thus children are more efficient thermoregulators at identical metabolic heat production when $T_{air}$ is lower than $\bar{T}_{sk}$ (Bar-Or, 1980). This does, however, not apply when $T_{air}$ is greater than $\bar{T}_{sk}$ and the only means of heat dissipation is through evaporation of sweat. Firstly, it is theorized that the peripheral system (the lower sweating pattern of the exercising child) results in more rapid build-up of body heat (Araki et al., 1979; Haymes et al.,1974; 1975 and Wagner et al., 1972) and secondly, that the low stroke volume and the inability of

children to cope with the diversion of BV to the subcutaneous and muscle tissues, limits the ability of the cardiovascular system to cope with the combined stress of exercise and heat (Drinkwater et al., 1977; Drinkwater and Horvath, 1979)

Besides the important role of BSA and body fat composition of children in the thermoregulatory response to exercise, there is a need to examine the sweating patterns and cardiac output distribution of children during exercise and under heat stress, as these also contribute significantly to thermoregulation.

The hypothesis of a less effective sweat pattern in children is based mainly on the observations of Araki et al.(1979), in which pre-adolescents subjects were found to have significantly lower SR (despite normalization for a unit BSA) and higher $\bar{T}_{sk}$ than a comparative post-adolescent group. Th' difference was more apparent during exercise at a heavy work load. It is noteworthy to mention that in the study of Araki et al.(1979), the post-adolescent subjects consisted of physical education students, whereas the pre-adolescent group did not engage in physical exercise. It is well accepted that the physically conditioned individuals will sweat more profusely and earlier at any given work load than untrained ones (Baum et al., 1976; Nadel et al., 1974). The subjects in the study of Araki et al.(1979) rested on a chair for more than 40 min in a climatic champer ($T_{air}$ 29 °C) and were deprived of water for two hours before and during exercise. Although these conditions may be tolerable to adults, in children they may be uncomfortable, predisposing to an inferior physiological response to exercise and heat. In addition, the intensity of exercise in Araki et al's.(1979) study, was determined only using the average pulse during exercise. The three work loads (light, moderate, heavy) corresponded to HR of 110-120, 130-150 and 160-170 bpm respectively. Since SR is mainly related to the absolute exercise intensity (Gullestad, 1975; Saltin and Hermansen, 1966), a specific HR range will correspond to a lower absolute workload and hence lower SR in children. The researchers, however, interpreted the findings as indicative of an inadequate sweating pattern in children.

The conclusion that children perspire less than adults, was also reached by Haymes et al.(1974; 1975) and Wagner et al.(1972). In the above mentioned research (Haymes et al., 1974; 1975; Wagner et al., 1972), comparisons of SR per unit of BSA were based on identical speeds run on a treadmill. Children and adults worked under three different environmental conditions, but possessed different $\dot{V}O_2$ values. The thermoregulatory responses may therefore have been influenced by differences in exercise capacity and not only by age alone. It is well established that $T_c$ is related to the relative metabolic rate (Astrand, 1960; Saltin and Hermansen, 1966; Gullestad, 1975; Davies et al., 1976), whereas SR is highly related to absolute metabolic rate (Saltin and Hermansen, 1966). Since Haymes et al.(1974; 1975) and Wagner et al.(1972) utilized an absolute work load, meaningful comparisons in SR and thermoregulatory responses are expected to vary between the groups depending on the relative and absolute exercise intensities. According to Nielsen(1980), differences in temperature regulation attributed to age and sex can be explained by differences in $\dot{V}O_2max$. When Dill et al.(1977) conducted an experiment taking into account metabolic rates and body size of children and adults, comparisons between adults and children of this study, revealed that SR relative to BSA was independent of age.

Drinkwater and Horvath(1979), in an attempt to assess the relationship between heat tolerance and age, used 38 unacclimatized females aged between 12 and 62 years. The subjects worked at 30-35 %$\dot{V}O_2$ max under three environmental conditions 28, 35, and 48 °C. Using tolerance time at 48 °C as a criterion for determining the ability to exercise in a hot environment, Drinkwater and Horvath(1979) concluded that cardiovascular instability was the primary predictor for tolerance time as expressed by %HRmax. "Evaporative" SR (Drinkwater and Horvath, 1979) added significantly to tolerance time regardless of age, and initial $T_{re}$ as the last predictive factor contributed significantly to the multiple regression equation ($\psi$ = 686.85 - 1,339%HRmax + 0.166ESR - 15.365$T_{re}$, R=0.87) and accounted for 76% of the total variation in tolerance time. Final $T_{re}$ was not included in the regression equation due to the artificial limitation placed on its ultimate level. Furthermore, fluid intake

was not permitted and termination of the exercise session and consequently tolerance time was determined by a) $T_{re}$>39, b) HR>90% of HRmax, c) abnormal electrocardiograph and d) subjective signs of distress. However, a fixed $T_{re}$ could not be considered as a reliable criterion of thermoregulatory stress especially when the fitness status of the subjects was heterogenous. Surprisingly, final $T_{re}$ was used as a criterion to terminate the exercise session, although it was not included in the regression equation that predicted heat tolerance. It is possible that its elimination from the statistical analysis influenced the interpretation of the results.

With regard to the HR criterion, maximum HR varies among children from 180-234 bpm (Cumming and Hnatiuk, 1980). Thus a set percentage of maximum HR (%HRmax) lacks accuracy. The problem becomes more complex when multiple regression analysis is performed to predict for example, tolerance time from evaporative SR, changes in PV and %HRmax, since physiologically these variables interrelated and a unit drop in osmolality, for example is known to influence HR, $T_c$ and consequently SR and SBF response (Fortney et al., 1984). Thus, tolerance time is not solely explained by the rise of PV, but rather by a combination of the above-mentioned factors. Heat tolerance in children should also be considered in view of the motivation of the child to complete such uninteresting protocols in unrealistic high environmental temperature of 48 °C.

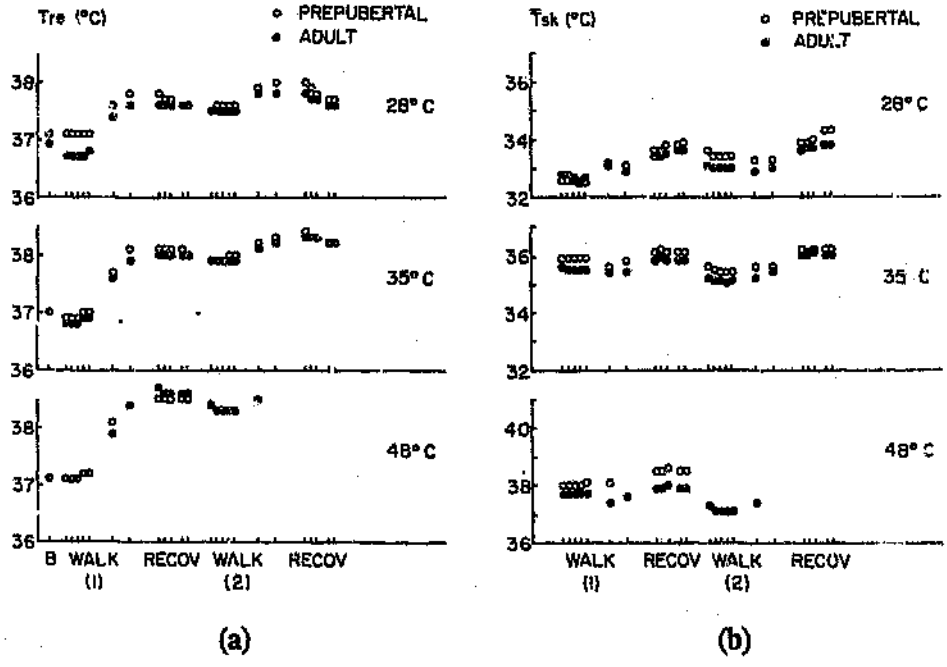
Cardiovascular instability was the reason given for the lower heat tolerance of children as compared to woman in the study of Drinkwater et al.(1977). The exercise protocol and environmental conditions utilized in this study were identical to those described in the work of Drinkwater and Horvath(1979). However, in the study of Drinkwater et al.(1977) the subjects (prepubertal and college women) were matched for maximal aerobic power ($\dot{V}O_2max$), to enable the researchers to control and explain the physiological responses to exercise in the heat more objectively (i.e. only on the basis of thermoregulatory response and not on differences in exercise metabolic rates). This study found that a lower stroke volume and an inability to shift fluid into the

cardiovascular space were the main age related differences with respect to heat tolerance time. The lower heat tolerance in children was explained by the larger BSA/mass ratio of children. The researchers found that when expressing blood and stroke volume relative to BSA ($ml.m^{-2}$), children have lower blood and stroke volume than adults. Hence, during exercise and heat stress, the children will require a larger proportion of BV to be diverted in order to maintain adequate peripheral blood flow and to serve the heat dissipation mechanisms, when compared to adults (Drinkwater et al., 1977). It has been found that as much as 8 $\ell.min^{-1}$ of cardiac output may be diverted to the subcutaneous tissues during prolonged direct heating of the entire body in adults (Rowell, 1974).

However, it is well documented that stroke volume ($ml.beat^{-1}$) reaches its maximum by the time the cardiac output has increased to only half of its maximum during high intensity exercise (Astrand and Rodahl, 1977; Guyton, 1986). Thus the question to be asked is whether children who are capable of sustaining a high HR in the face of a "lesser venous return" during exercise, will be as able to tolerate heat stress as well as adults. In view of a "lesser venous return" in children, one has to consider the reports that children have faster circulation times than adults (Cumming, 1978), different blood flow distribution patterns (Koch, 1980), and expansion of PV during exercise (Macek et al., 1976). The latter was also confirmed in the study of Drinkwater et al.(1977), in which the prepubertal girls responded with PV expansion ($\pm$ 4-5%, with marked increase in TPP) during exercise in all environments, whereas the college women responded with hemoconcentration (no marked shift of TPP was observed). It is thus possible that, in contrast to the findings of Drinkwater et al.(1977), the children's higher HR excitability (as indicated by their maximum HR), lower sympathetic activity (Lehmann et al., 1981) (as indicated by a low peripheral resistance) and the hemodilution response (as indicated by the PV expansion) during exercise may enhance cardiac output, facilitate a higher venous return and result in a more effective heat convection transfer from body core to periphery and thus prevent decrements in performance, when compared to adults. This question does, however, require further investigation.

A contradiction in the study of Drinkwater et al.(1977) was that although the groups were matched for aerobic power and exercised at the same relative work load (i.e. percentage of $\dot{V}O_2$ max), the SR responses ($g.m^{-2}.min^{-1}$) did not differ significantly between environmental conditions. If one calculates the absolute 'aerobic power' of the subjects, it becomes apparent that the prepubertal girls worked at approximately 0.5 $\ell.min^{-1}$ and the college women at 0.9 $\ell.min^{-1}$. However, this absolute difference in 'aerobic power' during exercise should have been reflected in SR, if SR is determined by the absolute exercise intensity (Saltin and Hermansen, 1966; Gullestad, 1975). This did not occur and was not identified as a shortcoming by the authors of the study.

It is worthwhile to reprint from Drinkwater et al.(1977) study, two figures illustrating the $\bar{T}_{sk}$ and $T_c$ responses of the two groups under the three environmental conditions.




(a) (b)

**Fig. 2.2.(a)** *Rectal temperatures (°C) of prepubertal (○) and adult (●) females during basal period (B); min 1-5, 20-25, and 40-45 of each work period; and during min 1-3, 9, and 10 of recovery periods in three environmental conditions. Reprinted with permission from Drinkwater et al.(1977).*

**Fig. 2.2.(b)** *Mean skin temperature (°C) of prepubertal (○) and adult (●) females during min 1-5, 20-25, and 40-45 of each work period; and during min 1-3, 9, and 10 of each recovery period in three environmental conditions. Average basal $\bar{T}_{sk}$ for prepubertal females was 33.4 °C; for women, 33.7 °C. Reprinted with permission from Drinkwater et al.(1977).*

From figures 2.2.(a) and 2.2.(b), it can be seen that the major differention in $\bar{T}_{sk}$ and $T_c$ responses between the girls and the women at the three environmental conditions was the relatively higher $\bar{T}_{sk}$ of prepubertal girls at 48 °C. This is an indication of high SBF response, occurring in order to deliver the heat necessary for the evaporation of sweat (Gisolfi and Wenger, 1984), and associated with active vasodilation (AVD) (Blair et al., 1960). Based on this observation it is logical to hypothesize that AVD occurs earlier in children than in adults. Supporting evidence for this hypothesis is provided by the fact that effector thermoregulatory responses (SBF, sweat) which serve the dissipation of heat, are proportional to changes in $T_c$ and $\bar{T}_{sk}$ (Stolwick and Hardy, 1977). Furthermore, since $\bar{T}_{sk}$ is related to body mass, the relative contribution of $\bar{T}_{sk}$ in initiating effector responses is greater in smaller species (Roberts et al., 1974).

Referring to subsection 2.3.2, as the load error quantity ($T_{wa}$-$T_{set}$) increases during exercise due to a rise in $T_{wa}$ ($T_c$+$\bar{T}_{sk}$), heat dissipating responses (e.g., SBF, sweat) are recruited at different stages. At the stage of the AVD response, large increases in SBF occur in the limbs (Blair et al., 1960). It would thus appear that the observed higher $\bar{T}_{sk}$ in children is accompanied by an earlier onset of AVD, due to the greater input of $\bar{T}_{sk}$ in the load error for the initiation of this particular effector response. Further support for this hypothesis is provided by the fact that AVD is needed for an intact sympathetic activity. If children respond with less sympathetic activity during exercise than adults (Lehmann et al., 1981), the fact that at 48 °C in the study of Drinkwater et al.(1977), the girls presented with a 13,7% increase in arm volume as compared to a 5,4% increase in the women tested, further confirms the hypothesis of an earlier and higher subcutaneous blood flow. This is confirmed by Falk et al.(1992a), who also reported significantly higher forearm blood flow among pre-pubertal boys as compared to late-pubertal boys during exercise in the heat. Although, the underlying mechanisms of AVD have not yet been determined (Guyton, 1986), AVD should be considered in conduction with a shift of a considerable amount of cardiac output (8 $\ell.min^{-1}$) to the subcutaneous tissues as has been observed in adults (Rowell, 1974). This would detrimentally effect the continuation of exercise due to

an increased demand of blood flow to the myocardium, skeletal muscles and lungs, which may not be met and this may have caused the premature termination of exercise. A possibly narrower prescriptive zone of $T_{re}$ and a lower sympathetic activity in children as compared to adults, could be suggested to explain the earlier and more intense secondary AVD in children when compared to adults. The higher $\bar{T}_{sk}$ and SBF between the two groups in the study of Drinkwater et al.(1977), at the same relative work loads, appears to support the above hypothesis.

Falk et al.(1992a) compared thermoregulory response of pre-pubertal, mid-pubertal and late-pubertal boys exercised (50% $\dot{V}O_2max$) in the heat (41-43 °C, humidity: 18-22), and concluded that the transition from a less effective thermoregulatory response of children to more effective response of that in adults does not occur during puberty. Although the late-pubertal boys exhibited significantly higher SR ($ml.min^{-1}.m^{-2}$) compared to pre-pubertal boys, the rate of heat storage was significantly higher in late-pubertal boys than in pre-pubertal boys. They hypothesized that a less effective sweating pattern in late-pubertal boys (presenting with an apparent "dripping", rather than "evaporation" of sweat), explains the significantly higher heat storage in late-pubertal boys as compared to a more effective sweating pattern in pre-pubertal boys (Falk et al., 1992a; 1992b). However, this hypothesis contrasts to the existing theory of increased heat storage of children during exercise in the heat, due to lower sweat rate and thus lower evaporative cooling than adults. Furtheremore, in the study of Falk et al.(1992a; 1992b), the higher SR of late-pubertal boys due to higher absolute work output was not taken into account, which may explain the higher SR calculated in late-pubertal than in pre-pubertal boys. With regard to the greater heat storage of the late-pubertal boys in the study of Falk et al.(1992), the importance of $\bar{T}_{sk}$ in the calculation of heat storage ($0.8T_{re} + 0.2\bar{T}_{sk}$), may be higher in children than in adults. This assumption is based on the fact that under the same relative work loads in the heat (Drinkwater et al., 1977; Falk et al., 1992), SBF (which is mediating from $\bar{T}_{sk}$) is positively related to age from pre-pubertal till adulthood.

A clear-cut answer the control mechanisms of thermoregulatory responses of children during exercise and heat stress has therefore not been established. Assumptions such as "children require a greater increase in $T_c$ to start perspiring, which suggests a higher hypothalamic threshold" (Bar-Or, 1983) or "tolerance time of children and young adolescents exercising in the heat are shorter than that of young adults" (Haymes, 1974; 1975; Wagner, 1972; Drinkwater, 1977; Drinkwater and Horvath, 1979), need to be further researched under more carefully controlled conditions. As noted in subsection 2.3.2, conditioning and heat acclimatization will effect time and (intensity) of SR response. Cognizance should be taken of the fact that beside $T_c$, $\bar{T}_{sk}$ contributes significantly to the sweat response during exercise. In the study of thermoregulatory responses the subject's conditioning and acclimatization status should be clearly outlined together with the $\dot{V}O_2$ max assessment data, to better understand the role and the background of $\dot{V}O_2$max. In addition, habitual activity and neuromuscular maturation in children may be a better predictor of heat tolerance to exercise in children.

Lastly, if children respond to exercise with a) less Na in sweat b) increase in BV (hypervolemia) and c) higher SBF than adults at a given work load (Koch, 1980; Drinkwater et al., 1977; Falk et al., 1992), which are all result in lower heat storage for a given $T_c$ (Sawka 1992) and are adaptive physiological responses of training and heat acclimatization procedure (Wyndham et al., 1968; Senay et al., 1976; Bar-Or, 1983), the theory that "children are less efficient thermoregulators than adults" is to be questioned.

### 2.5.4 Water requirements

Few studies have investigated the fluid replacement needs in children during exercise and under heat stress. Despite the previously discussed relationship between fluid status and thermoregulation, thermoregulatory responses have been investigated in the

absence of fluid replacement in the exercise protocols of Drinkwater et al.(1977) and Drinkwater and Horvath(1979). Since PV changes are related to thermoregulation (PV provides the precursor of the secreted sweat which enables insensible heat loss) (Fortney et al., 1984), fluid replacement can alter this relationship. Moreover, the potential of plasma volume expansion in children may also modify SBF and sweat responses. The disproportional distribution of extracellular and intracellular fluid volume in children, together with their BV expansion in exercise will further facilitate the convective transfer of heat from the body to surface area and enhance the heat dissipation mechanisms.

Water is necessary to support metabolism as it provides the medium for biochemical reactions and body solutes (Ganong, 1989). The water requirements of the body are determined by the amount of heat produced and by the concentrations of solutes in the body fluids (Diem and Lentner, 1975). It has been estimated that the daily requirements in $ml.Kg^{-1}$ is 70-85 for a 10 year-old child possessing a body mass of 29 Kg, while that for adult possessing a body mass of 70 Kg, is 21-43 $ml.Kg^{-1}$ (Diem and Lentner, 1975). The children's higher water requirements are probably due to a) the greater metabolic rate per Kg body mass, with result in a higher metabolic water loss and b) the fact that children's high levels of protein synthesis require that considerable amounts of water been processed.

The AAP (1982; 1983) recommends that prior to prolonged exercise, children should be fully hydrated and that during the activity, fluid replacement should be 150ml of cold tap water each 30 min for a child weighing 40 Kg, even when the child is not thirsty (AAP, 1982; 1983). Costill and Saltin(1974a) support the theory that cold water is emptied faster from the stomach than warm drink and is more palatable. This is, however, presently under question and requires further investigation.

The enforcement of drinking habits by the AAP (1982; 1983) stems from the findings of Bar-Or et al.(1980). These researchers found that children are voluntarily

dehydrated during exercise. They define voluntary dehydration as "a state of fluid deficit even though drinking is provided ad libitum". However, in the work of Bar-Or et al.(1980) the formula used to calculate percentage dehydration was based on body mass loss only and metabolic fuel utilised, metabolic water produced by mitochondrial oxidation and the considerable water volume released from glycogen breakdown was not included. The findings may thus not have been indicative of "dehydration". Consequently the contention of the AAP (1982; 1983) that, for a given level of dehydration, children are subject to greater increase in $T_{re}$ than are adults (Bar-Or et al.(1980) requires re-examination.

Furthermore, in the study of Bar-Or et al.(1980), PV changes were not measured. Since children, even in the absence of fluid replacement, respond with PV expansion to exercise (Drinkwater et al., 1977; Macek et al., 1976), the assumption can be made that stimulation of the thirst mechanism in children would be weaker if this mechanism is triggered when plasma electrolytes become concentrated (Senay, 1979; Guyton, 1986). Another factor is the unknown effect of direct transference of solar heat onto the skin and the possible stimulation of the thirst mechanism.

As the study of Bar-Or et al.(1980) appears to be the only work investigating the fluid replacement needs of children during exercise published to date and a number of questions still exist, further research is needed to provide definite answers. For example, it is well accepted that children possess a higher total body water proportion when compared to adults (Diem and Lentner, 1975; Rudolph, 1991). A given percentage of body mass loss will thus reflect a lower percentage of dehydration in children than in adults. Whether children's higher extracellular fluid proportion in relation to the lower intracellular fluid proportion plays a role during exercise and the importance of such a role in percent dehydration is another question requiring investigation.

2.5.6 Heat acclimatization in children

Research investigating the ability of children to acclimatize to exercise in the heat has shown that :

a) children do acclimatize to exercise in the heat (Wagner et al., 1972)

b) their rate of acclimatization is physiologically slower (Inbar, 1978), but "subjectively" faster, when compared to adults (Bar-Or and Inbar, 1977).

c) their level of acclimatization is somewhat lower than that of adults (Bar-Or, 1980).

d) they can acclimatize by exercising in a neutral environment or by sitting in a hot environment which is not always applicable to adults (Inbar et al., 1978).

e) acclimatization of boys to work in dry heat is less effective than that of older adolescents or adults (Wagner et al., 1972).

Shvartz et al.(1973) and Inbar et al.(1981) propose that the degree of heat acclimatization is a function of heat stored in the body during each session of a given acclimatization procedure. More specifically, Inbar et al.(1981) studied three acclimatization protocols on children (active acclimatization, exercise in a neutral climate, and passive acclimatization). These researchers found that initial heat storage caused by the three different acclimatization protocols varied, being highest in the active procedure, followed by the passive and then the exercise in neutral climate exposures. Therefore conditioning in young children in a neutral environment produces as high, if not higher adaptive responses than those occuring in adults subjected to exercise and the heat acclimatization procedure (Inbar et al.,

1981). It is believed (Buskirk et al., 1969) that children's morphological characteristics, in particular, the higher BSA/mass ratio plays a conducive role in the passive heat acclimatization procedure. The greater thermal load influx from the environment might be sufficient to result in more complete heat acclimatization in children than in adults.

The perception of a faster rate of acclimatization in children, together with the "underestimated" perception of exercise stress (Bar-Or and Inbar, 1977), might be regarded as an advantage to children exercising in the heat. However, it may also signify a hazard: the insufficiently acclimatized adult is reluctant to exert themselves in the heat, whereas the child may have a false sense of confidence and overexert himself in view of marked heat strain (Bar-Or, 1980).

## 2.6 SELECTED INSIGHTS INTO COMPETITIVE STRESS IN THE ATHLETE

### 2.6.2 Competitive anxiety

Spielberger(1966) defines trait anxiety as the individual's predisposition to perceiving competitive situations as threatening and responding to these situations with subjective feelings of apprehension or tension. These feelings are accompanied by the activation or arousal of the autonomic nervous system (Spielberger, 1966).

More recently, Martens et al.(1990) hypothesized that each individuals' A-trait personality disposition, in the presence of uncertainty of the outcome and importance attached the outcome, will respond with a certain level of A-state (threat) in a given competitive situation (fig. 2.3)

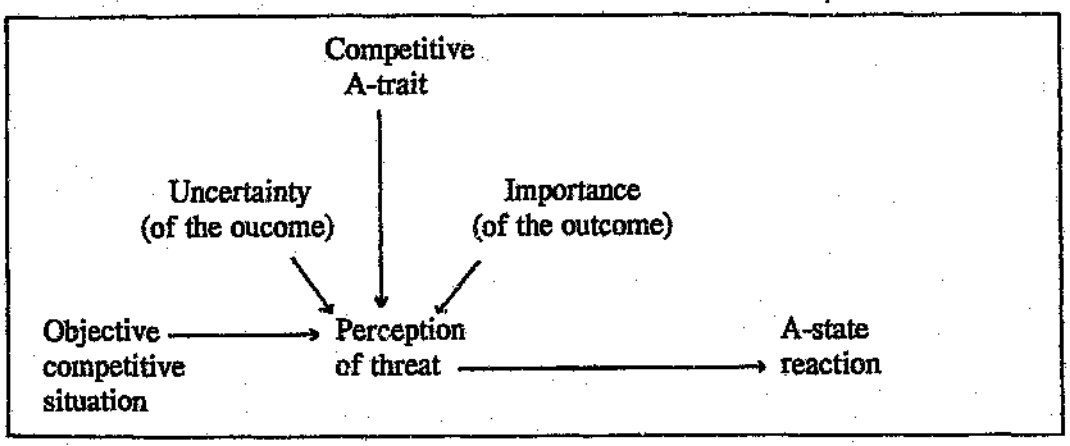



**Fig. 2.3** ***Interaction model for the competitive stress process. Adjusted from Martens et al.(1990).***

The greater the uncertainty, importance of the outcome and A-trait tendency to perceive competitive situations as threatening, the greater the threat in the objective competitive situation (Martens et al., 1990).

Martens et al.(1990) developed a sport specific questionnaire measure of competitive state anxiety, the Competitive State Anxiety Inventory-2 (CSAI-2). This measures cognitive anxiety, somatic anxiety and self-confidence. Cognitive anxiety and self confidence refer to the athlete's feelings of apprehension, which are related to performance expectancies (Martens et al., 1990) and previous competitive experiences (Gould et al., 1984; Feltz, 1984). Somatic anxiety generally reflects physiological condition or reflexive response associated with competitive stress (Martens et al., 1990).

Coping with competitive stress is a critical mediating factor used to control emotional arousal (Lazarus and Folkman, 1984). Performance expectancies are mediated by factors such as perceived importance of the competition (Sanderson and Ashton, 1981), prior knowledge of the opponent's ability (Wandlizak et al., 1982), past performance (Martens et al., 1990) and other external factors such as weather conditions (Martens et al., 1990). If an athlete perceives that external factors are

beyond his control, uncertainty about the performance outcome will increase. Festinger(1954) and Martens et al.(1990) believe that the primary means for reducing competitive stress is to obtain information that transforms uncertainty to certainty.

An alternative to theories of athletic performance based on stress, has been proposed by Hanin(1986). This theory utilizes an individual approach to anxiety and performance and supports the hypothesis that each athlete has an optimal anxiety level prior to competition. This optimal anxiety level may be low, moderate, or high, depending on the individual. Successful performance occurs when pre-competition anxiety is near optimum or falls within a Zone of Optimal Function (ZOF). When pre-competition anxiety falls outside the ZOF (i.e. either higher or lower), performance worsens. Although this theory is similar to inverted-U hypothesis (Yerkes and Dobson, 1908) in that there is an anxiety range, in which performance is optimized, the primary difference lies in the fact that the inverted - U hypothesis predicts that athletes perform best when anxiety level is moderate (Burton, 1988; Sonstroem and Bernardo, 1982; Weinberg and Genuchi, 1980), whereas ZOF demonstrates that the optimal anxiety level of some athletes, regardless of the sport or task within a sport, can be low or high (Hanin, 1986).

### 2.6.2 Psychophysiological responses to competitive stress

Borkovec et al.(1977) proposed three levels at which the individuals can respond to stress, namely behavioral, physiological and psychological. The tendency of an individual to become anxious in sport, has promoted investigators to attempt to identify sources of anxiety, in order to learn how an individual perceives these sources; and how these sources result in physiological, psychological and behavioral changes in an individual.

The physiological responses of the body to mental and physical stress are directly related to one another. Mental stress results in an enhanced sympathetic stimulation, which is known to have numerous physical effects some of which may be detrimental to his/her well being. The sympathetic and parasympathetic system of the autonomic nervous system activates the smooth-muscle (viscera) directly and the skeletal muscles indirectly (Guyton, 1986). Thus, the autonomic nervous system regulates reflexively the increase and decrease in the tension levels in the muscles and glands (Jordaan et al., 1975). Physiological measurements which serve as indices of physiological stress include HR, breathing rate, muscle tension, endocrine secretions, electric skin resistance (Jordaan et al., 1975; Samuel et al., 1978). However, these psychophysiological indications are only applicable within certain limits (Jordaan et al., 1975; Lade, 1969) and are based on the principle of homeostasis. Anxiety is known to alter gastric emptying rate (Murray, 1987) and to increase SR and HR (Martens, 1987). Furthermore, HR response to psychological challenge has been reported to be higher in children displaying lower physical activity (Solcova et al., 1990). Crews and Landers(1987), in a meta-analysis of 34 studies, supported this finding of Solcova et al.(1990) by revealing that subjects who were aerobically fit, demonstrate decreased psychosocial stress responses when compared to untrained groups.

The overall emotional reactions to competitive sport are, however, no greater than those towards other children's activities including academic tests and music competitions (Simon and Martens, 1979). The two most anxiety-provoking sports were found to be wrestling and gymnastics, both individual sports (Simon and Martens, 1979). Measurements of anxiety-performance relationships have been performed by sports psychologists in swimming (Burton, 1988); volleyball (Crocker, 1988); wrestling (Gould, 1984); basketball (Sonstroem and Bernardo, 1982); and golf (Weinberg, 1978). Research has revealed that individual sports impose greater stress upon athletes than team sports (Griffin, 1972).

## 2.7 CONCLUSION

The limited research addressing the fluid replacement needs in children highlights the need for further investigation into this area. However, in view of the considerable variability in physiological response to exercise between children, accurate documentation of subject selection, training intensity, environmental conditions and method of fluid replacement determination, is required in order to make both objective comparisons between studies and interpretations of results, feasible.

# CHAPTER THREE

# MATERIALS AND METHODS

3.1 RESEARCH DESIGN AND PROTOCOL

3.2 FIELD TESTING

3.3 LABORATORY ANALYSIS

3.4 FORMULAS UTILIZED IN THE DATA CALCULATIONS

3.5 STATISTICAL ANALYSIS OF DATA

## 3.1 Research design and protocol

The research design of the investigation can be classified as a single group experimental design. This involved utilising the same subjects in pre- and post-match testing and obtaining their pre-and post-play data. If the pre-match data were significantly different to the post-match data ($p < 0.05$), the differences were assumed to be due to the experimental factor - actual play situation.

The criteria used to separate the subjects into groups were tennis playing ability and age. Group one consisted of eight Southern Transvaal tennis players ranging between the ages of 12-13 [$\bar{X}$= 12.2($\pm$0.46) yrs]. Group two consisted of eight school tennis players, with average tennis ability and ranging between the ages of 11-12 [$\bar{X}$= 11.6($\pm$0.51) yrs]. Group three consisted of eight tennis players ranging between the ages of 14-15 [$\bar{X}$= 14.5($\pm$0.53) yrs], and with above average tennis performance level.

Prior to performing the study, clearance was obtained from the Committee for Research on Human Subjects of the University of the Witwatersrand. Informed consent

was obtained from both children and parents before participation in the investigation (Appendix $A_1$).

Physiological, biochemical and psychological variables before, during and after 85-90min of tennis play on tennis courts were assessed during the mid-summer season of 1991/1992. Matches were simulated for group two and three, whereas measurements for group one took place during an interprovincial Round Robin Tournament. The measurement stages of this investigation and time schedule of the study are provided diagrammatically in fig 3.1

Test results, comments and advice were supplied to the 24 participants in written form two months after completion of the data collection (Appendix B).

## 3.2 Field testing

Pre-match recordings and measurements included:

### 3.2.1 Questionnaires:

#### 3.2.1.1 Anxiety classification of subjects

The Competitive State Anxiety Inventory-2 (CSAI-2) developed by Martens et al.(1990) was administered to group three (13-15 year-old), and the Sport Competition Anxiety Test for children (SCAT-C), (Martens et al., 1977) was administered to all groups in order to assess the trait anxiety of the children (a copy of the SCAT-C questionnaire is included in Appendix $A_2$). The (CSAI-2) questionnaire comprises 27-items which, when scored, result in cognitive anxiety, somatic anxiety, and confidence subscale scores ranging from a low of 9 to a high of 36. The lowest score which can be achieved in (SCAT-C) is 10 and the highest 30. Children with trait scores of

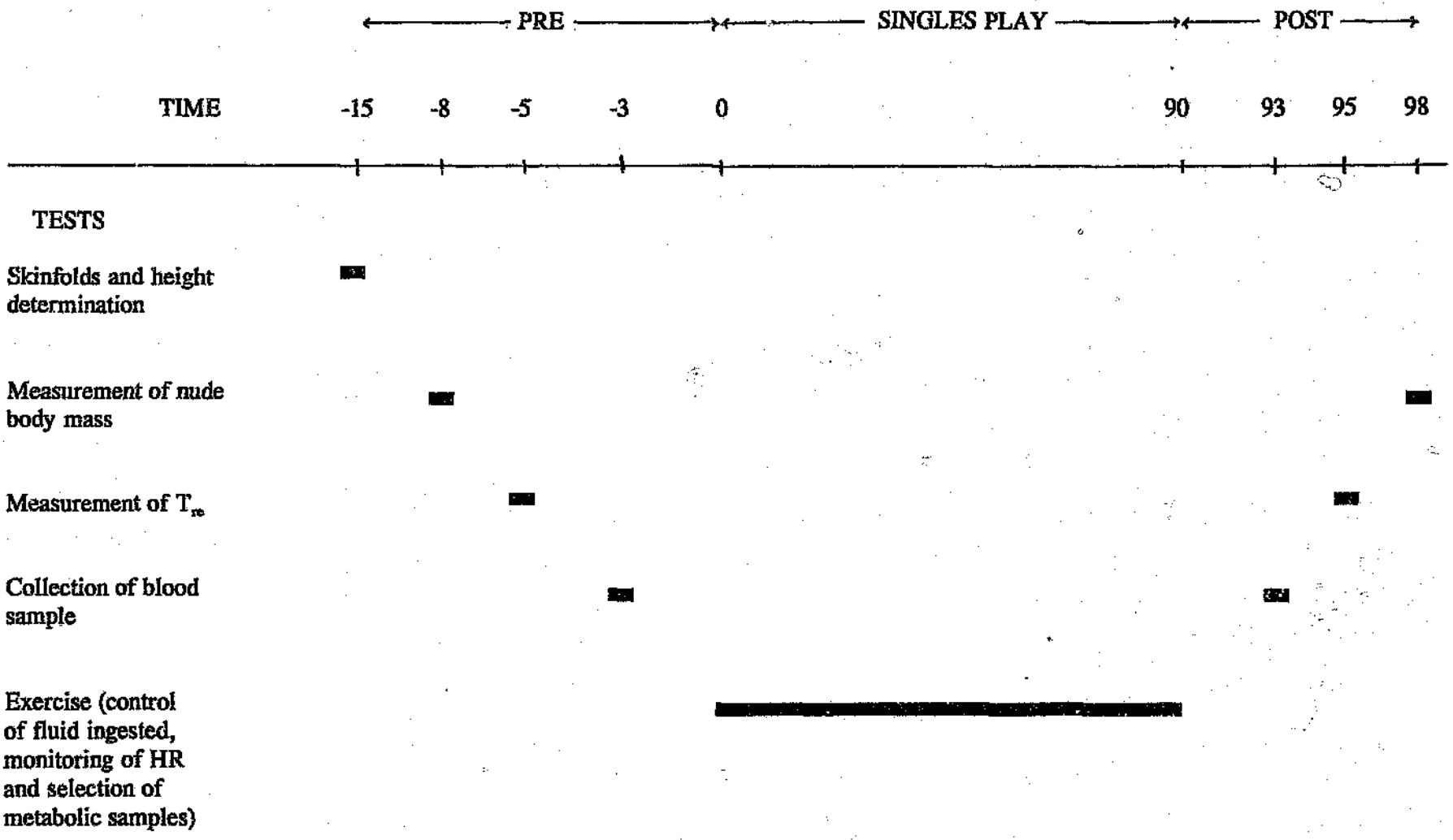



**Fig. 3.1.** ***Graphical representation of testing measurements and sequences.***
Time denotes minutes relative to the commencement of play. Pre- and post- denotes pre-match and post-match measurements respectively.

greater or equal to 25 (upper 25 percentile) were classified as possessing high trait anxiety while subjects with scores of less than or equal to 15 (lower 25 percentile) were classified as possessing low trait anxiety. The relationship between trait and state anxiety in the competitive situation was also determined in group three.

3.2.1.2 Degree of acclimatization to the heat

Degree of heat acclimatization was assessed by means of a questionnaire developed by the researcher, which elicited information regarding the most significant contributors to the acclimatization procedure i.e. exercise intensity, duration, type and time spent exercising in the heat during the month before initiation of the study.

3.2.1.3 Parental questionnaire

The parental questionnaire was administered with the aim of obtaining information about the health status of the subjects, type of reward system used by parents after a child's victory in a tournament, and symptoms of anxiety prior to participation in a tournament. A copy of this questionnaire is included in Appendix $A_3$.

3.2.2 Morphological measurements

The anthropometric measurements were taken by an experienced graduate assistant. Standardised calibrated anthropometric equipment was used to measure height and skin-folds of the subjects.

3.2.2.1 Measurement of height

The subject's height was measured using the portable stadio-meter (Holtain Ltd, Crymyth, UK) and was recorded to the nearest millimetre. Subjects were measured while standing bare-foot, with the heels in contact with each other and the ground, and the upper back and rear of the head in contact with the vertical section of the stadio-meter.

3.2.2.2 Skin-fold measurements

The subject's tricep, sub-scapular, supra-iliac, abdominal, front thigh, and medial calf skinfolds were measured using a Harpenden skinfold thicknesses (Holtain Ltd, Crymyth, UK) with a jaw pressure of 10 grams per square millimetre. The sum of the thickness of the six skin folds were utilized to determine the subject's percentage body fat (Yuhasz, 1974). The fold of skin was picked up between the thumb and index finger, and the calliper jaws were placed one centimetre from the fingers at a depth approximately equal to the thickness of the fold. The skin fold was held throughout the measurement and once the calliper indicator had become steady, a reading to the nearest 0,1 mm was taken (De Villiers and Tobias, 1974).

3.2.2.3 Measurement of nude body mass

The subject's nude body mass was determined before and after tennis matches, using the Seca 770 Alpha Electronic Digital Display Scale (Protea Medical, Johannesburg). The scale has a slip resistant mat, electronic digital display and is operated by a light touch of the foot on the contact bar. The subjects were requested to urinate before the initial body mass measurement and before the final measurement after the match. Volume of urine voided by the player was obtained with the use of a measuring cylinder.

3.2.3 Physiological measurements

Each member of the research team was responsible for a specific physiological assessment. All equipment was regularly calibrated and particular care was taken to ensure reliability and validity and to eliminate measurement and recording errors.

3.2.3.1 $T_{re}$

Pre- and post match $T_{re}$ was taken within 3 and 5 min of the subjects starting and completing the match respectively, using standard clinical sterilised indwelling rectal

thermometers. The rectal thermometer were inserted 5 cm into the rectum and left in place for a minimum of 3 min. The measurement followed immediately after the blood sampling. This sequence was selected due to the lesser sensitivity of $T_{re}$ changes as compared to the potential changes of BV, especially after the match.

During the match the following physiological recordings and measurements were recorded:

3.2.3.2 Measurement of fluid ingested by the player

Ad libitum liquid intake was allowed during the competition. The subjects were allowed to drink only water. Labelled plastic cups were available for each player to drink water during the playing breaks of the match. The volume of ingested water was determined by measuring the water intake by the subjects during the match to the nearest $\pm$10ml.

3.2.3.3 Measurement of HR response during tennis match

Intensity of exercise was indirectly measured by monitoring the HR response during tennis play. The Karnoven Method (KM) was used in order to set the target HR range (THRR) values of the subjects (Karnoven et al., 1957). With the KM, age and its effect on the resting and maximum HR were taken into account: the THR (HR training range for cardiorespiratory improvements) was determined by taking a chosen percentage of the difference between the maximal and resting HR values and then added to the resting HR value and expressing the THR as percentage of HRmax. This method of calculating the THR corrects for the nonzero value of resting HR. Resting and HRmax of the subjects groups were obtained from tables published by Siegel(1988).

The Sport Tester PE 300 (Unilife) was used to record the HRR by means of radiotelemetry and the PE 3000 (Polar) was used for the continuous measurement of HR response in a subset of 13 of the subjects. Transmitters were attached to the chest

by means of an electrode belt, and the receiver was worn in the form of a watch on the subjects' wrist. The duration of the periods during which the subject was within certain predetermined THRR during the match was recorded. The instantaneous HR at the onset and conclusion of the 60 sec[illegible]d during which the sample of expired air was collected, was averaged and related to $\dot{V}O_2$ during the 60 second period. Exercise intensity was determined from mean HR using the method described by Karnoven et al.(1957)

3.2.3.4 Expired sample volume collection

$\dot{V}O_2$ and corresponding KJ expenditure during tennis games were assessed using the open -circuit method of indirect calorimetry.

The subjects were fitted with a two-way Hans Radolf mask (Model No.7910,Hans Rudolf Inc., Kansas City, USA) specifically designed for children. It covered the nose as well as the mouth of the subject (fig. 3.2). Inhaled atmospheric air passed through a three way Otis McKeron breathing valve. The mask was connected with tubing to a light weight neoprene air collection bag (Dynamic Image, JNB). A 60 $\ell$ neoprene bag was used for the children and a 100 $\ell$ bag was used for the young adults. The subjects had been familiarized with the equipment prior to the match to ensure that they felt comfortable and relaxed wearing it while playing tennis and that it did not restrict their movement while on the court

The experimenter and two assistants secured the neoprene bag and mask onto the subject and recorded the duration of collection period (a minimum of sixty seconds) by using a stopwatch and opening or closing the valve which regulated the influx of expired air into the neoprene bag. Three to four collections of expired volume, were taken from each player in the neoprene bag. It took no more than 30 s for the subjects to remove the neoprene bags and resume play.





**Fig. 3.2.** ***Fitting of the Neoprene Bag and Hans Rudolf face mask. (Photographs: C. Nicholson)***

After the test, the neoprene bag was connected to a rubber bladder. A small fraction of the exhaled air was then diverted into the rubber bladder. The neoprene bag was then connected to a Harvard Dry Gas Meter and the total volume of the expired air was read off. The temperature of the expired air was recorded during the measurement of the total volume using an electronic thermometer (Physiotemp, BAT-12) which was placed into the outlet of the dry gas meter.

A total of 80 samples of expired air were collected during tennis play from the 24 subjects. The fractions of expired air collected in the rubber bladders, were analyzed for relative concentrations of $O_2$ and $CO_2$. These values, together with the recordings of $V_E$ ($l.min^{-1}$), barometric pressure and expired air temperature, were used to calculate $\dot{V}O_2$ consumption. Energy costs were calculated from the $\dot{V}O_2$, and the equivalent KJ cost (per $\ell$ of oxygen) was calculated from the corresponding RQ. Formulas used for the above mentioned calculations are given in 3.4.

### 3.2.4 Biochemical measurements

#### 3.2.4.1 Venous blood sample

The subjects were permitted to consume a breakfast or a light meal 2 hours before commencement of the matches. Whole blood specimens were taken immediately before and after competition in order to determine blood glucose concentration, Hct, Hb concentrations and electrolyte concentrations ($Na^+$, $K^+$, $Mg^{++}$, $Cl^-$).

The subjects were in a seated position during the blood sampling. A 13 ml whole blood sample of venous blood was withdrawn hypodermically from a superficial forearm peripheral vein of the non-playing arm with a vacu-test system by a qualified nursing sister. The tension placed by tourniquet application was released before one minute after the vein was punctured, and before the blood was sampled in order to prevent mechanical increase in postcapillary resistance which can raise mean capillary

pressure and cause a net filtration of venous blood flow with resultant increase in the Hct in relation to the duration of occlusion (Mollison, 1983).

Six ml of the sample were placed into a Lithium Heparin tube for the determination of electrolyte concentrations ($Na^+$, $K^+$, $Mg^{++}$, $Cl^-$) and TPP content using the Biuret method and four ml of the sample was placed into a ETDA tube for Hct determination using an automated Technicon H* 2 system and for Hb concentration using an automated coulter S-II counter. A further three ml was used for the determination of blood glucose analysis levels using the Beckman Oxygen Electrode method.

The prepared samples were stored on ice and delivered to a biochemistry and biochemical pathology laboratory for immediate analysis. Two children refused to have post match blood samples taken due to fear of the invasive nature of the procedure.

### 3.2.5 Assessment of environmental conditions

#### 3.2.5.1 Heat stress index

This was obtained using the Wet Bulb Globe Temperature index (WBGT) which incorporates air temperature, humidity and radiation as measured by three thermometers: a dry bulb (DB), a wet bulb (WB) and a black globe (G) and was determined with the use of the formula:

$$\text{WBGT index} = 0.7T_{wb} + 0.2T_g + 0.1T_{db}$$

A Casellas whirling Psychometer (London) was used for the measurement of dry and wet bulb temperature. Relative humidity was derived using the wet bulb and dry bulb temperatures.

A mechanical Pyranographer (Solar radiation recorder, Model 3010, Sacramento, California), was used to measure radiation in gm-cal.cm$^{-2}$.min$^{-1}$.

3.2.5.2 Measurement of wind speed

This was obtained using an electronic wind speed indicator (Turbo-Meter, Hayward, California), which measures wind speed in meters per second (m.sec$^{-1}$) and provides an instantaneous reading when held in the air.

3.2.5.3 Measurement of atmospheric pressure

This was obtained using a Wallis and Tiernan barometer (Model ML/102/D, Belleville, New Jersey) of known validity and reliability. The values were used for the calculation of $\dot{V}O_2$ and $\dot{V}CO_2$. At each experimental session the above indices of temperature, wind speed and barometric pressure were recorded throughout the tennis match. The minimum and maximum values were then determined to provide the session's range of values.

## 3.3 Laboratory analysis

The laboratory analysis included the determination of the rubber bladder's volume and the concentrations of $O_2$ and $CO_2$. The volumes of the rubber bladders were measured and added to the volume of the expired air ($V_E$) measured in each sample of air collected. This was done using the principle of Archimedes. The rubber bladder was inserted into a plastic bucket containing water and the water displaced by the volume of the rubber bladder was measured. The necessary corrections were made for the volume of the empty rubber bladder and hand of the researcher (which were previously measured).

Subsequently, the rubber bladder was connected to the gas analyser of the Oxycon IV (Mijhardt,Bunnik, Holland) to measure the concentration of $O_2$ and $CO_2$ in the expired air. It was hypothesized that due to the time lapse from the time of collection of

expired air until the time of analysis some diffusion of gas might have taken place. Thus, each rubber bladder's diffusion rate, was pre-determined over a three hour period. The estimated percentage of diffused gas was added to the actual concentration of $CO_2$ measured in the rubber gas collection bag. As no diffusion of $O_2$ out of the bag was observed during the three hour period, it was not neccessary to apply this correction factor to the $O_2$ concentration.

## 3.4 Formulas utilized in the data calculations

The basic anthropometric, physiological and biochemical measurements were substituted into formulae to obtain the derived morphological, physiological and biochemical data.

### 3.4.1 Body surface area

Predicted BSA was determined from the Du Bois Height-body mass Formula (Du Bois, 1916).

$$BSA = 71{,}84 \left( M^{0.425} \times S^{0.725} \right)$$

where BSA = body surface area ($m^2$)

M = body mass (kg)

S = stature (cm)

BSA/mass ratio was calculated as $m^2/Kg \times 10.000$.

### 3.4.2 Percentage body fat

The sum of the tricep, sub-scapular, supra-iliac, abdominal, front thigh and medial calf skin folds in millimetres were used (Yuhasz, 1974). The following formula was used to determine percentage body fat:

$$\text{Percentage fat} = [\ \Sigma\ 6 \text{ skin folds} \times 0.1051\ ] + 2.585 \quad \text{(Yuhasz, 1974)}$$

### 3.4.3 Mass-loss

The nett body mass change was used to determine the amount of mass-loss. The change was corrected for the liquid intake and urine voided during the match with the use of the formula:

$$TML = IBM - FBM + LI - UV$$

where TML = total mass loss (Kg)

IBM = initial body mass (Kg)

FBM = final body mass (Kg)

LI = liquid intake ($\ell$)

UV = urine voided ($\ell$)

This total mass loss included sweat losses, the respiratory tract water losses and the metabolic fuel utilized.

Sweat losses were calculated with the use of the formula:

$$SL = TML - (MFU + RWL)$$

where SL = sweat loss (Kg)

MFU = metabolic fuel utilized (Kg)

RWL = respiratory water loss (Kg)

Sweat losses were assumed to include plasma volume water losses, the water released from glycogen breakdown and the metabolic water produced by mitochondrial oxidation.

Fuel metabolized(g) was calculated using the estimated mean energy expenditure (KJ per $\ell$ of $O_2$) and the use of the corresponding non-protein respiratory quotient (RQ) for steady state exercise (Mc Ardle et al., 1991) to determine the relative usage of carbohydrates and fats. For the calculation of the quantity (g) of CHO and fat oxidized, the following assumptions were made: 17 KJ are released per gram of carbohydrate oxidized, and 40 KJ are released per gram of fat oxidized (Noakes et al., 1991b). Respiratory water loss was calculated as 0.026 $g.kJ^{-1}$ (Pugh, 1967).

Sweat loss in kg (mass) was converted to volume ($\ell$) by means of the formula:

$$V = M / D$$

where V = mass loss volume ($\ell$)

M = mass loss (Kg)

D = density of sweat (1.003)

Sweat rate ($\ell.hr^{-1}$) was determined by dividing the total sweat loss in $\ell$ by the duration of the tennis match in hr. Sweat rate was estimated relatively in $\ell.m^{-2}.hr^{-1}$ using the formula: $\frac{SR}{BSA}$

Water deficit (WD) was calculated by the use of the formula:

$$WD = [TML - (MFU + MWP)] / 2$$

where MWP = metabolic water produced (Kg)

The metabolic water produced by mitochondrial oxidation was estimated from the assumption that 0,55 g of water are produced per gram of carbohydrate oxidized and 1,07 g of water are produced per gram of fat oxidized (Noakes et al., 1991b).

Following the recommendations of (Olsson and Saltin, 1970; Noakes et al., 1988; 1991b), water released from glycogen breakdown was not considered as water deficit. According to the procedure described by Noakes et al.(1988), it was assumed that 50% of the WD consisted of water released from glycogen breakdown. Thus, by substracting this percentage volume, WD, level of dehydration and consequently more accurate fluid replacement needs were calculated.

Rate of water loss and fluid replacement was then calculated by expressing WD as a function of duration of the match in hours ($\ell.hr^{-1}$).

Percent dehydration was calculated as follows: % dehydration = WD / IBM x 100

### 3.4.4 Sample metabolic calculations

A series of formulae were used to create programmes to calculate the $\dot{V}O_2$, $\dot{V}CO_2$, RQ and energy expenditure ($kJ.min^{-1}$). The data collected consisted of: $V_E$ (ATPS); barometric pressure (mmHg); temperature of gas in the bag (°C); $\%O_2$ expired air (measured); $\%CO_2$ expired (measured); $\%N_2$ expired [ 100 - ($\%O_2$ expired + $\%CO_2$ expired) ].

$\dot{V}_E$ (ATPS) was converted to $\dot{V}_E$ (STPD) with the use of the following equation:

$$\dot{V}_E\ STPD = \dot{V}_E,\ ATPS \left(\frac{273}{273 + TC}\right) \left[\frac{P_B - P_{H_2O}}{760}\right]$$

where $T\ ^{\circ}C$ = temperature of the gas in the volume meter

273 = absolute temperature Kelvin

$P_B$ = the ambient barometric pressure

$P_{H_2O}$ = water vapour pressure

The vapour pressure ($P_{H_2O}$) of wet gas at a particular ambient temperature was obtained from the relevant table (Mc Ardle et al., 1991) and was expressed in mmHg.

To determine $\dot{V}O_2$ the following equation was used:

$$\dot{V}O_2, STDP = \dot{V}_E, STDP[(\%N_{2E} \times .265) - \%O_{2E}] \text{ (Mc Ardle et al., 1991)}$$

where $\%N_{2E}$ = percent nitrogen in expired air computed from gas analysis as [(100 $-(\%O_{2E} + \%CO_2)$].

$\%O_{2E}$ = percent oxygen in expired air determined by gas analysis

The carbon dioxide production per min $\dot{V}CO_2$ was calculated as follows:

$$\dot{V}CO_2, STDP = \dot{V}_E, STDP(\%CO_{2E} - 0.03\%) \text{ (Mc Ardle et al., 1991)}$$

where $\%\ CO_{2E}$ = percent carbon dioxide in expired air determined by gas analysis

0.03% = percent carbon dioxide in inspired air which is essentially constant

The non-protein RQ was calculated from the formula

$$RQ = \dot{V}CO_2/\dot{V}O_2$$

The absolute energy cost of competitive singles tennis playing was determined by means of the formula:

$$EE = \dot{V}O_2 \times CE \times 4.183$$

where EE = energy expenditure ($KJ.min^{-1}$)

$\dot{V}O_2$ = oxygen consumption ($\ell.min^{-1}$ STPD)

CE = caloric equivalent per $\ell$ $O_2$ at the given steady-rate RQ

4,183 = kJ conversion factor

Estimation of the percentage and quantity of fat and carbohydrate metabolized during each minute of tennis was used also to estimate grams of fat and carbohydrates utilized, by multiplying $\dot{V}O_2$ (STDP) with the corresponding grams of fat and carbohydrates utilized per $\ell$ of oxygen at the specific RQ (Mc Ardle et al., 1991).

### 3.4.5 Estimation of exercise intensity during the match

Exercise intensity was indirectly estimated using the HR. THRR was set according to the ACSM (1978) criteria for developing cardiovascular fitness i.e. under the target (<60% of HRmax reserve), within the target (60-85% of HRmax reserve) and over the target (>85% of HRmax reserve). Considering the problems arising from the factor, age (section 4.3.3), the three THRR for group one and two (children) were <160 bpm, 160-190 bpm and >190 bpm respectively whereas corresponding THRR in group three (young adults) were <155 bpm, 155-185 bpm and >185 bpm respectively.

Approximately 30 samples of expired air were collected from each group and the same number of HR responses were recorded in this study. Thus, regression equations (Astrand and Rodahl, 1977) were applied to each group and to all the subjects, in o der to compare and estimate mean $\dot{V}O_2$ between groups and during competitive tennis play. Furthermore, the data were also used to compare exercise intensity level in tennis with other type of sports.

### 3.4.6 Calculation of percentage changes in BV constituents during exercise

The Hct value [the ratio of red cell volume (RCV) to the BV], Hb and TPP concentrations were used to calculate intravascular volume responses and expressed as a percent change from control values.

BV (the sum of red cell and PV) changes were calculated from the Hb concentration changes. Taking the initial (control) BV to be unity and assuming no change in intravascular Hb concentration, any change in pre- and post- match Hb concentration in the blood were assumed to be due to BV changes. The following formula were utilized for the above notation (Dill and Costill, 1974):

$$BV_a = Hb_b \times 100/Hb_a$$
$$\%\Delta BV = BV_b - BV_a$$

where $BV_b$, is assumed to be 100 ml

Taking into account the %ΔBV, percent changes in red cell volume %RCV after the match were calculated with the use of the formula (Dill and Costill, 1974):

$$RCV_a = Hct_a \times BV_a / 100$$
$$\%\Delta RCV = RCV_a - Hct_b$$

Mean corpuscular hemoglobin concentration changes (ΔMCHC)(the Hb concentration in g.100 $ml^{-1}$ of blood divided by the packed cell volume, expressed as a percentage), were calculated in order to identify any changes in mean corpuscular volume (MCV) which may result from alterations in plasma osmolality (Costill et al., 1974). The following formulae (Costill and Fink, 1974) were used:

$MCHC_b = Hb_b/Hct_b$

$MCHC_a = Hb_a/Hct_a$

The difference in concentration in $MCHC_b$ and $MCHC_a$ was used to calculate percent changes in mean corpuscular volume (MCV) (Costill and Fink, 1974):

$$\%\Delta MCV = MCHC_b - MCHC_a \,/\, MCHC_b \times 100$$

Percent changes in PV were calculated from changes in both Hct and Hb to correct for any changes in cell volume that may be associated with changes in PV or osmolality (Strauss et al., 1951):

$$\%\Delta PV = 100 \times \left[ \frac{Hb_b}{Hb_a} \times \frac{100 - Hct_a}{100 - Hct_b} \right] - 100$$

To identify if any loss or gain of TPP had occurred from the circulation, the expected TPP concentration was calculated taking %ΔPV into account. For example, if no change in TPP had occurred, the increase in PV would imply(indicate) a TPP gain. Calculation of the expected total plasma protein ($TPP_E$) from %ΔPV was obtained by the use of the formula (Dill and Costill, 1974):

$$TPP_E = TPP_b \times 100 \text{ (+ or -) } \%\Delta PV \,/\, 100ml$$

Calculation of the true %ΔTPP was obtained from the following formula (Dill and Costill, 1974):

$$\%\Delta TPP = TPP_E - TPP_a \,/\, TPP_b \times 100$$

In all the preceding equations, Hb was expressed in $g.dl^{-1}$, Hct in %, TPP in $g.ml^{-1}$, and $_b$ and $_a$ refered to values before and after the match respectively.

## 3.5 Statistical analysis of data

Descriptive statistics (average, standard deviation, minimum and maximum values) were used on measured data obtained from the 24 subjects. To detect any significant differences between pre and post-matches measurements, one sample t-test were performed at a 95% confidence level. The Pearson product moment correlation coefficient was conducted to determine whether significant relationships ($p < 0.05$) between variables, were influenced post-match $T_{re}$, sweat losses and water ingested by the players. Where a significant correlation was found ($p < 0.05$), a simple linear regression was performed in order to predict values of the related variables. One way analysis of variance was performed to determine the significance of differences between group means and pre- and post-match measurements between groups. Where significant F ratios were obtained ($p < 0.05$), Scheffe's multiple comparison tests were applied to detect where the differences existed between the groups. Stepwise regression analysis was used to determine the proportion of the total variances in each dependant variable that could be accounted for by the measured variables. When necessary, confidence levels were set to 90% due to the small sample size of the subject groups. The above mentioned statistical analyses were performed using the Statgraphics (STSC), version 5, computer programme (Rockville, Maryland, USA, 1991).

# CHAPTER FOUR

# RESULTS

4.1 ANTHROPOMETRIC AND PHYSICAL MEASUREMENTS

4.2 ENVIRONMENTAL MEASUREMENTS AND INDICES

4.3 PHYSIOLOGICAL MEASUREMENTS

4.4 BIOCHEMICAL AND HEMATOLOGICAL MEASUREMENTS

4.5 QUESTIONNAIRES

## 4.1 Anthropometric and physical measurements

Mean (±SD) anthropometric and physical characteristics of the three groups are presented in Table 4.1. Raw data including pre- and post-match measurements of the above characteristics are shown in Appendix C.1.1. Post-match body mass measurements were significantly lower than pre-match body mass measurements in all groups (Table 4.9).

One way analysis of variance on the groups means (Appendix C.7.1) revealed significant differences only in age and height. Group one and two were younger than group three, and group three characterised by greater stature than group two ($p<0.05$). Mean body mass, BSA, BSA/mass ratio and percentage body fat between the groups were insignificantly different ($p>0.05$).

Pearson product-moment correlations on anthropometric and physical variables are presented in Appendix C.8.1. Only those correlations considered to be of relevance to this study are included. From Appendix C.8.1 it can be seen that stature is significantly correlated with age and mass in all groups.

## 4.2 Environmental conditions

Recordings of environmental conditions i.e. $T_{db}$, $T_{wb}$ and $T_g$, together with the derived ranges of WBGT index during the five data collection sessions are presented in Table 4.2. Additional recorded environmental conditions i.e. humidity, barometric pressure, wind speed and ground temperature are presented in Appendix C.2.

WBGT index ranged from 13-19. The average ambient temperature ranged from 25° to 28.5 °C, while relative humidity varied from 34 to 70 percent. Barometric pressure fluctuated between 635 and 643 mmHg.

**Table 4.1: Mean (±SD) anthropometric and physical characteristics of the three groups of subjects (n=24).**

| Variables | Group 1 (n=8) | Group 2 (n=8) | Group 3 (n=8) | Total Group |
|---|---|---|---|---|
| Age (years) | 12.79 (0.37) | 12.8 (0.64) | 14.8$^{+}$ (0.47) | 13.35 (1.31) |
| Mass (Kg) | 45.9 (6.2) | 45.9 (10.9) | 53.3 (8.94) | 48.41 (9.27) |
| Height (m) | 1.60 (0.07) | 1.54 (0.09) | 1.66$^{++}$ (0.08) | 1.60 (0.09) |
| BSA ($m^2$) | 1.44 (0.13) | 1.40 (0.19) | 1.58 (0.16) | 1.47 (0.17) |
| BSA/mass ratio | 313.7 (14.6) | 305.0 (39.3) | 296.4 (20.3) | 303.7 (26.8) |
| % Body fat (%) | 10.42 (3.59) | 10.69 (4.60) | 8.96 (2.78) | 10.02 (3.65) |

$^{+}$: significantly different ($p<0.05$) to group one and two

$^{++}$: significantly different ($p<0.05$) to group two

## 4.2 Environmental conditions

Recordings of environmental conditions including $T_{db}$, $T_{wb}$ and $T_g$, together with the derived ranges of WBGT index during the five data collection sessions are presented in Table 4.2. Additional recorded environmental conditions including humidity, barometric pressure, wind speed and ground temperature are presented in Appendix C.2.

WBGT index ranged from 13-19. The average ambient temperature ranged from 25° to 28.5 °C, while relative humidity varied from 34 to 70 percent. Barometric pressure fluctuated between 635 and 643 mmHg.

**Table 4.2: Environmental conditions during the five experimental sessions: Ambient Temperature.**

| | Date | $T_{db}$ (°C) | $T_{wb}$ (°C) | $T_g$ (°C) | WBGT index (cal.cm$^{-2}$. min$^{-1}$) |
|---|---|---|---|---|---|
| Group 1 | 22.02.92 | 25-28.5 | 22.5-23 | 0.5-1.6 | 18-19 |
| Group 2 | 07.03.92 | 25.4-25.6 | 20-20.1 | 0.6-1 | 17-17 |
| | 19.03.92 | 26-27.5 | 18-19 | 0.5-1.5 | 15-16 |
| Group 3 | 26.03.92 | 24-25.5 | 15.5-16 | 0.8-1.5 | 13-14 |
| | 02.04.92 | 25-27 | 15-16 | 0.7-1.7 | 13-14 |

All values in $T_{db}$ $T_{wb}$ $T_g$ and WBGT index are expressed as maximum and minimum

$T_{db}$: Dry bulb temperature

$T_{wb}$: Wet bulb temperature

$T_g$: Globe bulb temperature

WBGT: Wet Bulb Globe Temperature index

## 4.3 Physiological variables

The group body mass changes, recorded measurements and calculated water deficit, as well as estimated fluid replacement needs are presented in Table 4.3.1 and Table 4.3.2 respectively. The above fluid balance components are presented for each subject, in Appendix C.6.

On average the subjects (n=24) ingested 0.427ℓ of water, and the mean total mass loss was 0.907 Kg. It was calculated that sweat contributed 0.780 ℓ to total fluid loss, whereas respiratory water loss and metabolic fuel utilized accounted for 0.120 ℓ. Calculated water deficits of 0.390 ℓ resulted in 0.80 % dehydration. Mean fluid replacement needs of the subjects (n=24) was determined to be 0.390(±0.141) ℓ during the 90 min of tennis play (Table 4.3.2). This is equivalent to a rate of ±262 $ml.hr^{-1}$ or ±5.41 $ml.kg^{-1}.hr^{-1}$.

The time(min) spent within each HRR of the groups and total group is shown in Table 4.4. Time spent(min) within each HRR by the subjects is shown in Appendix C.3. Related variables such as $T_{re}$, recorded playing time are also presented in Table 4.4.

The total group HRR are presented graphically in figure 4.1. The time spent within each HRR are expressed in percentages. The percentage of time spent within the THRR, which is necessary for cardiovascular development (ACSM, 1978), is presented in the off-set piece included in figure 4.1. From the figure it is apparent that a very short time(min) was spent by the subjects within the target HRR.

The HR measurements obtained during continuous testing in a subsample of subjects together with measured and related physiological responses are presented in Table 4.5.

Post-match $T_{re}$ was significantly greater than the pre-match value in all groups ($p<0.05$) (Table 4.9). One way analysis of variance revealed that exercise intensity, as reflected by the time spent within each HRR, did not show any significant

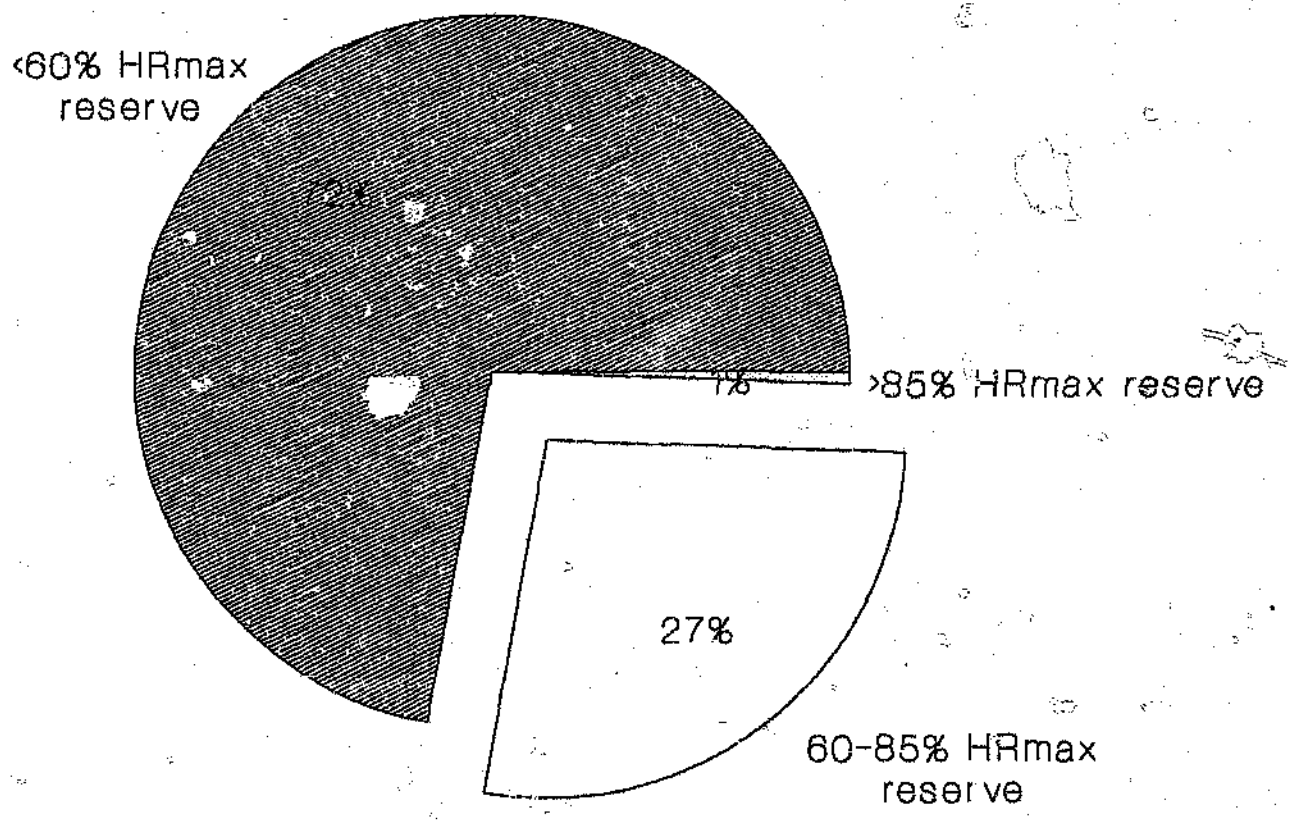



**Fig. 4.1.** *Percentage of time within each of the three HR ranges recorded during 9hmin of competitive tennis play (n=24).*

difference between the groups. However group one spent relatively more time(min) playing within the THRR than group two and three (33 vs 22 and 18min respectively). The analysis also revealed significantly higher SR, dehydration (%), total mass loss, and water loss (gr) in group one as compared to group two (Appendix C.7.1). Percent dehydration (total and $.h^{-1}$) was also significantly higher in group one when compared to group three.

Although significantly lower oxidation of CHO was found in group two than in group one and three, oxidation of fat was found to be significantly greater in group two than in group one and three (Appendix C.7.1). Metabolic water released from fuel oxidation was significantly lower in group two than in group three.

One way analysis of variance indicated that pre- and post-match $T_{re}$ were insignificantly different between the three groups (Appendix C.7.2). It was also of interest to test for time dependency in $T_{re}$ control measurements. A one way analysis of variance on pre-match $T_{re}$, after grouping the subjects accordingly to recorded time of $T_{re}$, revealed that $T_{re}$ was also insignificantly different ($p<0.05$) between morning and afternoon measurement sessions.

Pearson product-moment correlations on post-match $T_{re}$ conducted in the three subjects groups are presented in Appendix C.9. Time(min) spent within the THRR and over the THRR in group one, were the only variables correlated significantly with post-match $T_{re}$ ($p<0.05$).

The simple linear regression equations of HR-$\dot{V}O_2$ relationship of each group and in the total group are presented in Appendix C.10.1. The regression line of total group is shown in figure 4.2. In addition, simple linear regression equations of physiological variables within the groups are also presented in Appendix C.10.1.

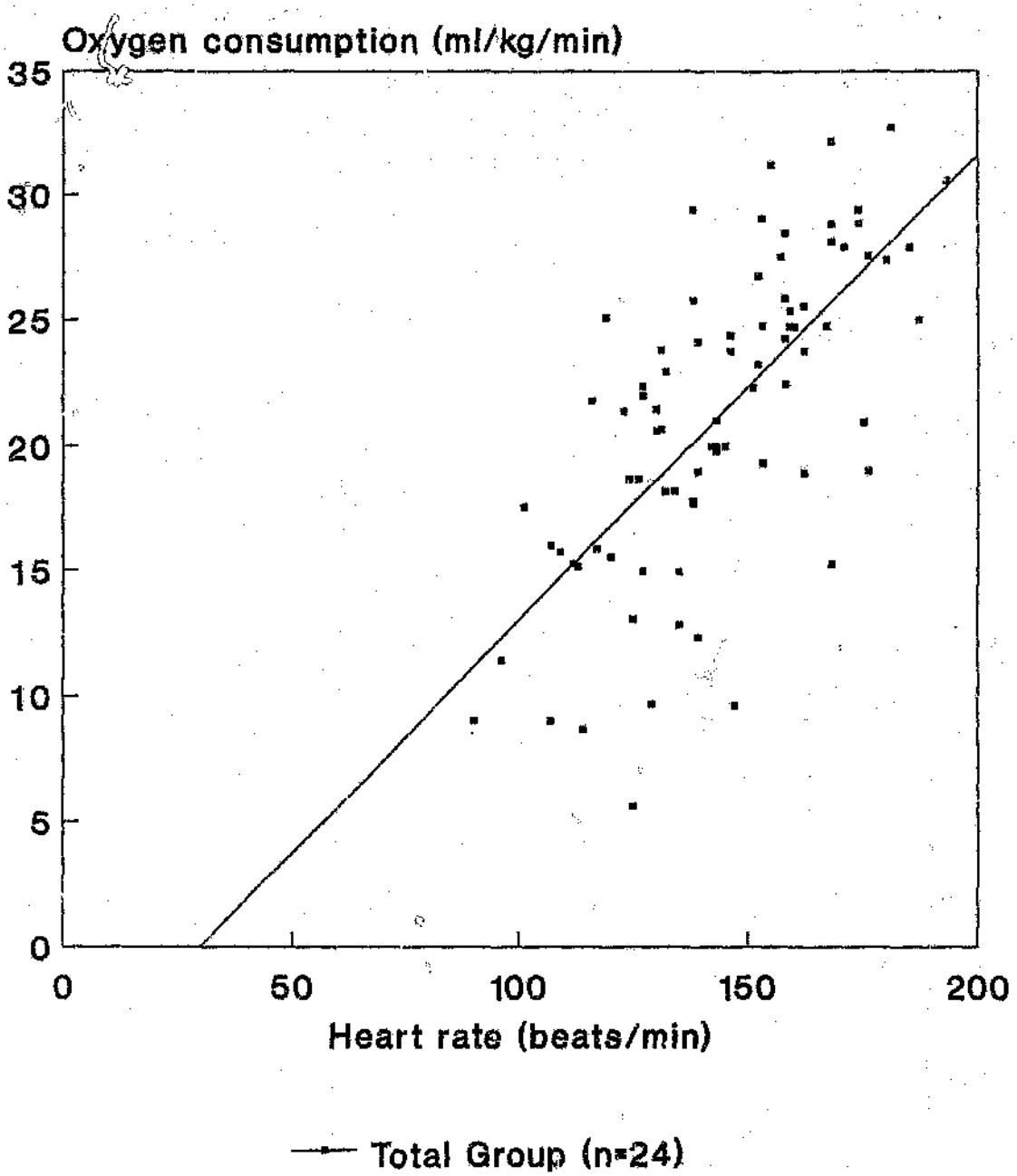



*Fig. 4.2. Linear regression of HR-$\dot{V}O_2$ relationship of the subjects (n=24).*

Backward stepwise regression equations were determined for post-match $T_{re}$ and sweat loss. The subjects in group one displayed the least variability in exercise intensity compared to the other two groups. Therefore multiple linear regression equations of $T_{re}$ and sweat loss responses were only applied to group one. Two significant models were detected (Table 4.6). It was seen that final $T_{re}$, could be predicted from time spent within the THRR and from time spent under the THRR. Sweat loss could be predicted from total dehydration (%) and from post-match $T_{re}$.

**Table 4.3.1: Mean(±SD) measured total mass and fluid losses of the three groups of subjects (n=24).**

| Variables | Group 1 | Group 2 | Group 3 | Total Group |
|---|---|---|---|---|
| IBM (Kg) | 45.91<br>(6.27) | 45.93<br>(10.99) | 53.38**<br>(8.94) | 48.41<br>9.27 |
| FBM (Kg) | 45.32<br>(6.24) | 45.73<br>(10.91) | 52.73**<br>(8.71) | 47.93<br>9.12 |
| ΔBM (Kg) | 0.59<br>(0.13) | 0.20+<br>(0.2) | 0.65<br>(0.28) | 0.48<br>0.29 |
| LI ($\ell$) | 0.473++<br>(0.189) | 0.503+++<br>(0.236) | 0.306<br>(0.102) | 0.427<br>(0.197) |
| LI ($\ell.hr^{-1}$) | 0.318++ | 0.340+++ | 0.204 | 0.286 |
| LI ($ml.Kg^{-1}.hr^{-1}$) | 6.92 | 7.40 | 3.82* | 5.90 |
| UV ($\ell$) | 0 | 0 | 0 | 0 |
| TML (Kg) | 1.063**<br>(0.188) | 0.703<br>(0.243) | 0.956<br>(0.307) | 0.907<br>(0.258) |

IBM: initial body mass; FBM: final body mass; ΔBM: change in body mass; LI: liquid intake; UV: urine voided; TML: total mass loss

+ $p<0.05$ G2 vs G1 and G3; ++ $p<0.001$ G1 vs G3; +++ $p<0.001$ G2 vs G1 and G3;
* $p<0.001$ G3 vs G1 and G2; ** $p<0.05$ G1 vs G2

**Table 4.3.2: Mean(±SD) calculated and estimated fluid losses and fluid replacement needs in the three groups of subjects(n=24).**

| Variables | Group 1 (n=8) | Group 2 (n=8) | Group 3 (n=8) | Total Group (n=24) |
|---|---|---|---|---|
| MFU (Kg) | 0.079 (0.023) | 0.053$^{+}$ (0.053) | 0.091 (0.015) | 0.074 (0.022) |
| MWP ($\ell$) | 0.051 (0.014) | 0.043$^{++}$ (0.010) | 0.059 (0.00) | 0.051 (0.012) |
| RWL ($\ell$) | 0.045 (0.011) | 0.040$^{+++}$ (0.009) | 0.052 (0.007) | 0.046 (0.010) |
| SL ($\ell$) | 0.936$^{*}$ (0.189) | 0.609 (0.243) | 0.813 (0.322) | 0.786 (0.282) |
| SL ($\ell.hr^{-1}$) | 0.631$^{*}$ | 0.411 | 0.542 | 0.528 |
| Playing time(min) | 89 (13.2) | 88.7 (0.88) | 90 (0) | 89.3 (7.33) |
| WD ($\ell$) | 0.465$^{**}$ (0.095) | 0.303 (0.122) | 0.403 (0.162) | 0.390 (0.141) |
| Deh., (%) | 1.01$^{***}$ (0.19) | 0.65 (0.19) | 0.74 (0.24) | 0.80 (0.25) |
| Deh., ($\%.hr^{-1}$) | 0.68$^{***}$ | 0.44 | 0.49 | 0.54 |
| FRN ($\ell$) | 0.465$^{\#}$ (0.095) | 0.303 (0.122) | 0.403 (0.162) | 0.390 (0.141) |
| FRN ($\ell.hr^{-1}$) | 0.313$^{\#}$ | 0.204 | 0.268 | 0.262 |
| FRN ($ml.Kg^{-1}.hr^{-1}$) | 6.81$^{\#\#}$ | 4.44 | 5.02 | 5.41 |

MFU: metabolic fuel utilized; MWP: metabolic water produced; RWL: respiratory water loss; SL: sweat loss; WD: water deficit; Deh: dehydration; FRN: fluid replacement needs

$^{+}$ $p<0.05$ G2 vs G1 and G3; $^{++}$ $p<0.05$ G2 vs G3; $^{+++}$ $p<0.05$ G2 vs G3;
$^{*}$ $p<0.05$ G1 vs G2; $^{**}$ $p<0.001$ G1 vs G2; $^{***}$ $p<0.05$ G1 vs G2 and G3;
$^{\#}$ $p<0.05$ G1 vs G2; $^{\#\#}$ $p<0.05$ G1 vs G2 and G3

**Table 4.4: Mean (±SD) time spent under, within and above the target HR zone and $T_{re}$ changes of the subjects (n=24).**

| Variables | Group 1 | Group 2 | Group 3 | Total Group |
|---|---|---|---|---|
| U, THRR(min) | 55.37 (3.073) | 67.25 (17.32) | 71.6 (22.82) | 64.38 (18.55) |
| W, THRR(min) | 33.12 (15.45) | 21.87 (29.12) | 18.37 (17.32) | 24.38 (21.39) |
| O, THRR(min) | 0.43 (1.23) | 1 (2.82) | 0 (0) | 0.48 (1.75) |
| Playing time(min) | 89 (13.2) | 88.7 (0.88) | 90 (0) | 89.25 (7.33) |
| Pre- $T_{re}$ (°C) | 37.48 (0.09) | 37.42 (0.20) | 37.28 (0.29) | 37.4 (0.22) |
| Post-$T_{re}$ (°C) | 38.23 (0.30) | 38.17 (0.16) | 38 (0.23) | 38.13 (0.25) |
| $\Delta T_{re}$ (°C) | 0.75 (0.35) | 0.70 (0.25) | 0.75 (0.19) | 0.73 (0.26) |

U, THRR: under the target heart rate range; W, THRR: within the target heart rate range
O, THRR: over the target heart rate range; Pre: before the match; Post: after the match

**Table 4.5: Mean HR of continuous HR recordings during the 90 min of play and $\dot{V}O_2$, carbon dioxide production and related measurements taken at selected intervals during the 90 minute period of play (n=13).**

| Gr. | Sub. | HR bpm ($\bar{X}$,SD) | $\dot{V}O_2$ $\ell.Kg^{-1}.min^{-1}$ | $\dot{V}O_2$ $\ell.min^{-1}$ | $\dot{V}CO_2$ $\ell.min^{-1}$ | RQ | Total KJ | $KJ.hr^{-1}$ | $KJ.Kg^{-1}.min^{-1}$ | Playing time (min) |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | K.R | 154(13) | 22.56 | 1.26 | 1.12 | 0.89 | 2134 | 1561 | 0.46 | 82 |
| | B.C | 158(14) | 24.02 | 0.94 | 0.85 | 0.91 | 1862 | 1165 | 0.49 | 96 |
| | B.D | 152(17) | 22.69 | 0.84 | 0.74 | 0.89 | 1540 | 1039 | 0.46 | 89 |
| | B.R | 162(13) | 24.82 | 1.19 | 1.08 | 0.91 | 1724 | 1476 | 0.51 | 70 |
| 2 | M.J | 171(16) | 24.63 | 0.66 | 0.56 | 0.85 | 1239 | 816 | 0.50 | 91 |
| | G.H | 113(19) | 14.38 | 0.83 | 0.62 | 0.75 | 1456 | 991 | 0.28 | 88 |
| | E.Z | 127(12) | 17.14 | 0.83 | 0.64 | 0.78 | 1477 | 996 | 0.34 | 89 |
| | S.W | 143(14) | 19.75 | 0.74 | 0.59 | 0.80 | 1339 | 876 | 0.39 | 90 |
| 3 | K.K | 133(15) | 20.62 | 1.08 | 1.01 | 0.94 | 2034 | 1356 | 0.42 | 90 |
| | D.G | 146(13) | 23.01 | 1.06 | 1.01 | 0.96 | 2005 | 1335 | 0.48 | 90 |
| | A.R | 147(16) | 22.19 | 0.95 | 0.84 | 0.89 | 1762 | 1175 | 0.45 | 90 |
| | P.J | 155(12) | 24.66 | 1.08 | 1.01 | 0.94 | 2051 | 1353 | 0.51 | 90 |
| | C.B | 126(15) | 19.34 | 1.13 | 1.02 | 0.91 | 2264 | 1408 | 0.39 | 90 |
| $\bar{X}$ | | 145 | 21.52 | 0.96 | 0.85 | 0.87 | 1760 | 1100 | 0.44 | 88 |
| SD | | 16 | 3.17 | 0.18 | 0.20 | .06 | 328 | 403 | 0.07 | 6.1 |

**Table 4.6: Stepwise regression equation on final $T_{re}$ and SL of group one (n=8).**

| Response variable | Equation | SE | (Adj.$R^2$) | n |
|---|---|---|---|---|
| Final $T_{re}$(°C) | $\psi_1 = 36.836536 + .02526X_1 + + .01003X_2$ | 0.12 | 0.81 | 8 |
| Sweat loss($\ell$) | $\psi_2 = 11.580 + 402.263077X_3 - - 300.807773X_4$ | 1.75 | 0.73 | 8 |

The predictor $X_1$, $X_2$, $X_3$, $X_4$, variables represent:

$X_1$ = min spent within the target HRR

$X_2$ = min spent under the target HRR

$X_3$ = total dehydration (%)

$X_4$ = final $T_{re}$

SE: standard error of estimation

Adj.$R_2$: adjusted squared multiple correlation coefficient

n: numbers of observations

## 4.4 Biochemical and haematological measurements

The group mean and standard deviation of the pre- and post-match blood Hct, Hb, glucose, TPP and electrolyte concentrations are shown in Table 4.7. The derived percentage changes of the above measurements and BV compartments are shown in Table 4.8. Furthermore, the above pre- and post-match measurements of the subjects as well as the derived calculated BV compartment changes are presented in Appendix C.5.1 to C.5.3.

Insignificant changes in $Mg^{++}$ were observed in all groups (Table 4.9). However, significant changes with different significance levels ($p<0.05$; $p<0.001$) were observed for the other parameters. These include the significant rise ($p< 0.05$) between pre- and post-match measurements in $Na^{+}$ (group one and three), in $Cl^{-}$ (group two and three) and TPP (group one). Although, fluid ingested by the players did not contain sugar at all, post-match blood glucose levels were significantly higher in group one ($P<0.05$) and they were insignificantly changed in group two and three as compared to the pre-match blood glucose levels (Table 4.9). The increase in blood glucose level in group one, who also spent the most time within the THRR, was $0.71 mmol.l^{-1}$.

Since measurements of Hct and Hb blood parameters were not conducted in group one, it was not possible to assess whether differences in PV and BV responses existed in this group. However, analysis of variance showed that percent ΔTPP calculated from percent ΔPV changes was significantly higher ($p<0.05$) in group two and three (Appendix C.7.1). Furthermore, the analysis revealed that group three had significantly greater MCV and RCV changes than group two but Hb, Hct, BV and PV changes were insignificantly different between the two groups.

One way analysis of variance on the base-line Hb, Hct and TPP values did not reveal significant differences between the groups ($p>0.05$). However, pre-match blood

glucose levels of groups one and two show significantly lower values than group three (Appendix C.7.2).

Pearson product-moment correlations analysis revealed that percent ΔMCV was inversely strongly related to percent ΔRCV in groups two and three (Appendix C.8.2). Furthermore, in both groups MCV was found to be inversely related to MCHC. Percent ΔBV was positively strongly related to percent ΔPV, which, in turn, was also strongly positively related to percent ΔPV. The detected positive correlation between percent ΔBV with percent ΔPV and percent ΔTPP is displayed by the regression equations in group two with the exception of the percent ΔBV and percent ΔTPP regression equation in group three (Appendix C.10.2).

Lastly, as biochemical constituents and BV compartment changes were not found to discriminate significantly, the formation of a multiple regression equation was not applicable. This may have been due to the great sensitivity of the blood constituent changes in terms of posture, type and intensity of exercise.

**Table 4.7: Mean measured concentrations of selected blood and plasma electrolytes of the three subject groups (n=24).**

| Variables | Group 1 | | Group 2 | | Group 3 | | Total Group | |
|---|---|---|---|---|---|---|---|---|
| | pre | post | pre | post | pre | post | pre | post |
| Hb ($g.dl^{-1}$) | - | - | 13.87 | 13.87 | 14.20 | 14.31 | 14.03 | 14.09 |
| | - | - | (0.43) | (0.57) | (0.89) | (1.06) | (0.70) | (0.85) |
| Hct (%) | - | - | 36.86 | 36.81 | 37.27 | 36.93 | 37.06 | 36.87 |
| | - | - | (1.53) | (1.57) | (2.50) | (2.62) | (2.01) | (2.09) |
| Glucose ($mmol.\ell^{-1}$) | 4.30 | 5.02 | 4.16 | 4.85 | 5.76 | 5.26 | 4.67 | 5.04 |
| | (0.68) | (0.54) | (0.85) | (1.07) | (0.71) | (0.71) | (1.07) | (0.76) |
| TPP ($g.\ell^{-1}$) | 81.5 | 85.6 | 77.5 | 77.3 | 79.4 | 79.1 | 79.47 | 80.4 |
| | (2.50) | (4.03) | (3.16) | (3.62) | (6.24) | (4.07) | (4.32) | (5.15) |
| Na ($mmol.\ell^{-1}$) | 144.3 | 146.1* | 139.2 | 139.3 | 140.1 | 141.8 | 141.25 | 142.13 |
| | (1.68) | (1.16) | (1.38) | (2.06) | (1.55) | (1.72) | (2.72) | (3.21) |
| K ($mmol.\ell^{-1}$) | 4.2 | 4.2* | 3.73 | 3.78 | 3.73 | 3.70 | 3.89 | 3.86 |
| | (0.24) | (0.22) | (0.24) | (0.22) | (0.29) | (0.22) | (0.33) | (0.29) |
| Cl ($mmol.\ell^{-1}$) | 109.3 | 112.0* | 107.1 | 108.6 | 107.7 | 110.5 | 108.0 | 110.2 |
| | (2.13) | (1.67) | (1.88) | (1.99) | (1.90) | (2.07) | (2.12) | (2.30) |
| Mg ($mmol.\ell^{-1}$) | 1.02 | 1.04** | 0.99 | 0.96 | 0.89 | 0.91 | 0.96 | 0.96 |
| | (.038) | (.053) | (0.08) | (0.08) | (0.07) | (0.06) | (.022) | (.025) |

Values are expressed in mean (SD); *: n=6; **: n=5

**Table 4.8: Mean(±SD) of measured and derived percent Hct, Hb, TPP and blood volume compartment changes of groups two and three (n=16).**

| Variables | Group 2 | Group 3 | Total Group |
|---|---|---|---|
| Hb | $8.75 \times 10^{-3}$ (2.98) | 0.743 (1.87) | 0.37 (2.43) |
| Hct | -0.1 (2.87) | -0.332 (2.11) | -0.21 (2.44) |
| TPP* | -0.17 (1.63) | 1.418 (2.80) | 0.50 (2.26) |
| BV | 0.028 (3.23) | -0.8 (2.00) | -0.38 (2.62) |
| MCV | 0.085 (0.99) | 1.71 (1.16) | 0.95 (1.34) |
| RCV | -0.080 (0.86) | -1.62 (1.21) | -0.88 (1.27) |
| PV | 0.15 (4.85) | -0.21 (2.78) | -0.03 (3.82) |
| TPP** | -0.022 (4.59) | 1.91 (3.90) | 0.80 (4.26) |

Values are expressed in: $\bar{X}$ (SD)

*: calculated from pre- and post-match TPP values, **: calculated from PV changes

**Table 4.9: Differences in pre- and post-match body mass, $T_{re}$, values and selected blood constituents within each group as derived from a paired sample t-test analysis (n=24).**

| | Parameter | Mean(SD) | NS | $p<0.05$ | $p<0.01$ | actual p | n |
|---|---|---|---|---|---|---|---|
| Group 1 | Body mass (Kg) | -0.58(0.13) | | | ** | $5.52807\times10^{-6}$ | 8 |
| | $T_{re}$ (°C) | +0.75(0.35) | | | ** | $5.14887\times10^{-4}$ | 8 |
| | Na(mmol.$\ell^{-1}$) | +2.16(1.60) | | * | | 0.021177 | 6 |
| | K (mmol.$\ell^{-1}$) | -0.06(0.05) | | * | | 0.025031 | 6 |
| | Cl (mmol.$\ell^{-1}$) | +2.33(2.33) | NS | | | 0.058293 | 6 |
| | Mg (mmol.$\ell^{-1}$) | +0.13(0.47) | NS | | | 0.560041 | 5 |
| | Glucose(mmol.$\ell^{-1}$) | +0.71(0.57) | | * | | 0.027590 | 6 |
| | TPP (g.$\ell^{-1}$) | +4.66(1.63) | | | ** | $9.16747\times10^{-4}$ | 6 |
| Group 2 | Body mass (Kg) | -0.2(0.2) | | * | | 0.025463 | 8 |
| | $T_{re}$ (°C) | +0.71(0.26) | | | ** | $1.23512\times10^{-4}$ | 8 |
| | Na (mmol.$\ell^{-1}$) | +0.12(1.72) | NS | | | 0.843605 | 8 |
| | K (mmol.$\ell^{-1}$) | +0.05(0.25) | NS | | | 0.598331 | 8 |
| | Cl (mmol.$\ell^{-1}$) | +1.5(1.77) | | * | | 0.047944 | 8 |
| | Mg (mmol.$\ell^{-1}$) | -0.02(0.05) | NS | | | 0.155648 | 8 |
| | Hb (g.dl$^{-1}$) | 0(0.41) | NS | | | 1 | 8 |
| | Hct (%) | -0.05(1.03) | NS | | | 0.895587 | 8 |
| | Glucose(mmol.$\ell^{-1}$) | +0.68(0.96) | NS | | | 0.082785 | 8 |
| | TPP (g.$\ell^{-1}$) | -0.12(1.24) | NS | | | 0.784882 | 8 |
| Group 3 | Body mass (Kg) | -0.65(0.28) | | | ** | $3.71875\times10^{-4}$ | 8 |
| | $T_{re}$ (°C) | +0.75(0.19) | | | ** | $1.13281\times10^{-5}$ | 8 |
| | Na (mmol.$\ell^{-1}$) | +1.75(1.66) | | * | | 0.209376 | 8 |
| | K (mmol.$\ell^{-1}$) | -0.03(0.35) | NS | | | 0.775811 | 8 |
| | Cl (mmol.$\ell^{-1}$) | +2.75(2.25) | | * | | 0.010633 | 8 |
| | Mg (mmol.$\ell^{-1}$) | +0.02(0.08) | NS | | | 0.505239 | 8 |
| | Hb (g.dl$^{-1}$) | +0.11(0.26) | NS | | | 0.276428 | 8 |
| | Hct (%) | -0.33(0.68) | NS | | | 0.205736 | 8 |
| | Glucose(mmol.$\ell^{-1}$) | -0.5(0.82) | NS | | | 0.131425 | 8 |
| | TPP (g.$\ell^{-1}$) | -0.83(4.11) | NS | | | 0.641215 | 6 |

## 4.5 Questionnaires

### 4.5.1 Tennis participation and parental response to questionnaires

Group one commenced tennis playing (Table 4.10) at a significantly younger age than group two and three ($p<0.05$). An analysis of variance also revealed significantly ($p<0.05$) more years of playing tennis in group one when compared to group two.

Higher percentage frequencies were found in group one and three with respect to paternal participation in tennis (Table 4.11) when compared to group two. Two subjects from group three reported the use of an extrinsic type of reward system. The remaining 91% of the total group therefore relied solely on intrinsic factors. Furthermore, a higher percentage frequency in pre-competition behavioral problems was displayed by the subjects in group three, compared to the lower incidence of these symptoms in group one and two.

Seventeen (16.6) percent of young tennis players reported possessing asthma in this study.

### 4.5.2 Acclimatization

All groups reported to perceive their activities as high in intensity ( >75%) during the last two months. With respect to frequency of play (times/week), the majority of the subjects reported at least three to four exercise sessions per week performed during afternoon hours (14h00-16h00). These included tennis as well as a variety of other sport activities within the school context.

### 4.5.3 Trait-state anxiety

Mean and standard deviation of the trait anxiety questionnaire responses are presented in Table 4.12. It can be seen that all groups experienced moderate trait anxiety levels ($\bar{X}$= 21, SD=4.1).

Table 4.13 shows that a significant relationship was only found between trait anxiety and somatic state anxiety response (r=.92) and between self confidence and cognitive state anxiety response (r=.92). Responses in both trait and state anxiety were obtained from only six subjects of group three, since only these subjects participated in official competition after this study and since a state anxiety questionnaire was only available for this age group ($\bar{X}$= 14.8yrs). Interestingly of the six subjects all of whom were defeated, four possessed the reported pre-competitive behavioral symptoms and two obtained an extrinsic reward from their parents. Although the behavioral stress symptoms and performance outcome may have been interpreted as indicators of high levels of competitive stress, high trait and state anxiety responses were not present in the questionnaires.

**Table 4.10: Tennis participation of the three groups of subjects (n=24).**

| Variables | Group 1 (n=8) | Group 2 (n=8) | Group 3 (n=8) | Total Group (n=24) |
|---|---|---|---|---|
| Present age (yrs) | 12.8(0.4) | 12.8(0.6) | 14.8(0.5) | 13.3(1.3) |
| Starting age (yrs) | 6.8(0.8) | 9.2(1.0) | 9.2(1.2) | 8.2(1.7) |
| Playing experience (yrs) | 5.9(1.5) | 3.6(1.2) | 5.6(2.7) | 5.0(2.1) |

Values are expressed as means(SD)

**Table 4.11: Asthma, father's tennis participation and related competitive anxiety variables of the three subject groups (n=24).**

| Variables | Group 1 (n=8) | | Group 2 (n=8) | | Group 3 (n=8) | | Total Group (n=24) | |
|---|---|---|---|---|---|---|---|---|
| | f | %f | f | %f | f | %f | f | %f |
| Asthma | 2 | 25 | 1 | 13 | 1 | 13 | 4 | 17 |
| Paternal tennis participation | 5 | 63 | 0 | 0 | 4 | 50 | 9 | 38 |
| Pre-competition behavioural problems | 1 | 13 | 2 | 25 | 4 | 50 | 7 | 29 |
| Intrinsic(RS) | 8 | 100 | 8 | 100 | 6 | 75 | 22 | 92 |
| Extrinsic(RS) | 0 | 0 | 0 | 0 | 2 | 25 | 2 | 8.3 |

f: frequency; %f: percentage frequency; (RS): reward system

**Table 4.12: Mean(±SD) trait anxiety scores (n=24).**

| | Group 1 | Group 2 | Group 3 |
|---|---|---|---|
| Low trait anxiety | - | - | - |
| Moderate trait anxiety | 19.0(3.7) | 18.2(4.4) | 21.0(4.1) |
| High trait anxiety | - | - | - |

Means and standard deviation for trait anxiety scores as achieved in the Illinois Competition Anxiety Test. The lowest score is 10 and the highest 30. Children with trait scores of greater or equal to 25 (upper 25 percentile) were classified as high trait anxiety while subjects with scores of less than or equal to 15 (lower 25 percentile) were classified as low trait anxiety. One way analysis of variance on group means revealed no significant difference between the groups ($p < 0.05$).

**Table 4.13: Relationship between trait anxiety and state anxiety subcomponents of group three (n=6).**

| | Correlation coefficient (significance level) | | |
|---|---|---|---|
| Parameter | Self confidence | Somatic state anxiety | Cognitive state anxiety |
| 1. Trait anxiety | -.1106 (.8348) | .9292 (.0073*) | .0684 (.8975) |
| 2. Self confidence | | .0287 (.9750) | .9290 (.0074*) |
| 3. Somatic state anxiety | | | -.1692 (.7486) |
| 4. Cognitive state anxiety | | | |

The competitive state anxiety inventory (CSAI-2) comprises 27-items which, when scored, results in cognitive anxiety, somatic anxiety, and confidence subscale scores ranging from a low of 9 to a high of 36. Further analysis revealed no significant differences between the subcomponents of the state anxiety level in group three ($p<0.05$).

*: $p<0.05$

# CHAPTER FIVE

# DISCUSSION

5.1 MORHOLOGICAL OBSERVATIONS
5.2 QUESTIONNAIRES
5.3 PHYSIOLOGICAL OBSERVATIONS
5.4 BIOCHEMICAL OBSERVATIONS
5.5 FLUID REPLACEMENT NEEDS

## 5.1 Morphological observations

### 5.1.1 Anthropometric and physical characteristics

The very strong positive correlation between body mass and body stature ($r = .97$, $p < 0.001$) (Appendix C.8.1) confirms the findings of Copley(1980). However, due to the great variability in growth rates, the use of body mass to predict morphological and physiological parameters would not be considered as appropriate for the subjects investigated in this study.

As can be seen from Table 4.1, the players of above average tennis ability (group one) were on the average, taller than the players of average tennis ability (group two). However, this difference was insignificant and thus could not be attributed to continuous tennis participation, especially as the subjects monitored in this study were possibly experiencing developmental growth spurts.

### 5.1.2 Skinfolds and percentage body fat

The differences in upper and lower body skinfold thicknesses between the groups were insignificant ($p>0.05$) and all three groups exhibited low percentage body fat levels (Table 4.1). These findings must, however, be evaluated in terms of the specific formula utilized (Yuhasz, 1974) and according to the increased ratio of lean body mass to body fat mass which is characteristic of the growth period of the subjects. Differences in fat deposition between the subjects could also be attributed to genetic predisposition and diet (Mc Ardle et al., 1991).

## 5.2 Questionnaires

The questionnaires administered to the subjects attempted to describe the response and attitude of the young tennis players towards competition. Although the tension and stress associated with competing in an individual sport such as tennis is far greater than that of a team sport (Griffin, 1972), the subjects, on average, experienced moderate trait anxiety in response to competitive situations (Table 4.10). Only one player (KW) scored a value which indicates a level of high trait anxiety which according to Roberts(1986), can result in a decrease in the athlete's physical energy level, performance level, as well as his enjoyment level. The parental questionnaire of this subject, confirmed that the child was experiencing certain behavioral problems prior to competition such as low self esteem, anxiety, moodiness, and decreased appetite. It is possible that this individual's greater anxiety may also have resulted from stress created by parents, coach or league administrators emphasizing the importance of winning (Doganis, 1988; Martens et al., 1990). Inability to meet the demands of competitive situations in order to obtain the favourable outcomes that were perceived as more important to this subject than to the other competitors (Martens, 1988), may also have played a role.

It is well described that subjects with moderate trait anxiety responses will respond with moderate state trait anxiety responses (Martens et al., 1990) and that significant relationship exists between trait anxiety and state somatic anxiety level (Maynard and Howe, 1987; Karteroliotis and Gill, 1987). This was confirmed by the findings of this study in which a correlation of .92 ($p<0.01$) was obtained between trait anxiety and somatic state anxiety (group three) (Table 4.13). According to the inverted -U theory (Yerkes and Dobson, 1908), the moderate state anxiety levels of the subjects in group three, however, may not have been optimal for succesful performance. In addition, the physiologic: factors and/or lack of technical and tactical skills may have contributed to the poor achievement levels of this group, who were described as 'under-achievers' by their coach.

The mean trait anxiety levels of the three groups and the mean state anxiety levels of the six subjects in group three are within the normal moderate anxiety range for trait and state anxiety levels, according to age group as given by Martens(1977). However, more attention ought to be paid to the individually stressed child. In the case of this child, the theory of ZOF (Hanin, 1986) explaining the anxiety-performance relationship may be more appropriate. Early signs of stress should be recognized and stress management procedures followed (Martens et al., 1990).

It was also recorded that seven subjects came from families in which the father participated in tennis (Table 4.11). This supports the notion that the sport or form of physical activity played by the father plays the greatest motivational role in a child's participation and selection of dominant sporting activity, especially with respect to boys (Doganis, 1988).

In this study the use of extrinsic reward systems (e.g., material, money) was absent (Table 4.11). This observation eliminates the possibility described by Sage(1977) that the substitution of intrinsic motives by extrinsic ones can lead to withdrawal from the sport, due to the absence of the initial intrinsic motives, when extrinsic motives cease.

On the other hand, based on the contention of Doganis(1988) that withdrawal of extrinsic rewards or changing one's perceptions of the significance of these rewards, can reduce levels of state anxiety, the only moderate anxiety levels of the subjects in this study may have been the result of the absence of an external reward system.

The most significant finding of the questionnaires administrated to the three groups, was the absence of high state and/or trait anxiety levels. This is of particular relevance in assessing physiological responses to 90 min of tennis play in these subjects. As these levels were only moderate, it is unlikely that mean HR and/or $\dot{V}O_2$ levels during play would have been greatly influenced by the psychological status of the players.

## 5.3 Physiological observations

### 5.3.1 Heart Rate

Based on the method described by Siegel.(1988), the mean 13.3 years of age and the mean HR response of 145bpm in these subjects, resulted in the derivation of a mean intensity of effort of 55% of the age adjusted HRmax reserve. This is not within the 60%-85% range of HR max reserve recommended by the ACSM (1978) for optimal cardiovascular development.

As studies have pointed to intensity of exercise as the single most important consideration in cardiorespiratory conditioning (Faria, 1970; Sharkley and Hollenman, 1967; Shephard, 1968) and it has been stated that the same exercising components (intensity, duration and mode) that are applied to adults should be applied to children if the aim is to develop the cardiovascular fitness in children (Bar-Or, 1983; Rowland, 1985; Ross and Gilbert, 1985; Mc Keag, 1991), a fundamental criterion for optimal cardiovascular development was not accomplished.

However, an important finding of this study was the relatively low time period spent within the THRR during the 90 min of tennis play (Table 4.4; Figure 4.1). Approximately one third of the total duration of tennis play (i.e. 24.3 min, SD= 21.3min) was played within 60-85% of HRmax reserve. The ACSM (1978) recommends that the duration of continuous aerobic activity within the HR range should range between 20 and 60 min. It must, however, be taken into account that the mean 24.3 min period of play during which the HR range was between the recommended 60-85% of HRmax reserve, was spread over 90 min of match play and can therefore not be equated with 20 min of continuous activity within the prescribed HR range. In this respect, it is questionable whether tennis meets the ACSM (1978) criterion of duration for optimal cardiovascular development.

One could attribute this low HR response to the fact that tennis matches were simulated during 2nd, 3rd, 4th and 5th sessions, and that the accompanying lack of motivation in the subjects may have resulted in subjects not playing at maximal effort, as would have been the case in real competition. A finding which is discrepant to this theory is, however, the mean HR response of group one who participated in a highly competitive Round Robin Tournament. In one of the tennis players of group one (i.e. subject KR, ranked number 10 in his age group in South Africa) a HR of less than 60% of HRmax reserve was recorded for 57 min and for only 25 min of play, was the HR range within the target zone of 60-85% of HRmax reserve.

In the interpretation of the low HR response two additional factors should be taken into account: Firstly, it is well described that during an identical time period in which the same amount of work is performed, the mean HR during intermittent exercise is higher than during continuous exercise (Astrand et al., 1960). Secondly, at a given submaximal $\dot{V}O_2$, the HR during arm exercise may be 10-50 bpm higher than that during leg exercise (Asmussen and Hemingsen, 1958; Clausen et al., 1973).

With regard to mode, the recommendation of the ACSM (1978) is that the exercise type include primarily large muscle group activity. As tennis is a combination of locomotor activity involving the large muscle groups of the lower extremity and non-locomotor upper limb activity which may not recruit as large a proportion of large muscle groups, it is to be questioned whether the mode of exercise is suited to cardiovascular development. However, as the intensity of effort is dependent on the amount of locomotor activity in a specific game and is not consistent, it cannot be standardized and is highly variable between games and between individuals.

From the findings of this study, it can therefore be concluded that during simulated and competitive tennis match play, this sport does not appear to meet the ACSM (1978) requirements for optimal cardiovascular development and does not support the classification of the Committee of Sports Medicine (1988) that tennis is a "strenuous child sport". Due to the limited sample size of the players participating in the highly competitive Round Robin Tournament (only 8 subjects of the 24 subjects monitored), further work is, however, required to establish whether highly competitive tennis match-play in young tennis players meets the requirements of ACSM (1978) and supports the classification of the Committee of Sports Medicine (1988). Although studies of Seliger et al.(1973), Dorherty(1982) and Bergeron et al.(1991) indicate that this is not the case, these studies were performed on adults and not on children.

It is of interest that the mean HR response during tennis play recorded in this study (145bpm, SD=16bpm) (Table 4.5), is identical to mean adult male HR responses in the study of Bergeron et al.(1991) which was performed for the same duration of tennis play, but under more temperate environmental conditions (ambient temperature= 17°C). With the exception of the work reported by Seliger et al.(1973), the recorded HR response in the young subjects monitored in this study was lower than those previously reported in adults (Dawson et al., 1985; Elliot et al., 1985; Therminarias et al., 1991). In view of the fact that the above studies [with the exception of the study of Dawson et al.(1985) in which the WBGT index was 30.9

and relative humidity, 6[illegible] were performed in a temperate environment, an overiding factor to wh[illegible] higher HP response may be attributed, is the greater mean body mass of adults and therefore greater muscle mass recruited, when compared to that in children.

The findings of this study, however, conflict with those of Friedman et al.(1984) and Elliot et al.(1985). It is possible that this contradiction may have been due to the lack of standardization of the method used to predict the percent of HRmax reserve. The calculation of a particular fraction of the HRmax may result in a significant overestimation of exercise intensity when the resting HR (the non- zero value of resting HR) is not taken into account (Davies and Convertino, 1975). The method used to predict HRmax was not specified in the studies.

HRmax can vary from 180 to 234 bpm in healthy children (Cumming and Hnatiuk, 1980). It is therefore recommended that the commonly used formula (HRmax= 220-age) be re-examined in terms of its application to children.

This study took into consideration the degree of the effort exerted by the players, the age of the players and the environmental conditions in which the study took place. Absence of control for age and environmental conditions is, however, evident in the previous studies of Friedman et al.(1984) and Seliger et al.(1973).

Within the limited scope thereof, a limitation of this study was that it was not possible to measure precisely each individual's resting HR and monitor HR response to [illegible]ndardized increases in workload in a laboratory. It was therefore necessary to rely on estimation of HRmax reserve according to the recommendations of Siegel(1988) which takes into account age in the prediction of resting HR and HRmax of the subjects.

Besides the exercise mode, duration and intensity, other underlying mechanisms which may influence the HR response, include environmental conditions and emotional status of the subjects.

It is well documented that environmental temperature has an indirect effect on HR; the increased $T_c$ excites the heart muscle with a resulting increase in HR (Guyton, 1986). As the environmental conditions in this study (Table 4.2) were be classified as temperate, it is assumed that a substantial effect on the HR response, was not applicable.

It is well known that emotional stress can also effect the HR response to exercise, with children showing greater rise in HR than adults (Saris, 1986). As the subjects of this study subjects were classified as experiencing moderate levels of anxiety according to the anxiety questionnaire responses (Table 4.12), this factor is unlikely to have contributed significantly to the HR response in this study.

With regard to the HR data obtained in this study it can thus be concluded that the moderate mean recorded exercise intensity of 55% of the age adjusted HRmax reserve, the relatively low time period spent within the target HR range and the intermittent exercise mode are of overriding significance and appear to indicate limited possibilities for optimal cardiovascular development during tennis play.

### 5.3.2 Oxygen consumption

In this study, energy cost was determined from direct measurements of $\dot{V}O_2$ during tennis play. Previous studies (Kozar and Hnusicker, 1963; Skubic and Hodgkins, 1965; Stamford, 1986) however, relied on measurement of HR only during play and compared the HR-$\dot{V}O_2$ relationship established during steady state exercise on a laboratory ergometer to estimate corresponding $\dot{V}O_2$ during play. This, together with

the fact that all previous reports of $\dot{V}O_2$ during tennis and related sport play (Kozar and Hnusicker, 1963; Skubic and Hodgkins, 1965; Stamford, 1986) measured the $\dot{V}O_2$ on adults only, places the validity of possible comparisons in question.

Generally, there is a linear relationship between $\dot{V}O_2$ and HR during steady state exercise (Astrand and Rodahl, 1977). Despite the fact that tennis was used as exercise mode, this relationship was confirmed by the findings of this study (r=0.69). According to Davies and Convertino(1975), the Karnoven(1957) method of prediction of exercise intensity (%HRmax), yields net $\%\dot{V}O_2$ max values which are not significantly different from measured values. Thus, the 55% of the age adjusted HRmax reserve in this study corresponds to an estimated 50-55% of $\dot{V}O_2$max.

The actual measurements obtained in this study revealed a mean HR of 145bpm and a mean relative $\dot{V}O_2$ of 21.4 $ml.Kg^{-1}.min^{-1}$, thus appearing to indicate that playing at a high percentage of $\dot{V}O_2$ max is not prerequisite for tennis match success and that a high level of metabolic stress is not imposed by this sport.

However, it has previously been documented that type of exercise (intermittent vs continuous) influences the HR and $\dot{V}O_2$ ($\ell.min^{-1}$) relationship (Klausen et al., 1986). As the calculated energy expenditure derived from the $\dot{V}O_2$ measurements taken during tennis play in this study is low in relation to the corresponding HR readings, these findings appear to support the assumption that during an identical time period in which the same amount of work is performed, the mean HR during intermittent exercise will be higher than during continuous exercise (Astrand et al., 1960) . Furthermore, at a given submaximal $\dot{V}O_2$, the HR during arm exercise may be 10-50 bpm higher than during leg exercise and the difference increases as the $\dot{V}O_2$ increases (Asmussen and Hemingsen, 1958; Clausen et al., 1973). The low energy expenditure relative to HR displayed during the tennis matches of this study could thus be attributed to the considerable amount of upperbody movement.

### 5.3.3 Energy cost

This study was a unique, first attempt to determine energy expenditure in children during tennis competitive play. Although the mean total energy cost during the 90min period of play of 1723 KJ in group one was slightly higher than the value of 1604 KJ found in group two and lower than the value of 1989 KJ in group three, the differences of the mean between the groups were, however, insignificant (Appendix C.7.1). Excluding group three from the comparison due to their higher mean body mass and taking into account the homogenous physical characteristics of groups one and two, the higher energy cost in group one as compared to group two could be attributed to the type of tennis game played by the subjects of these groups. Subjects in group one demonstrated an offensive tennis game with repeated and rapid net approaches, which require more physical effort than a defensive baseline game which was predominant in the games of the players in group two.

Energy expenditure values of 2160 $KJ.hr^{-1}$ (Copley, 1980), 1800 $KJ.hr^{-1}$ (Clarke, 1975) and 2640 $KJ.hr^{-1}$ (Seliger, 1968) during competitive tennis play, and 1860 $KJ.hr^{-1}$ in recreational tennis play utilizing the Douglas bag method (Sinclair and Goldsmid, 1978), have been reported. However, variability in energy expenditure could also be attributed to differences in a) the extent to which subjects exerted themselves, b) method used to determine energy expenditure, c) characteristics of each game, d) skill level of the players e) age (biochemical and physiological differences) and f) environmental conditions. Furthermore, the length of the rallies, the time spent retrieving balls, the frequency with which the player hits the ball and the relative playing abilities and efforts of the opponents are additional factors. Unfortunately, with respect to the above mentioned factors, meaningful inter-study comparisons in total energy expenditure is difficult and a great variance in intra-subject and intra-match energy expenditure, is likely.

Of interest is the fact that the differences between this study and the above studies disappear if the mean energy expenditure of all studies is expressed in $KJ.kg^{-1}.min^{-1}$ and an average adult body mass of 70Kg is assumed. The derived mean relative energy expenditure during tennis play is approximately 0.45 $KJ.Kg^{-1}.min^{-1}$ for all the above mentioned studies, which does agree with the mean relative energy expenditure of 0.44 $KJ.Kg^{-1}.min^{-1}$ (Table 4.5), derived from the $\dot{V}O_2$ measurements taken in this study.

Sinclair and Goldsmid(1978) using novice and intermediate racquetball players, found energy expenditure values of 3.420 $KJ.hr^{-1}$ and 3.600 $KJ.hr^{-1}$ respectively. Whereas Montpetit et al.(1987), when investigating squash and racquetball among intermediate players obtained a value of 2.820 $KJ.hr^{-1}$ and 2.520 $KJ.hr^{-1}$ respectively, these values are approximately triple (Sinclair and Goldsmid, 1978) and double (Montpetit et al., 1987) the values of this study and they suggest that tennis produces lower metabolic demand in comparison to squash and racquetball.

In this study the question arises, as to whether the children's metabolic rates would have been higher in a more uncomfortable environment. The dominant opinion in the literature would support this (Dimri et al., 1980; Fink et al., 1975; Callaham, 1979; Brooks et al., 1971). It can be hypothesized that the individual's discomfort in the warm and hot environment may have resulted in more extraneous movements being performed.

A further question requiring consideration in interpreting these results, is the effect of heat acclimatization levels. As metabolic rates during submaximal exercise in the heat have not been reported to be influenced by subjects' heat acclimatization levels (Young, et al., 1985), it is not considered necessary to include this factor in the interpretation of the $\dot{V}O_2$ results.

5.3.4 $T_{re}$

The thermal stress in this study classified according to the average WBGT index, would be represented by the amber flag for group one and green flag (ACSM, 1985) for groups two and three. It thus falls into the low to low moderate category of heat stress. A shortcoming of the WBGT index is, however, that if significantly different clothing is worn, the potential for heat exchange and the empirical relationships that are the basis for the recommendations for the WBGT index are altered (Santee and Gonzalez, 1988). The environmental conditions presented mild effects on the thermoregulatory mechanism of the subjects (Table 4.2). The variability of ground temperatures despite relative consistency in WBGT index, indicates a possible shortcoming of this index in reflecting thermal load. This applies particularly in the game of tennis which does not incorporate a great degree of locomotor movement.

The $T_{re}$ response in this study showed an increase of 0.73 °C (Table 4.4). Post-matches $T_{re}$ did not differ significantly between the groups, although group one spent relatively more time within the THRR of HRmax reserve. A possible explanation is that the high tennis ability level of group one, accompanied by enhanced physiological adjustments, may have resulted in a reduced physiological stress response, including $T_{re}$, during tennis play.

The use of relative exercise intensity (%$\dot{V}O_2$max), rather than actual metabolic rate (absolute exercise intensity), removes most of the inter-subject variability for the $T_c$ during exercise (Astrand, 1960). In this study it was shown that post-match $T_{re}$ is related to the intensity of exercise (HR) in subject group one (Appendix C.10.1). The finding that post-match $T_{re}$ is related to the exercise metabolic rate is in agreement with the laboratory findings of Saltin and Hermansen (1966) and field studies of Davies(1979). The failure to detect the same relationship in the other

subject groups, could be explained by the displayed variability in competitive performance between the groups. Only the subjects in group one faced a real competitive situation (i.e. playing in a Round Robin Tournament). This variability in actual performance of the subjects, could also explain the limited relationships found between physiological and metabolic variables within the total group (Appendix C.10.1).

Since the increase in $T_{re}$ is proportional to the relative exercise intensity (%HRmax) and is according to Nielsen(1938), Lind(1963) and Nadel and Horvath(1970), nearly independent of environmental temperature over a fairly wide range, the mild $T_{re}$ response of the subjects in this study may be dependant upon a) the low metabolic rate of the tissues involved b) the magnitude of blood flow and c) the temperature gradients between contiguous body regions. According to these contentions and assuming that temperature within the active skeletal muscle exceeds $T_c$ (Saltin, et al., 1970; Saltin and Hermansen, 1966) and the temperature within the inactive muscles does not increase (Aikas, et al., 1962), the larger the mass of inactive muscle in the subjects of this study, the higher the rate of heat exchange between the active and inactive muscle mass. In addition, the resting intervals between the tennis play, may have provided a means of heat dissipation to the tennis players. Thus, blood flow magnitude during the rest periods and the observed low intensity of exercise may have favoured heat dissipation and a mild $T_{re}$ response in these young subjects during tennis play.

Investigations performed by Bar-Or et al.(1980), Drinkwater and Horvath(1979), have indicated that the level of mild dehydration (mass loss) and the sweating patterns in children and adults are not highly correlated with post-match $T_{re}$. In all subject groups in this study, the level of dehydration developed during the 90 min of competitive tennis play was unrelated to post-match $T_{re}$ (Appendix C.9). This observation is in accordance with other studies on outdoor sports, during which

almost steady state type of exercise was performed (Maughan, 1985; Noakes et al., 1988, 1991b). If percent dehydration, which is a principal concern and component of prolonged steady state exercise, cannot explain the post race $T_{re}$ in marathon runners, it is to be questioned whether percent dehydration should be able to explain $T_{re}$ in young tennis players. Thus the findings of this study confirm the notion that the level of dehydration is not the principal factor determining the degree to which the $T_{re}$ rises during tennis play under the specific conditions of this particular study.

The relatively small influence of SR and therefore, percent dehydration on $T_{re}$ response, is also explained by another observation. The significantly higher percent dehydration levels of group one as compared to group two and three (1.01% vs 0.65% and 0.74% respectively), did not result in any significant difference in $T_{re}$ response between the groups.

### 5.3.6 Acclimatization

Although prolonged heat exposure during rest results in acclimatization (Machle and Hatch, 1947), it is known that regular heavy exercise in the heat is the most effective stimulus for developing heat acclimatization (Wenger et al., 1988).

The experimental sessions of this study took place from the beginning of February until the middle of March 1992. On 26th of February 1992, South Africa experienced a strong heat wave (Climate Information Service, 1992). As the subjects reported practising an average of 5.7($\pm$1.5) times per week for a mean duration of 1.8($\pm$0.4) hours per session during the months of December and January, and these playing sessions most frequently occurred between 14h00 and 16h00, the criteria for acclimatization appear to have been met by the subjects investigated in this study. The relatively low post match $T_{re}$, high SR, low HR and high TPP (particularly in group one) observed in the subjects (n=24), also appear to support this assumption.

## 5.4 Biochemical observations

### 5.4.1 Blood glucose

As fluid ingested by the players did not contain sugar at all, post-match blood glucose levels confirm previous findings of specific intensity and duration related blood glucose responses to exercise (Felig, 1975). Pre- and post-match blood glucose levels differ significantly in group one ($p<0.05$) and in group two ($p<0.1$), but was insignificantly different in group three ($p>0.05$). The highest increase was observed in the subjects of group one, who spent the greatest playing time within the THRR, whereas a milder increase was observed in group two which spent less time within the THRR. In turn, a small decrease in blood glucose levels occured in group three who spent the least time within the THRR. As percent changes in blood glucose levels of group two were not correlated significantly to percentage changes in PV ($p>0.05$), the possibility of hemoconcentration can be ruled out in this group. The unavailability of PV data in group one, limits the interpretation of the blood glucose response in this group.

As Copley(1980) included glucose in the fluid ingested by the players in his study, comparison of the findings of this study with those of this study is not warranted. Oseid and Hermansen(1971), Lehmann et al.(1981) and Berg et al.(1980) found a disposition towards a higher blood glucose concentration after exercise in children when compared to that of adults and this has been attributed to the possible age-dependent regulation of insulin metabolism (Oseid and Hermansen, 1971; Wirth et al., 1978). This indication may also be evident in the findings of this study when analyzing the age of the children. It was in the two younger groups of this study (one and two) that significant ($p<0.5$ and $p<0.1$ respectively) post-match blood glucose rises were evident as compared to the insignificant drop of post-match blood glucose levels in the older group three. Due to the influence of additional variables including

the intensity and duration of exercise, it is, however, not possible to ascertain the magnitude of the effect of age as a variable. This requires further investigation.

The absence of a significant decrease in blood glucose values may be indicative of adequate functioning of blood glucose regulatory mechanisms when tennis is played at a moderate exercise intensity and for a duration of 90min. From these results, it would appear that the metabolic demand was not high enough to result in liver glycogen depletion and subsequent need for carbohydrate supplementation.

### 5.4.2 Plasma volume changes

In this study MCV measurements were calculated based on MCHC obtained from the Technicon H* 2 system. This system has been found to be superior to other automatic systems as it is free from the MCHC artefact. Each red blood cell is measured in order to determine the MCV and consequently Hct measurement is considered more reliable when compared to other automated analysis methods (England, 1988).

Insignificant percentage change in PV was evidenced in group three. As significant increases in $Na^+$ concentration were present in this group, the high percentage change in MCV in this group, may be attributed to the increases in osmolality (Costill et al., 1974).

Contraction of intravascular volume has been reported to result from fluid losses due to sweating, low fluid intake and intra-cellular shift due to osmotic forces (Felig et al., 1982). Furthermore, the maintenance of PV and the distribution of fluid between the plasma and the interstitial space is largely dependent on the 'effective' oncotic pressure of the plasma proteins resulting from differences in protein

concentration (Harrison, 1985). Percentage changes in PV in groups two and three were highly correlated with percentage changes in TPP (Appendix C.8.2) in these groups, and the maintenance of the size of the PV compartment could thus attributed to the 'effective' oncotic pressure of the plasma proteins.

Although the mean percentage changes between pre- and post PV were insignificantly different, great variability between the subjects was observed. This heterogeneity in PV response could be explained by differences in exercise intensity and the physical fitness of the subjects (Harisson, 1985). It would, however, be attributed to the intermittent exercise mode as the rest intervals during tennis play provide the circulatory system with recovery periods during which homeostatic control can be achieved. However, the maintenance of PV may also have suggested effective reflex adjustments in children that compensate for transvascular shifts in PV.

Harrison(1985), believes that changes in RCV cannot be distinguished from MCV changes. Furthermore, an inverse relationship between MCHC and MCV has been reported by Costill et al.(1974), who also did not distinguish MCV changes from RCV changes. Although the same inverse relationship was found between percent changes in RCV and MCV in this study, this may simply be the result of the calculation procedures used in determining these volumes.

It is well known that the dehydration status of the subjects affects the intracellular and extracellular fluid compartments, due to free fluid exchange. It has been reported (Costill et al., 1976), that at low levels of body water loss, the water deficit primarily occurs at the expense of the extracellular space. The absence of a significant relationship between total dehydration (%) and percentage changes in PV and BV as well as the fact that children possess a higher extracellular/ intracellular space ratio when compared to adults may decrease the effect of the moderate amount of water loss in the extracellular body fluid compartment.

A highly positive relationship was evident between percentage changes of BV in groups two and three and percentage changes in PV (r=.98). Furthermore, percentage changes in PV in both groups were positively correlated with percentage changes in TPP (r= 0.85,p=<0,001). This thus may be indicative of TPP shifts between the intravascular and extravascular compartment and factors associated with these shifts. The pre-match TPP of group one 79.4($g.\ell^{-1}$), showed higher values than the suggested normal values for their age group 69.17($g.\ell^{-1}$) (Diem and Lentner, 1975). TPP ($g.\ell^{-1}$) values have been reported to increase with age (Diem and Lentner, 1975). The considerably higher pre-match TPP in group one when compared to group two and three suggest that training may elicit the same response (i.e. an elevated TPP which results in expansion of PV), regardless of age. This confirms previous findings of high resting TPP observed in well trained pubertal boys (Koch and Rocker, 1977). These results imply that the trained boys in this study (group one) showed the same adaptive changes with respect to TPP as endurance trained athletes (Koch and Rocker, 1977).

### 5.4.3 Plasma Electrolytes

From the findings of this study, it would appear that the metabolic activity of the subjects during tennis play resulted in minimal changes in the solute composition and water content of their bodies.

Firstly, if fluid shifts between the intracellular and extracellular compartments result primarily from movements of $Na^+$ and $K^+$ (Hubbard and Armstrong, 1988), it can be hypothesized that the insignificant changes of $Na^+$ and $K^+$ values in group two (Table 4.9), reflected the absence of the above mentioned fluid shifts in these groups of subjects. In contrast, groups one and three responded with significant rises in $Na^+$ and a mean fall in $K^+$. Concomitant fluid shifts between compartments may thus have taken place in the subjects of these groups.

In comparison to the acute exercise decrease in $Mg^{++}$ reported in adults (Hubbard and Armstrong, 1988), $Mg^{++}$ levels were maintained in all groups of this study. However, an unchanged $Mg^{++}$ value does not necessarily mean that no extracellular or intracellular changes took place, but can simply also mean an equal shift of electrolytes from the interstitial fluid space to both compartments. On the other hand, intensity and duration of the exercise, was not enough to elicit the previously reported drop in $Mg^{++}$ in adults (Hubbard and Armstrong, 1988).

Since $Na^+$ and $Cl^-$ are primarily responsible for the elevated plasma osmolality during dehydration (Senay, 1979), an elevation in plasma osmolality and/or decrease in PV will elicit vasopressin secretion, which reduces urinary water loss and stimulates thirst (Guyton, 1985). However, the insignificant percentage changes in PV in group two and three, the significant increases in $Cl^-$ in both groups, and the insignificant changes of $Na^+$ in group two, did not explain why group two consumed significantly larger water volumes than group three.

Lastly, the significant increase in plasma $Na^+$ and $Cl^-$ concentration after competitive tennis matches may also suggest that under the conditions of this study in particular, the workload intensity and amount of water ingested, supplementation of NaCl is contraindicated.

## 5.5 Fluid replacement needs

Sweat loss in a laboratory setting has been shown to be positively related to the absolute metabolic rate (Saltin and Hermansen 1966; Gullestad, 1975). In this study no relationship was evident between sweat loss and exercise intensity (HR ranges) (Appendix C.10.1). This may, however, have been related to the irregular, intermittent nature of the exercise and to the individual variations in sweat rates.

Despite significant decreases in mass loss (0.4 Kg) during 90min of tennis play ($p<0.05$), the mean 0.80% of dehydration could be considered mild when the fact that performance decrements only occur at >3% dehydration level (Noakes, 1992; Guyton, 1986). Furthermore, children possess a higher total water proportion when compared to adults (Diem and Lentner, 1975; Rudolph, 1991), which implies that the dehydration rate of 0.54 $\%.hr^{-1}$ is effectively lower than the equivalent rate of dehydration in adults. This is confirmed by the findings of this study which reveal that the mean recorded rate of water ingested in this study ($\pm$286 $ml.hr^{-1}$) was adequate to prevent decreases in PV (Table 4.8).

Since the subjects in group one exercised at relatively higher intensities and a higher heat stress index when compared to the other two groups, higher water losses and greater water ingested volumes would have been expected in this group (Table 4.3.1). This was confirmed by both the higher absolute sweat losses and the greater fluid replacement needs (Table 4.3.2) calculated in this group. It is, however, of interest that the subjects in this group, consuming a mean 318 $ml.hr^{-1}$, were better able to subjectively estimate their fluid replacement needs than those in groups two and three. This may simply have been the result of their relatively greater tennis playing experience.

In contrast, in group two there was a tendency to consume a considerably higher quantity of fluid than was necessary to replace sweat and respiratory water loss. These results obtained from group two support the notion that fluid intake tend to be greater amongst subjects exercising at low intensity (Noakes et al., (1988).

Particular attention should therefore be paid to those young tennis players who exercise at low intensities for prolonged periods. Since sweat rate and metabolic water released from glycogen breakdown are the major contributors to water deficit, it was estimated that extrapolation of the rate of drinking patterns in group two

would have resulted in overhydration, if the moderate exercise intensities and heat stress index were sustained for another 90min. This would may have direct consequences in terms of the well-being.

The AAP (1982; 1983) suggest the enforcement of freely-chosen drinking patterns in children. This may, however, have accelerated the onset of water intoxication in group two, had the duration of the matches been extended beyond 90min. Furthermore, the higher extracellular/ intracellular fluid space ratio of the child's body as compared to adults, should be considered as an additional factor predisposing children to water intoxication, when freely-chosen drinking patterns are enforced during exercise at moderate intensities.

The recommendation of the enforcement of drinking in children (AAP, 1982; 1983) stems solely from the findings of Bar-Or et al.(1980) who found that children are voluntarily dehydrated during prolonged mode of exercise (cycling). However, in the study of Bar-Or et al.(1980), the formula used to calculate percentage dehydration was based on mass loss only, metabolic fuel utilised, metabolic water produced by mitochondrial oxidation and the considerable water volume released from glycogen breakdown was not included. From the above, it can be concluded that, as has so frequently previously occurred in adults (Noakes et al., 1988; 1991b), fluid replacement volumes were overestimated in children. The main factor to which this may be attributed is the failure to take into account the considerable water released from glycogen breakdown, in the hydration status of the subjects.

In this study, had the water released from glycogen stores not been taken into account in the calculation of water deficit, the dehydration levels of the groups would have been double. Thus, a false mean value of 1.60% in total group would not be in accordance with the controlled moderate exercise intensity, the relatively high ingested volumes of fluid and with the insignificant PV changes of group two and three.

Thus the findings of this study do not support the contention of the AAP (1982; 1983) that voluntary dehydration is common among exercising children. In contrast, the subjects in this study, who were permitted non-enforced (voluntary) freely-chosen drinking patterns, and to whom water was readily available during the rest intervals between games, either correctly gauged (group one), overestimated (group two) or slightly underestimated (group three) their fluid replacement needs during tennis play.

Two questions arise at this point. Firstly, is the recommended fluid replacement of 300ml.hr$^{-1}$ of the AAP (1982; 1983) for a child possessing body mass of 40 Kg, appropriate to prevent dehydration in the sample of children investigated in this study?.

The most significant finding of this study was that the fluid replacement needs of the children were 313 ml.hr$^{-1}$ in group one, 204 ml.hr$^{-1}$ in group two and 268 ml.hr$^{-1}$ in group three. Based on the recommendations of the AAP (1982; 1983), fluid replacement needs (adjusted for body mass) in this study represent 344ml.hr$^{-1}$ for group one and two and 400ml.hr$^{-1}$ for group three. Therefore, when answering the question as to whether the findings of this study support the fluid replacement need recommendations of the AAP (1982; 1983), the answer is negative for all groups.

However, mass differences (and corresponding age differences) of the subjects investigated in this study is another important factor to which the large variance in fluid replacement needs can be attributed. When differences exist in body mass between groups, comparisons of fluid replacement are more appropriately expressed in ml.Kg$^{-1}$.hr$^{-1}$. It becomes apparent that between groups one and two, even with the same average body mass of 45.9 Kg, fluid replacement volumes differ when expressed in ml.kg$^{-1}$.hr$^{-1}$ (6.81 vs 4.44 respectively). This difference of the fluid replacement volumes adjusted for body mass are in accordance with the higher exercise intensity and environmental heat stress index experienced in group one when compared to group two. Fluid replacement needs in group three, when normalized

for body mass, fall between the fluid replacement needs of group two and one (5.02ml.$Kg^{-1}$.$hr^{-1}$). It would thus appear that variations in both exercise intensity and environmental heat stress index have an overriding effect on fluid replacement needs in children, when these are expressed relative to body mass (ml.$Kg^{-1}$.$hr^{-1}$).

The second question to be addressed is whether the enforced fluid replacement rates of 11.4 ml.$Kg^{-1}$.$min^{-1}$ prescribed by Mitchell et al.(1992), could be justified in the subjects of this study. However, if in the study of Mitchell et al.(1992) fluid balance estimations of the subjects were based on body mass loss, then the 0.907 Kg in mass loss, is too small to account for the considerable metabolic water released from glycogen breakdown, metabolic fuel utilized and the metabolic water produced by mitochondrial oxidation during the three hours of tennis play in the heat (WBGT index 27). As neither of these factors contribute to the fluid balance of the body, and neither of them were accounted for in the fluid balance estimations of the study of Mitchell et al.(1992), the high rates of 11.4 ml.$Kg^{-1}$.$min^{-1}$ may have resulted in net overhydration.

In conclusion, the findings of this study therefore reject the recommendation of one fluid replacement rate for children based only on body mass (AAP, 1982; 1983) and question the high rates recommended in the study of Mitchell et al.(1992). The major recommendation of the study is that a range of fluid replacement volumes, ought to be determined according to mass and exercise intensity of the subjects and the heat stress index on the day of the match play.

# CHAPTER SIX

# CONCLUSIONS

The natural setting and competitive atmosphere in which match-play took place in this study, provided some realistic insights to both the psychological and physiological response and tolerance of children to competitive tennis play.

Firstly, in adressing the primary problem examined in this study, calculation of their fluid replacement needs indicated that a) under warm environmental conditions (WBGT index= 13-19) and an exercise intensity of approximately 55% of the age adjusted HRm. reserve or 50-55% of $\dot{V}O_2max$, consumption of 4.44-6.81 ml.Kg$^{-1}$.hr$^{-1}$ or 200-360 ml.hr$^{-1}$ of water is recommended in young tennis players possessing a body mass of 45-53 Kg, in order to prevent harmful levels of dehydration. The initial hypothesis (1.5.1) set in chapter one is thus confirmed by the findings of this study.

With respect to the subproblems which were identified in chapter one, the following final conclusions can be reached.

6.1 The players investigated in this study did not exhibit signs of high trait and state anxiety which may have significantly influenced their physiological response to match-play.

6.2 Although tennis is characterized by sporadic high intensity exercise, the overall metabolic response (as reflected by HR, $\dot{V}O_2$ and estimating energy expenditure) imposed by tennis reflects a mean moderate-intensity of exercise and the discontinous type of tennis play does not appear to promote optimal rates of cardiovascular development in children. The classification of tennis as a strenuous child sport by the Committee of Sports Medicine (1988) is therefore questioned and the hypothesis set in chapter one (1.5.3) is accepted.

6.3 The $T_{re}$ changes are in accordance with the moderate exercise intensity experienced during tennis play. Thus, it can be concluded hyperthermia is not a common feature of children's tennis competition under the conditions of this study.

6.4 Relationships did not exist between $T_{re}$ and percent body fat, and range of exercise intensity with the exception of group one.

6.5 The blood glucose response indicates that the incidence of hypoglycaemia is not prevalent and does not support the need for CHO supplementation during 90 min of tennis play under the conditions of this study.

6.6 Based on the variabilty in blood volume compartment responses in this study the hypothesis set in chapter one (1.5.4) is accepted and is in accordance with the measured low exercise intensity exhibited in this study. The average PV maintenance, the positive relationship between percentage changes in TPP and percentage changes in PV and the significant higher control TPP values in group one as compared to the other two groups in this study, may indicate that regular moderate tennis exercise intensity results in the enlargment of the vascular volume.

6.7 The often reported (AAP, 1982; 1983) incidence of voluntary dehydration in children (Bar-Or, 1980) did not occur in this study and the hypothesis set in chapter one (1.5.2) is thus rejected. The subjects monitored their fluid needs adequately in order to prevent harmful dehydration levels (fig. 6.1).

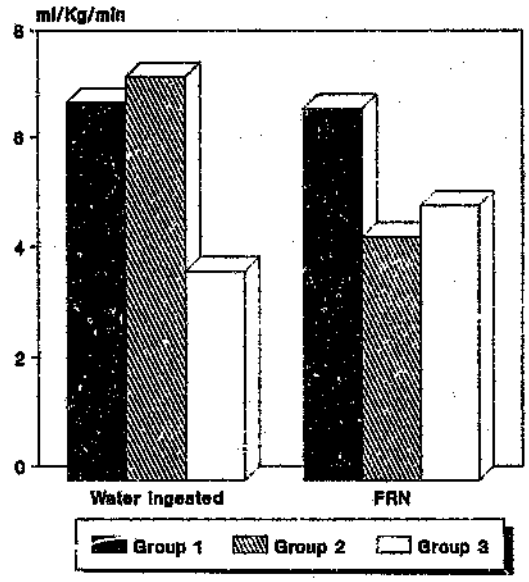



***Fig. 6.1. Calculated fluid replacement needs ($ml.Kg^{-1}.hr^{-1}$) and measured freely-chosen drinking patterns ($ml.Kg^{-1}.hr^{-1}$) of the three subject groups during a 90 min tennis match ($n=24$).***

This study, in attempting to examine the fluid replacement needs of the exercising young tennis player revealed, in contrast to the published existing theories of inferior behavioral and metabolic responses to the stress of exercise (Bar-Or, 1980; AAP, 1982; 1983;), that children are capable of responding adequately to the water needs of their body. It can be concluded that young tennis players respond within physiological limits, and that their thermoregulatory ($T_{re}$) and osmoregulatory (PV, electrolytes) systems functioned adequately in order to cope with the metabolic stress imposed upon their bodies.

What emerges from this investigation, is the individualistic nature of the physiological and psychological responses of children to exercise. This approves and signals the need for an interdisciplinary approach whenever study is undertaken of the variable nature of the developing child. Thus, in considering the child's mental and physical well being and in an attempt to understand and enhance performance, sports physiologists should link their forces with sport psychologists.

# CHAPTER SEVEN

# RECOMMENDATIONS

7.1 RECOMMENDATIONS FOR FURTHER RESEARCH

7.2 RECOMMENDATIONS TO SCHOOL TENNIS COACHES

## 7.1 Recommendations for further research

7.1.1 It is generally agreed that $T_{re}$ is related to relative metabolic rate in steady state exercise (Astrand, 1960; Saltin and Hermansen, 1966; Davies et al., 1976). However, the significance of other components such as negative force generation and the locomotion pattern of tennis play (especially that of novice tennis players) on $T_{re}$ response requires investigation. It is of interest to study whether these factors could better predict $T_{re}$ response during tennis play.

7.1.2 The relative contribution of the extracellular/intracellular compartments to dehydration in children when taking into consideration that children possess a higher extracellular/intracellular space ratio when compared to adults, requires examination.

7.1.3 Validation of the significantly higher TPP concentrations measured in the elite tennis players, requires investigation.

7.1.4 The possible significant difference between the natural environment and indoor laboratory environment, on fluid replacement patterns under the same exercise protocols in children and adults, ought to be examined.

7.1.5 The findings of this study indicate the need for the establishment of a nomogram which will incorporate mass, exercise intensity and heat stress index in the calculation of fluid replacement needs in children is a recommendation. This will be of practical relevance to coaches of children as takes into account body mass variability, percent effort and environmental conditions.

## 7.2 Recommendations to school tennis coaches

Physiological and biochemical factors:

7.2.1 Periodic drinking (approximately 50-90 ml every 15 min) should be encouraged during prolonged activities for a child possessing a body mass of 48 Kg under moderate tennis play and environmental conditions. The exact amount within this range varies according to the body mass, exercise intensity and environmental conditions. A heavier child exposed to higher exercise and environmental conditions will require high rates of fluid replacement, i.e. closer to the top of the range specified.

7.2.2 Glucose solutions are not necessary for fluid replacement in children playing tennis of 90min in duration and could be considered as fluid replacement forms, only to enhance fluid palatability and intestinal absorption, and therefore increase fluid replacement volume in tennis play of longer duration and more extreme environmental conditions, when compared to conditions of this study. Furthermore, under the conditions of this study, in particular the workload intensity, duration of the matches and amount of water ingested, it is recommended that supplementation of NaCl is contraindicated.

7.2.3 As can be seen from Table 4.5 the intersubject variability of the mean HR response during the matches, in the 13 subjects whom continuous HR data were obtained, ranged between 12-19bpm. Despite the start and stop nature and the periods of high-intensity exercise during tennis play, small fluctuations in HR response were exhibited, indicating a relatively consistent intensity of exercise. This further suggests that the form of a more persistent type of tennis practice i.e. continuous rallies from the baseline or non-stop exchange of strokes between player and coach at the net, could possibly stress the cardiovascular system of the child athlete. This is an encouraging finding for tennis coaches. Tennis coaches in search of ways to increase the cardiovascular fitness level of their young athletes within the sport, do thus not have to resort to other forms of prolonged continuous exercise. Coaches simply have to, during their practice sessions, include a non-stop type form of skill practice ensuring that the players continuously cover the tennis court. A limitation to the application of the above, is however, ability level of the players. A high ability tennis level is required to respond efficiently to this type of practice. Thus, this type of practice could not be applied to players of low and average tennis ability level. Consequently, other forms of prolonged, continuous exercise (running, bicycling) should be incorporated in the exercising regimen of the novice tennis player to promote cardiovascular fitness, by following the recommendations in terms of exercise intensity and duration, set by the ACSM (1978; 1990).

7.2.4 An understanding of the significance of each player's state and trait anxiety and the ability to distinguish between the different emotional states of each athlete is a fundamental prerequisite in coaching. This will enable the coach to utilize the most appropriate stress management strategies for each athlete. As Martens(1988) has stated "coaches coach individuals, not teams".

Finally, it is important for physical educators coaches and parents to be aware that physical fitness is the synthesis of many variables such as growth rates, gender, state of health, heredity, cardiovascular-respiratory fitness, physique, structure, motor function and many histochemical and biochemical factors, as well as psychological parameters. Since these factors are extremely variable, children's physiological and psychological response and tolerance to exercise is different for each individual child.

Educators, whether they be in the classroom or on the sport field, should accept a role towards children that is both protective and educational. Children are not small pictures of adults; they possess individual potential for physiological and psychological development and require a well rounded education including the opportunities for healthy physical development. Thus, children should be granted the opportunity to participate in physical exercise programmes which involve a wide variety of motor skills, and avoid early specialization. The primary responsibility of the sport's coach is to contribute towards a happy childhood; and to facilitate the optimal development of the child's personality. One must develop the personality of the child within the context of a healthy physical development and bear this in mind when organizing top-level sports for children. Finally, an educator's objective is to both use movement to guide children to reach the limits of their potential and to educate coaches as to what is enjoyable and beneficial for children. It is an educator's sensitivity which will determine the fine line between physical exercise being a challenge and physical exercise being detrimental to the child's well being.

# LIST OF APPENDICES

## APPENDIX A

**APPENDIX $A_1$:**

INFORMATION SHEET

DIVISION OF PHYSICAL EDUCATION
SPORT SCIENCE LABORATORY

Dear parent\Guardian

re: FLUID REPLACEMENT STUDY IN YOUNG TENNIS PLAYERS

Young adults playing tennis are often exposed to the combined stresses of exercise and environmental heat. These may place a marked strain on the thermoregulatory and hydration stata of their bodies and affect the well-being of the young adult. The purpose of my investigation is to therefore determine the fluid balance level in young tennis players.

In order to understand and determine your son's hydration status and physiological reaction to exercise in the heat, the following tests would have to be performed on your son:

(a) Measurements of height and skinfold thicknesses prior to the match and mass, body core temperature, before and after the match.
(b) Monitoring heart rate and the collection of samples of expired air during the match.
(c) The extraction of a 13 ml blood sample from subcutaneous antecubital vein by a qualified medical practitioner, before and after the match.
(d) The completion of a questionaire by both parent and player prior to the match.

The tests will not cause your son any harm, nor, in any manner, detrimentally affect his performance capabilities during the match.

The investigation will provide the data on which a Master's dissertation is to be based. It would therefore be greatly appreciated if you could please grant your consent and approval for your son to form part of this investigation. If your son is to form part of the investigation, we will provide you with feedback of the results obtained as well as any emerging findings and recommendations.

Yours faithfully

Kostas Kavasis
TEL: (011) 642-4267
Student undertaking research

Mrs. E.M. Futre
Supervisor
TEL: (011) 716-5721

# PARENTAL CONSENT FORM

## DIVISION OF PHYSICAL EDUCATION
## SPORT SCIENCE LABORATORY

I, _____ hereby grant permission for my son to participate in the investigation to be performed by Mr Kostas Kavasis of the Division of Physical Education of the University of the Witwatersrand.

I understand that it is to involve:

(a) Completion of the Illinois Competition Questionnaire
(b) Measurements of height and skinfold thicknesses prior to the match and mass, core body temperature, before and after the match.
(c) Monitoring heart rate and the collection of samples of expired air during the match.
(d) The extraction of a 13 ml blood sample from subcutaneous antecubital vein by a qualified medical practitioner, before and after the match.
(e) The completion of a questionnaire by myself.

I am aware of the following:

(a) Participation is voluntary, and refusal to participate will involve no penalty or loss of benefits to which the subject is otherwise entitled.

(b) The subject may discontinue participation at any time without penalty or loss of benefits.

SIGNED : _____

DATE : _____

INVESTIGATORS SIGNATURES : _____

: _____

# CHILD'S CONSENT FORM

## DIVISION OF PHYSICAL EDUCATION
## SPORT SCIENCE LABORATORY

I, _____ hereby agree to participate in the research to be performed by the Division of Physical Education of the University of the Witwatersrand.

I understand that it is to involve:

(a) Completion of the Illinois Competition Questionnaire.

(b) Measurements of height, skinfold thicknesses prior to and mass, body core temperature and blood pressure, before and after the match.

(c) Monitoring of heart rate and collection of expired air during the match.

(d) Extraction of a 13 ml blood sample from a subcutaneous antecubital vein by a qualified medical practitioner before and after the match.

The details of the above procedures have been explained to me in full. I am aware that the measurements will be taken before and after the match and will not have any harmful effect on my match or performance level.

I am aware of the following:

(a) Participation is voluntary, and refusal to participate will involve no penalty or loss of benefits me, to which I am otherwise entitled.

(b) I may withdraw from the study at any time, without penalty or loss of benefits.

SIGNED : _____

DATE : _____

INVESTIGATORS SIGNATURES : _____

: _____

**APPENDIX $A_2$:**

## ILLINOIS COMPETITION QUESTIONNAIRE

UNIVERSITY OF THE WITWATERSRAND
DIVISION OF PHYSICAL EDUCATION

Name .....

Directions: Below are some statements about how persons feel when they compete in sports and games. Read each statement and decide if you HARDLY EVER, or SOMETIMES, or OFTEN feel this way when you compete in sports and games. If your choice is HARDLY EVER, blacken the square labeled B, and if your choice is OFTEN, blacken the square labeled C. There are no right or wrong answers. Do not spend too much time on any one statement. Remember to choose the word that describes how you usually feel when competing in sports and games.

A= Hardly-ever

B= Sometimes

C= Often

(1) Competing against others is socially enjoyable.

A B C

(2) Before I compete I feel uneasy.

A B C

(3) Before I compete I worry about not performing well.

A B C

(4) I am good sport when I compete.

A B C

(5) When I compete I worry about making mistakes.

A B C

(6) Before I compete I am calm.

A B C

(7) Setting a goal is important when competing.

A B C

(8) Before I compete I get a queasy feeling in my stomach.

A B C

(9) Just before competing I notice my heart beats faster than usual.

A B C

(10) I like to compete in games that demand considerable physical energy.

A B C

(11) Before I compete I feel relaxed.

A B C

(12) Before I compete I am nervous.

A B C

(13) Team sports are more exciting than individual sports.

A B C

(14) I get nervous wanting to start the game.

A B C

(15) Before I compete I usually get uptight.

A B C

Martens, (1977).

**APPENDIX $A_3$:**

## PARENTAL QUESTIONNAIRE

**UNIVERSITY OF THE WITWATERSRAND**
**DIVISION OF PHYSICAL EDUCATION**
**SPORT SCINCE LABORATORY**

Dear parent

Please would you complete the following brief questionnaire. This will be of assistance to us in the research which we are conducting. Your responses will be treated as confidential.

Many thanks
K. Kavasis

---

(A)

Son's name.....
Postal address.....
Son's date of birth.....
Number of years your son has been playing tennis.....

Has your son had any serious illnesses or injuries? .....
If yes, please specify.
.....
.....

---

(B)

Are there any other members of your family who participate in competitive sports?

No, no one in the family ☐

Yes

| | | |
|---|---|---|
| | | |
| | | |
| | | |

(C)

Is your son a member of a sports-team at school or elsewhere?

No ☐

Yes, within school ☐

Yes, representing the school ☐

Yes, other..... ☐

Yes, in the past, but no more ☐

---

(D)

Do you observe any problems in your son before or after a tennis match? eg. sleeping, eating habits, change of behaviour, desire to withdraw, anxiety, lack of concentration.

No ☐

If yes, please specify.....
.....

(E)

What kind of reward system do you use when your son wins a match or competition?

Oral ☐

Present or any kind of material gift ☐

Please give details :.....
.....
.....
.....

# APPENDIX B

**APPENDIX $B_1$:**

## FEEDBACK TO SUBJECTS

Sport Science Laboratory
Division of Physical Education
University of the Witwatersrand
Tel: 716-5716

Dear .....,

Thank-you very much for participating in our investigation on .../.../1992. We appreciate your co-operation.

The results which we obtained from you are as follows:

(a) Percentage body fat .....%

This is within the normal range for your age-group.

(b) Rectal temperature before match .....°C
Rectal temperature after match .....°C

The reason for taking rectal temperature, and not oral temperature, is that rectal temperature gives the truest reading of your core body temperature.

Your rectal temperature increased by ....°C as a result of the match. Rectal temperatures of 39 degrees celsius and above indicate hyperthermia. If the core body temperature rises above 40,5 degrees celsius, your central nervous system is affected, and heatstroke may occur.

The major factor determining the degree of rise in your core body temperature is intensity of exercise. The secondary factor is the environmental condition. As you will note from these results, your thermoregulatory adjustments were very efficient. Despite .... minutes of play in environmental temperature of ....°C, your body core temperature only rose by.....°C.

(e) Body mass before match .....Kg
Body mass after match .....Kg

As a result of the match, you lost ....Kg

However, you ingested .... liter of water during the match your nett mass loss is therefore .... + .... = .....liter

The reasons for this mass loss can include sweating, fuel breakdown and respiratory water loss. With reference to fuel, breakdown you must account for the breakdown of

muscle glycogen which also causes the release of water. This can account for up to 50 % of your mass loss. The water released by the breakdown of glycogen should not be considered as mass loss.

Thus, you only need to replace about 50% of ..... liter of water during 90 minutes of play which is equal to 50% of ..... liter per hour. The water that you need to replace was therefore .....liter per hour. (Remember that one tin of coke is containing 350ml).

**From the findings of this study, we have determined that your fluid requirements are ..... milliliters per hour if you play at this intensity of exercise and in a warm environmental condition.**

..... was a warm day (.... °C), and therefore you did not perspire as much as you would have if the temperature was higher. Therefore, you did not loose as much weight as you would have if it were hotter.

(f) Heart rate (HR) during the match is an indication of exercise intensity. Your target heart rate range can be calculated using this formula:

Step 1. 220 - age = HRmax.
STEP 2. (HRmax) - HRrest = trainable heart rate.
Step 3. (Trainable heart rate x .6) + HRrest = lower value
Step 4. (Trainable heart rate x .85) + HRrest + upper value.
Step 5. Target Heart Rate Range (THRR) = lower to upper values.

For example: To calculate the target heart rate range for a ten year-old child the steps are as follows.

Step 1. 220 - 10 = 210(HRmax)
Step 2. 210 - 90 = 120(trainable heart rate)
Step 3. 120 x .6 = 72 +90 = 162 =lower value.
Step 4. 120 x .85 = 102 + 90 = 192 =upper value.
Step 5. 162 - 192 = target heart rate range (THRR)

Exercising at this particular heart rate range, will improve the conditioning of your cardiovascular system. The duration spent within your target heart rate range was very short ....min and not enough to reach the expected cardiovascular training zone. Generally speaking tennis is not a sport for cardiovascular conditioning, since level of skill is the primary determinant of success during a match. Therefore, if you intend to improve your stamina, other types of sports should be included in your training programme. For example distance running or cycling, or any other exercise involving prolonged, continous exercise.

(g) We determined your oxygen consumption during the match collecting samples of expired air in our light-weight aluminium bags. From this we determined how intensively you exercised and how mutch energy you utilized during your game. On average your $\dot{V}O_2$ (oxygen consumption) was ....L/min which translates into an energy expenditure of ....Kjoules/min. We estimate you expended a total of ..... Kjoules in the 90 min.

(h) Hematocrit before match .....
Hematocrit after match .....

Hematocrit refers to the percentage of red blood cells in your blood. Red blood cells carry oxygen to the exercising muscles, and are therefore very important during exercise.

If you loose a lot of water due to excessive perspiration, this decreases the amount of water in your blood and usually your percentage red blood cells will rise. We can calculate the change in your plasma volume from your hematocrit levels and hemoglobin concentration. This is an indication of extracelluar fluid loss. At this intensity of exercise and with the amount of fluid that you ingested during the game, there was no significant change in your hematocrit levels.

(i) Blood glucose level before match .....mmol/L
Blood glucose level after match .....mmol/L

Blood glucose refers to the concentration of glucose in your blood. After the match your blood glucose levels are often higher because the liver is pouring hepatic (from the liver) glucose into the blood as an extra energy source and it takes a few minutes to adjust to the reduced demand after you have stoped playing. As you can see your blood glucose rose/dropped but not significantly and this indicates that liquids containing glucose need not be ingested during matches.

(k) The electrolytes sodium and potassium play an important role in fluid volumes within your body. They did not differ significantly from pre-match to post-match levels. Also they were within the normal range for your age group. This, together with the insignificant change in plasma volume (.... %) indicates that Round Robin Tournament play does not put a marked strain on your circulatory system.

Remember the following:

(a) If you play in a hot climate, or at the start of the summer sporting season, you should allow for 10-15 days for heat acclimatization to occur. During this time, you should gradually increase the intensity and duration of your exercise sessions and practice at the time of day at which you usually play your matches.

(b) You should wear light-weight porous clothing and drink fluids regularly during exercise $\pm$ 200-300ml per hour (if you weigh between 45-50 kilograms). This is dependant on the intensity of your game and the environmental conditions. You must remember to drink before and during the match. It is better to drink water every 10 minutes than to drink a greater quantity at less frequent intervals.

(c) If the environmental temperature is very hot (greater than 33 °C), you should decrease the intensity of your exercise, or should avoid exercising for longer than 30 minutes at a time.

I would like to advise you to keep this information for the purpose of comparison, in the event of your child participating in a similar investigation in the future.

Thank-you very much for your support in our investigation. Please feel free to contact me should you have any questions regarding the findings of the study.

**APPENDIX $R_2$:**

FEEDBACK TO COACHES

Sport Science Laboratory
Division of Physical Education
University of the Witwatersrand
Tel: 716-5716

Dear .....,

Thank you for participating in our investigation on ..../..../1992. We appreciate your co-operation. With regard to the results of the study, I should like to report as follows:

a)

| Percentage body fat | % |
|---|---|
| Subjects | ... |
| ..... | ... |
| ..... | ... |
| ..... | ... |
| ..... | ... |
| ..... | ... |
| ..... | ... |

All fall within the normal range for this age-group.

b) Rectal temperatures (°C):

| Subjects | pre | post |
|---|---|---|
| ..... | ... | ... |
| ..... | ... | ... |
| ..... | ... | ... |
| ..... | ... | ... |

The reason for taking rectal temperature, and not oral temperature, is that rectal temperature gives the truest reading of the core body temperature.

The rectal temperature of your players increased by .....°C as a result of the match. Rectal temperatures of 39 degrees celsius and above indicate hyperthermia. If the core body temperature rises above 40,5 degrees celsius, the central nervous system is affected, and heatstroke may occur.

The major factor determining the degree of rise in core body temperature is intensity of exercise. The secondary factor is the environmental condition. As you will note from these results, the thermoregulatory adjustments of your players were very efficient. Despite a mean of 90 minutes of play in environmental temperature ranging from ....°C to ....°C, the mean body core temperature only rose by ....°C.

c) Body mass changes (Kg) are included in the table below:

| Subjects | pre | post |
|---|---|---|
| ..... | ... | ... |
| ..... | ... | ... |
| ..... | ... | ... |
| ..... | ... | ... |

The reasons for this mass loss, include mainly sweating, fuel breakdown and respiratory water loss.

d) To determine sweat rate (SR, Liter/hour) we use the formula:

$$SR = \frac{\text{fluid loss in liters}}{\text{playing time in hours}}$$

To convert body mass to volume you need to consider the density of sweat which is 1,003

$$\text{Since Volume} = \frac{\text{mass}}{\text{density}} = \frac{\text{mass}}{1{,}003} = \text{liters}$$

You must however account for the breakdown of muscle glycogen which also causes the release of water. This will account for $\pm$ 50 % of your mass loss. It is therefore a great mistake to encourage players to replace all of the mass lost during exercise. This mistake has resulted in the increasing prevalence of water intoxication or overhydration in distance runners and can be vary dangerous. As a coach, you need to be very careful about this.

The sweat-rates (liter/hour) of your players are included in the table below:

| Sweat-Rate | Liter/hour |
|---|---|
| ..... | ..... |
| ..... | ..... |
| ..... | ..... |
| ..... | ..... |
| ..... | ..... |

The mean sweat rate of your tennis players was ..... Liter/hour. If we take the loss of metabolic water into account, this leads us to a mean fluid replacement need of ...Liter/hour, when the ambient temperature is ... °C.

e) Mean blood\glucose level before match .... mmol/l
Mean blood\glucose level after match .... mmol/l

Blood glucose refers to the concentration of glucose in the blood. After the match blood glucose levels are often higher because the liver is pouring hepatic (from the liver) glucose into the blood as an extra energy source and it takes a few minutes to adjust to the reduced demand after exercise. As you can see the mean blood glucose levels of your players rose and this indicates that liquids containing glucose need not be ingested during matches.

f) Heart rate (HR) during the match is an indication of exercise intensity. Target heart rate range can be calculated using this formula:

Step 1. 220 - age = HRmax.
STEP 2. (HRmax) - HRrest = trainable heart rate.
Step 3. (Trainable heart rate x .6) + HRrest = lower value
Step 4. (Trainable heart rate x .85) + HRrest + upper value.
Step 5. Target Heart Rate Range (THRR) = lower to upper values.

For example: To calculate the target heart rate range for a ten year-old child the steps are as follows.

Step 1. 220 - 10 = 210(HRmax)
Step 2. 210 - 90 = 120(trainable heart rate)
Step 3. 120 x .6 = 72 +90 = 162 =lower value.
Step 4. 120 x .85 = 102 + 90 = 192 =upper value.
Step 5. 162 - 192 = target heart rate range (THRR)

Exercising at this particular heart rate range, will improve the conditioning of the cardiovascular system. From figure 1 it can be seen that the duration spent within the target heart rate range of your players was very short and not enough to reach the expected cardiovascular training zone. Generally speaking tennis is not a sport for cardiovascular conditioning, since level of skill is the primary determinant of success during a match. Therefore if you intend to improve the stamina of your players, other types of sports should be included in the training programme. For example distance running or cycling, or any other exercise involving prolonged, continuous exercise.

Conclusions reached by the study:

The young tennis players in this study were able to balance their fluid needs and regulate thermal balance within homeostatic limits during competitive situation.

This study confirms that dehydration induced hyperthermia is not a common feature of tennis competition under the conditions of this study.

Rates of fluid intake (.... L/hr) and sweat loss (... L/hr) were sufficiently balanced to prevent significant dehydration in this competitive situation, when considering the contribution of water released by the glycogen breakdown.

Glucose solutions are not necessary for fluid replacement in children playing tennis of 90min in duration.

Metabolic rates, appear to be important determinant of rectal temperature responses under mild or warm environmental conditions.

Lastly, competitive tennis should not be considered as a sport for developing optimal cardiovascular fitness especially in novice tennis players. Other continuous forms of exercise should be incorporate in the training of children and young adults if the cardiovascular system is to be developed.

Remember the following:

(a) If your players play in a hot climate, or at the start of the summer sporting season, you should allow for 10-15 days for heat acclimatization to occur. During this time, you should gradually increase the intensity and duration of the exercise sessions and practice at the time of day at which you usually play the matches.

(b) The players should wear light-weight porous clothing and drink fluids regularly during exercise $\pm$ 200-300ml per hour (if they are weigh between 45-50 kilograms). This is dependant on the intensity of the game and the environmental conditions. They must remember to drink before and during the match. It is better to drink water every 10 minutes than to drink a lot at less frequent intervals.

(c) If the environmental temperature is very hot (greater than 33 degrees celsius), you should decrease the intensity of their exercise, or should avoid exercising for longer than 30 minutes at a time.

We once again thank you for your assistance in our research project and apologise for the delay in sending you the results. We do hope that you will find this information of interest.

Kostas Kavasis

Edith Futre

# APPENDIX C

**Appendix C.1.1: Pre-match anthropometric and physical characteristics of the subjects (n=24).**

| Group | Sub-ject | Age (years) | Mass (kg) | Height (m) | BSA* ($m^2$) | BSA/mass ratio | % Body fat |
|---|---|---|---|---|---|---|---|
| 1 | R.W | 13.5 | 41.6 | 1.55 | 1.35 | 324 | 6.70 |
| | B.C | 12.8 | 39.2 | 1.49 | 1.28 | 326 | 7.51 |
| | K.K | 12.7 | 48.3 | 1.66 | 1.52 | 311 | 14.01 |
| | L.P | 12.4 | 50.0 | 1.62 | 1.51 | 302 | 11.43 |
| | K.R | 12.4 | 56.2 | 1.73 | 1.66 | 295 | 12.46 |
| | M.R | 13.1 | 46.2 | 1.60 | 1.45 | 313 | 8.30 |
| | B.R | 12.6 | 48.1 | 1.63 | 1.49 | 309 | 16.14 |
| | B.D | 12.8 | 37.2 | 1.52 | 1.27 | 341 | 6.81 |
| 2 | G.H | 12.4 | 58.1 | 1.70 | 1.67 | 287 | 8.36 |
| | J.V | 11.4 | 46.7 | 1.57 | 1.43 | 306 | 8.93 |
| | M.J | 12.2 | 27.2 | 1.40 | 1.05 | 386 | 7.04 |
| | P.W | 12.7 | 37.5 | 1.53 | 1.28 | 341 | 6.62 |
| | E.Z | 11.4 | 48.6 | 1.52 | 1.42 | 292 | 12.56 |
| | P.M | 11.2 | 55.8 | 1.48 | 1.48 | 265 | 17.82 |
| | K.W | 12.6 | 56.0 | 1.65 | 1.61 | 287 | 17.19 |
| | S.W | 12.7 | 37.6 | 1.51 | 1.27 | 337 | 7.02 |
| 3 | C.B | 14.2 | 58.8 | 1.65 | 1.64 | 278 | 10.90 |
| | B.W | 15.5 | 69.7 | 1.77 | 1.86 | 266 | 9.75 |
| | D.G | 14.2 | 46.3 | 1.53 | 1.40 | 302 | 14.86 |
| | A.V | 15.1 | 55.8 | 1.74 | 1.67 | 299 | 7.52 |
| | A.R | 15.1 | 43.0 | 1.64 | 1.43 | 332 | 7.31 |
| | K.K | 14.9 | 52.7 | 1.67 | 1.58 | 299 | 7.52 |
| | K.B | 14.8 | 56.8 | 1.74 | 1.68 | 295 | 6.91 |
| | P.J | 15.2 | 44.0 | 1.56 | 1.39 | 315 | 6.97 |

* Body surface area

**Appendix C.1.2: Skinfold measurements (mm) of the subjects (n=24).**

| Group | Subject | Tricep | Subscapular | Abdominal | Suprailliac | Thigh | Calf |
|---|---|---|---|---|---|---|---|
| 1 | R.W | 8.2 | 5.0 | 5.2 | 3.4 | 9.2 | 8.2 |
| | B.C | 9.6 | 5.1 | 5.2 | 3.9 | 15.0 | 8.1 |
| | K.K | 7.7 | 7.7 | 12.2 | 7.8 | 10.2 | 6.0 |
| | L.P | 16.0 | 9.6 | 16.8 | 9.2 | 16.4 | 16.2 |
| | K.R | 16.1 | 10.9 | 23.8 | 15.6 | 25.4 | 21.2 |
| | M.R | 10.0 | 6.0 | 9.6 | 4.8 | 13.0 | 11.0 |
| | B.R | 19.4 | 7.4 | 12.2 | 5.4 | 21.4 | 14.6 |
| | B.D | 9.2 | 4.8 | 4.2 | 2.8 | 12.2 | 7.0 |
| $\bar{X}$<br>SD | | 12.02<br>4.44 | 7.06<br>2.26 | 11.15<br>6.71 | 6.61<br>4.24 | 15.35<br>5.58 | 11.5<br>5.33 |
| 2 | G.H | 8.0 | 6.8 | 9.4 | 5.8 | 13.8 | 11.2 |
| | J.V | 14.0 | 6.6 | 6.6 | 4.0 | 17.6 | 11.6 |
| | M.J | 8.6 | 3.8 | 4.4 | 3.2 | 11.8 | 10.6 |
| | P.W | 8.0 | 4.8 | 4.4 | 3.2 | 11.2 | 6.8 |
| | E.Z | 16.4 | 9.6 | 19.2 | 10.0 | 22.4 | 17.4 |
| | P.M | 20.6 | 19.4 | 24.4 | 22.4 | 33.8 | 24.4 |
| | K.W | 20.4 | 13.6 | 32.4 | 20.4 | 26.2 | 26.0 |
| | S.W | 7.0 | 5.4 | 6.0 | 4.4 | 10.0 | 9.4 |
| $\bar{X}$<br>SD | | 12.87<br>5.73 | 8.75<br>5.31 | 13.35<br>10.65 | 9.17<br>7.87 | 18.35<br>8.46 | 14.6<br>7.15 |
| 3 | C.B | 13.2 | 8.2 | 14.0 | 14.0 | 15.5 | 14.3 |
| | B.W | 9.6 | 6.6 | 10.6 | 5.6 | 21.0 | 14.8 |
| | D.G | 18.8 | 16.0 | 26.6 | 15.4 | 24.0 | 16.0 |
| | A.V | 11.0 | 7.0 | 8.4 | 4.0 | 8.4 | 8.2 |
| | A.R | 7.8 | 4.6 | 5.4 | 4.0 | 12.6 | 10.6 |
| | K.K | 9.2 | 6.0 | 7.6 | 3.2 | 12.6 | 8.4 |
| | K.B | 6.0 | 6.0 | 7.8 | 4.2 | 10.0 | 7.2 |
| | P.J | 5.6 | 5.0 | 5.0 | 5.4 | 13.0 | 7.8 |
| $\bar{X}$<br>SD | | 10.15<br>4.30 | 7.42<br>3.64 | 10.67<br>7.04 | 6.97<br>4.84 | 14.63<br>5.34 | 10.9<br>3.57 |

**Appendix C.2: Environmental conditions during the five experimental sessions: Ground Temperature, Relative Humidity, Wind speed and Barometric Pressure.**

| | Date | Relative Humidity (%) | $T_{Ground}$ (°C) | Wind speed ($m.sec^{-1}$) | Baromet. pressure (mmHg) |
|---|---|---|---|---|---|
| Group 1 | 22.02.92 | 70 | 25.6-32 | 0.8-1 | 643 |
| Group 2 | 07.03.92 | 60 | 24.1-26.8 | 0.2-0.8 | 636.5 |
| | 19.03.92 | 46 | 30-30.4 | 0.3-0.7 | 635 |
| Group 3 | 26.03.92 | 38 | 28-32.2 | 0.1-0.2 | 636.5 |
| | 02.04.92 | 34 | 34.4-36 | 0.3-0.8 | 636 |

Values are expressed as minimum and maximum

$T_{Ground}$: Ground temperature

**Appendix C.3: Time spent under, within and above the target HR zone and $T_{re}$ changes of the subjects (n=24).**

| Group | Sub. | U THRR (min) | W THRR (min) | O THRR (min) | Playing time (min) | pre $T_{re}$ | Post $T_{re}$ | $\Delta T_{re}$ (°C) |
|---|---|---|---|---|---|---|---|---|
| 1 | R.W | 49 | 31 | 0 | 80 | 37.6 | 38 | 0.4 |
| | B.C | 48 | 48 | 0 | 96 | 37.3 | 38.5 | 1.2 |
| | K.K | 55 | 54 | 3.5 | 113 | 37.5 | 38.8 | 1.3 |
| | L.P | 54 | 30 | 0 | 84 | 37.6 | 38 | 0.4 |
| | K.R | 57 | 25 | 0 | 82 | 37.4 | 38 | 0.6 |
| | M.R | 93 | 5 | 0 | 98 | 37.5 | 38 | 0.5 |
| | B.R | 26 | 44 | 0 | 70 | 37.5 | 38.4 | 0.9 |
| | B.D | 61 | 28 | 0 | 89 | 37.5 | 38.2 | 0.7 |
| 2 | G.H | 88 | 0 | 0 | 88 | 37.5 | 37.8 | 0.3 |
| | J.V | 88 | 0 | 0 | 88 | 36.8 | 37.7 | 0.9 |
| | M.J | 16 | 66 | 8 | 90 | 37.2 | 38 | 0.8 |
| | P.W | 55 | 33 | 0 | 88 | 37.2 | 37.9 | 0.7 |
| | E.Z | 89 | 0 | 0 | 89 | 37.7 | 38 | 0.3 |
| | P.M | 88 | 0 | 0 | 88 | 37.2 | 38.1 | 0.8 |
| | K.W | 26 | 63 | 0 | 89 | 37.6 | 38.5 | 0.9 |
| | S.W | 79 | 11 | 0 | 90 | 37.1 | 38 | 0.9 |
| 3 | C.B | 87 | 3 | 0 | 90 | 37.6 | 38 | 0.4 |
| | B.W | 71 | 19 | 0 | 90 | 37.2 | 37.9 | 0.7 |
| | D.G | 67 | 23 | 0 | 90 | 37.4 | 38.3 | 0.9 |
| | A.V | 87 | 3 | 0 | 90 | 37.6 | 38.4 | 0.8 |
| | A.R | 54 | 36 | 0 | 90 | 37.2 | 38.2 | 1 |
| | K.K | 81 | 9 | 0 | 90 | 37.2 | 38.1 | 0.9 |
| | K.B | 86 | 4 | 0 | 90 | 37.7 | 38.3 | 0.6 |
| | P.J | 40 | 50 | 0 | 90 | 37.5 | 38.2 | 0.7 |

U, THRR: under the target heart rate range
W, THRR: within the target heart rate range
O, THRR: over the target heart rate range
Pre: before the match
Post: after the match

**Appendix C.4: Estimated mean grams of carbohydrates and fat oxidized and the energy released by their oxidation during the 90 min of tennis play.**

| Group | Sub-ject | RQ | Total gr CHO | Total gr fat | KJ from CHO | KJ from fat |
|---|---|---|---|---|---|---|
| 1 | K.R | 0.89 | 77 | 20 | 1309 | 800 |
| | B.C | 0.91 | 75 | 14 | 1275 | 560 |
| | B.D | 0.89 | 56 | 14 | 952 | 560 |
| | B.R | 0.91 | 69 | 13 | 1173 | 520 |
| 2 | M.J | 0.85 | 35 | 15 | 595 | 600 |
| | G.H | 0.75 | 12 | 31 | 204 | 1240 |
| | E.Z | 0.78 | 21 | 28 | 357 | 1120 |
| | S.W | 0.80 | 24 | 22.5 | 408 | 900 |
| 3 | K.K | 0.94 | 94 | 10 | 1598 | 400 |
| | D.G | 0.96 | 101 | 6 | 1717 | 240 |
| | A.R | 0.89 | 64 | 16 | 1088 | 640 |
| | P.J | 0.94 | 95 | 10 | 1615 | 400 |
| | C.B | 0.91 | 85 | 16 | 1445 | 640 |
| $\bar{X}$<br>SD | | 0.87<br>0.06 | 62.15<br>30.28 | 16.58<br>7.15 | 1056.6<br>514.6 | 681.5<br>286.4 |

**Appendix C.5.1: Pre- and post- match values of selected blood constituents of the subjects.**

| Group | Sub-ject | Hb(g.dl$^{-1}$) pre | post | Hct(%) pre | post | Gluc(mmol.$\ell^{-1}$) pre | post | TPP(g.$\ell^{-1}$) pre | post |
|---|---|---|---|---|---|---|---|---|---|
| 1 | R.W | - | | - | | 4.3 | 4.9 | 83 | 90 |
| | B.C | - | | - | | 4.3 | 4.4 | 79 | 83 |
| | K.K | - | | - | | 4.8 | 5.6 | 79 | 84 |
| | L.P | - | | - | | 4.9 | 5.0 | 85 | 90 |
| | K.R | - | | - | | 4.5 | 5.7 | 82 | 87 |
| | M.R | - | | - | | 3.0 | 4.5 | 78 | 80 |
| | B.R | - | | - | | 3.9 | - | 83 | - |
| | B.D | - | | - | | 3.0 | - | 83 | - |
| 2 | G.H | 14.5 | 15.0 | 38.1 | 39.1 | 2.5 | 4.4 | 75 | 75 |
| | J.V | 13.8 | 13.3 | 36.7 | 35.5 | 4.0 | 4.4 | 78 | 78 |
| | M.J | 14.0 | 13.4 | 37 | 35.4 | 3.9 | 3.6 | 80 | 79 |
| | P.W | 14.3 | 14.3 | 38.4 | 38.0 | 3.8 | 5.1 | 81 | 81 |
| | E.Z | 14.0 | 14.2 | 38.4 | 38.8 | 4.6 | 4.2 | 75 | 73 |
| | P.M | 13.1 | 13.6 | 33.8 | 35.3 | 4.7 | 4.9 | 75 | 77 |
| | K.W | 13.8 | 13.6 | 36.2 | 36.2 | 5.4 | 7.5 | 82 | 83 |
| | S.W | 13.5 | 13.6 | 36.3 | 36.2 | 4.4 | 4.7 | 74 | 73 |
| 3 | C.B | 13.5 | 13.2 | 35.2 | 34.6 | 5.7 | 5.4 | 73 | 75 |
| | B.W | 14.5 | 14.5 | 39.0 | 38.1 | 7.3 | 5.1 | 78 | 79 |
| | D.G | 13.1 | 13.4 | 33.5 | 34.1 | 5.4 | 5.5 | 74 | 75 |
| | A.V | 15.8 | 16.3 | 40.9 | 41.1 | 5.4 | 5.3 | - | - |
| | A.R | 13.6 | 13.4 | 35.7 | 34.1 | 5.0 | 5.6 | 79 | 80 |
| | K.K | 15.1 | 15.3 | 38.9 | 38.8 | 6.1 | 5.3 | 87 | - |
| | K.B | 14.0 | 14.1 | 36.1 | 35.9 | 5.3 | 4.7 | 89 | 86 |
| | P.J | 14.0 | 14.3 | 38.9 | 38.8 | 5.9 | 5.2 | 76 | 80 |

Figures for players resident at the Witwatersrand altitude (+1740m)

**Appendix C.5.2: Pre- and post-match concentrations of selected plasma electrolytes (mmol.dl$^{-1}$) of the subjects (n=24).**

| Group | Sub-ject | $Na^+$ pre | $Na^+$ post | $K^+$ pre | $K^+$ post | $Cl^-$ pre | $Cl^-$ post | $Mg^{++}$ pre | $Mg^{++}$ post |
|---|---|---|---|---|---|---|---|---|---|
| 1 | R.W | 146 | 147 | 4.0 | 4.0 | 110 | 114 | 0.70 | 0.70 |
| | B.C | 144 | 145 | 4.7 | 4.6 | 111 | 113 | 0.90 | 0.70 |
| | K.K | 141 | 146 | 4.1 | 4.0 | 106 | 112 | 1.00 | 1.98 |
| | L.P | 143 | 145 | 4.4 | 4.3 | 110 | 110 | 0.90 | - |
| | K.R | 145 | 148 | 4.2 | 4.1 | 108 | 110 | 1.00 | 0.90 |
| | M.R | 145 | 146 | 4.2 | 4.2 | 113 | 113 | 1.92 | 0.90 |
| | B.R | 146 | - | 4.0 | - | 109 | - | 1.00 | - |
| | B.D | 145 | - | 4.0 | - | 108 | - | 0.70 | - |
| 2 | G.H | 140 | 137 | 3.9 | 4.0 | 107 | 108 | 1.05 | 1.04 |
| | J.V | 139 | 138 | 3.6 | 3.9 | 107 | 106 | 1.02 | 1.03 |
| | M.J | 137 | 138 | 3.3 | 3.8 | 104 | 106 | 1.00 | 0.97 |
| | P.W | 139 | 141 | 4.1 | 4.0 | 107 | 108 | 1.14 | 1.10 |
| | E.Z | 141 | 141 | 3.9 | 3.8 | 109 | 109 | 0.94 | 0.90 |
| | P.M | 138 | 137 | 3.7 | 3.8 | 109 | 111 | 0.87 | 0.90 |
| | K.W | 139 | 141 | 3.6 | 3.3 | 105 | 110 | 0.99 | 0.85 |
| | S.W | 141 | 142 | 3.8 | 3.7 | 109 | 111 | 0.93 | 0.92 |
| 3 | C.B | 138 | 140 | 4.0 | 3.8 | 106 | 111 | 0.97 | 0.94 |
| | B.W | 139 | 143 | 4.1 | 3.6 | 106 | 111 | 0.81 | 0.91 |
| | D.G | 139 | 142 | 3.8 | 3.8 | 109 | 110 | 1.00 | 0.97 |
| | A.V | 142 | 143 | 3.3 | 3.4 | 106 | 110 | 0.92 | 0.91 |
| | A.R | 141 | 143 | 3.9 | 3.5 | 110 | 110 | 0.83 | 0.81 |
| | K.K | 142 | 141 | 3.7 | 4.1 | 109 | 109 | 0.95 | 0.88 |
| | K.B | 141 | 144 | 3.3 | 3.8 | 110 | 115 | 0.82 | 0.86 |
| | P.J | 139 | 139 | 3.8 | 3.6 | 106 | 10[illegible] | 0.82 | 1.01 |

pre: pre-match values
post: post-match values

**Appendix C.5.3: Percent Hct, Hb, TPP and blood volume compartment changes of groups two and three.**

| Group | Sub. | Hb | Hct | TPP* | BV | MCV | RCV | PV | TPP** |
|---|---|---|---|---|---|---|---|---|---|
| 2 | G.H | +3.44 | +2.62 | 0 | -3.4 | +0.79 | -0.83 | -5.20 | -5.20 |
| | J.V | -3.62 | -3.26 | 0 | +3.7 | -0.53 | +0.29 | +5.90 | +5.90 |
| | M.J | -4.28 | -4.32 | -1.25 | +4.3 | 0 | -0.21 | +7.50 | +6.25 |
| | P.W | 0 | -1.04 | 0 | 0 | +1.08 | -1.04 | +0.60 | +0.61 |
| | E.Z | +1.42 | +1.04 | -2.66 | -1.4 | +0.27 | -0.37 | -2.10 | -4.76 |
| | P.M | +3.81 | +4.43 | +2.66 | -3.8 | -0.52 | +0.46 | -6.30 | -3.63 |
| | K.W | -1.44 | 0 | +1.21 | +1.5 | -1.57 | +1.50 | +1.40 | +2.61 |
| | S.W | +0.74 | -0.27 | -1.35 | -0.7 | +1.08 | -0.97 | -0.60 | -1.95 |
| 3 | C.B | -2.20 | -1.70 | +2.73 | +2.3 | -0.29 | +0.53 | +3.14 | +5.87 |
| | B.W | 0 | -2.30 | +1.28 | 0 | +2.37 | -2.30 | +1.47 | +2.75 |
| | D.G | +2.29 | +1.79 | +1.35 | -2.2 | +0.56 | -0.44 | -3.18 | -1.82 |
| | A.V | +3.16 | +0.48 | - | -3.1 | +2.57 | -2.62 | -3.42 | - |
| | A.R | -1.47 | -4.48 | +1.26 | +1.5 | +3.18 | -3.08 | +3.92 | +5.18 |
| | K.K | +1.32 | -0.25 | - | -1.3 | +1.70 | -1.54 | -1.23 | - |
| | K.B | +0.71 | -0.55 | -3.37 | -0.7 | +1.14 | -1.24 | -0.48 | -3.85 |
| | P.J | +2.14 | -0.25 | +5.26 | -2.1 | +2.45 | -2.33 | -1.93 | +3.33 |

*: calculated from pre- and post exercise TPP values, **: calculated from PV changes

**Appendix C.6: Measured and estimated total fluid loss, and fluid replacement needs of the subjects (n=24).**

| GROUP | Sub. | IBM Kg | FBM Kg | ΔBM Kg | LI *l* | TML Kg | MFU Kg | MWP *l* | RWL *l* | SL *l* | WD *l* | % deh. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | R.W | 41.6 | 41.1 | 0.5 | 0.500 | 1.000 | 0.048 | 0.029 | 0.026 | 0.926 | 0.462 | 1.11 |
| | B.C | 39.2 | 38.8 | 0.4 | 0.530 | 0.930 | 0.089 | 0.056 | 0.049 | 0.792 | 0.393 | 1.00 |
| | K.K | 48.8 | 48.3 | 0.5 | 0.630 | 1.130 | 0.119 | 0.075 | 0.065 | 0.946 | 0.468 | 0.95 |
| | L.P | 50.0 | 49.3 | 0.7 | 0.190 | 0.890 | 0.075 | 0.050 | 0.044 | 0.771 | 0.383 | 0.76 |
| | K.R | 56.2 | 55.7 | 0.5 | 0.640 | 1.140 | 0.097 | 0.063 | 0.056 | 0.987 | 0.490 | 0.87 |
| | M.R | 46.2 | 45.5 | 0.7 | 0.680 | 1.380 | 0.054 | 0.040 | 0.040 | 1.286 | 0.643 | 1.39 |
| | B.R | 48.1 | 47.3 | 0.8 | 0.410 | 1.210 | 0.082 | 0.053 | 0.045 | 1.083 | 0.538 | 1.11 |
| | B.D | 37.2 | 36.6 | 0.6 | 0.210 | 0.810 | 0.070 | 0.046 | 0.040 | 0.700 | 0.347 | 0.93 |
| 2 | O.H | 58.1 | 57.6 | 0.5 | 0.610 | 1.110 | 0.043 | 0.040 | 0.038 | 1.029 | 0.514 | 0.88 |
| | J.V | 46.7 | 46.7 | 0.0 | 0.970 | 0.970 | 0.054 | 0.047 | 0.045 | 0.871 | 0.435 | 0.93 |
| | M.J | 27.2 | 27.2 | 0.0 | 0.390 | 0.390 | 0.050 | 0.035 | 0.033 | 0.307 | 0.153 | 0.56 |
| | P.W | 37.5 | 37.2 | 0.3 | 0.170 | 0.470 | 0.051 | 0.039 | 0.035 | 0.384 | 0.190 | 0.50 |
| | E.Z | 48.6 | 48.6 | 0.0 | 0.550 | 0.550 | 0.049 | 0.042 | 0.039 | 0.462 | 0.230 | 0.47 |
| | P.M | 55.8 | 55.7 | 0.1 | 0.580 | 0.680 | 0.078 | 0.067 | 0.063 | 0.539 | 0.268 | 0.48 |
| | K.W | 56.0 | 55.6 | 0.4 | 0.360 | 0.760 | 0.058 | 0.042 | 0.038 | 0.664 | 0.330 | 0.58 |
| | S.W | 37.6 | 37.3 | 0.3 | 0.400 | 0.700 | 0.047 | 0.037 | 0.035 | 0.618 | 0.308 | 0.81 |
| 3 | C.B | 58.8 | 58.1 | 0.7 | 0.330 | 1.030 | 0.101 | 0.064 | 0.060 | 0.869 | 0.432 | 0.73 |
| | B.W | 69.7 | 68.6 | 1.1 | 0.190 | 1.290 | 0.079 | 0.072 | 0.062 | 1.149 | 0.570 | 0.81 |
| | D.G | 46.3 | 46.1 | 0.2 | 0.360 | 0.560 | 0.107 | 0.062 | 0.053 | 0.400 | 0.196 | 0.42 |
| | A.V | 55.8 | 55.0 | 0.8 | 0.290 | 1.090 | 0.090 | 0.059 | 0.052 | 0.948 | 0.471 | 0.84 |
| | A.R | 43.0 | 42.4 | 0.6 | 0.300 | 0.900 | 0.080 | 0.052 | 0.046 | 0.774 | 0.384 | 0.89 |
| | K.K | 52.7 | 52.3 | 0.4 | 0.170 | 0.570 | 0.104 | 0.063 | 0.053 | 0.413 | 0.202 | 0.38 |
| | K.B | 56.8 | 55.9 | 0.9 | 0.500 | 1.400 | 0.063 | 0.042 | 0.037 | 1.300 | 0.648 | 1.14 |
| | P.J | 44.0 | 43.5 | 0.5 | 0.310 | 0.810 | 0.105 | 0.063 | 0.054 | 0.651 | 0.321 | 0.72 |

**Appendix C.7.1: One way anova on group means for selected anthropometric physiological, metabolic and biochemical characteristics at the 5% significance level.**

| Parameters | F-ratio | Sign. | Location of difference |
|---|---|---|---|
| Age (years) | 66.886 | .0001 | 1<3, 2<3 |
| Mass (Kg) | | NS | |
| Height (m) | 3.672 | .0429 | 2<3 |
| BSA ($m^2$) | | NS | |
| BSA/mass ratio | | NS | |
| Fat (%) | | NS | |
| Skinfolds | | NS | |
| Under THRR (min) | | NS | |
| Within THRR (min) | | NS | |
| Over THRR (min) | | NS | |
| Water ingested (kg) | 23.192 | .0001 | 2>1, 2>3, 1>3 |
| $T_{re}$ changes (°C) | | NS | |
| Total Sweat Loss ($\ell$) | 3.782 | .0396 | 1>2 |
| Sweat-rate ($\ell.hr^{-1}$) | 3.543 | .0472 | 1>2 |
| Total dehydration (%) | 8.697 | .0018 | 1>2, 1>3 |
| $.h^{-1}$ dehydration (%) | 7.936 | .0027 | 1>2, 1>3 |
| TML (Kg) | 5.690 | .0106 | 1>2 |
| Water deficit ($\ell.h^{-1}$) | 3.601 | .0452 | 1>2 |
| Total KJ | | NS | |
| Total CHO oxidized (gr) | 22.272 | .0001 | 2<1, 2<3 |
| Total fat oxidized (gr) | 8.036 | .0026 | 2>1, 2>3 |
| Metabolic water produced (gr) | 4.037 | .0329 | 2<3 |
| Hb | | NS | |
| Hct | | NS | |
| TPP* | | NS | |
| BV | | NS | |
| MCV | 7.912 | .0138 | 2<3 |
| RCV | 7.912 | .0138 | 2<3 |
| PV | | NS | |
| TPP** | 8.634 | .0026 | 1>2, 1>3 |

where 1: group one; 2: group two; 3: group three
*: calculated from pre- and post-match TPP values; **: calculated from plasma volume changes

**Appendix C.7.2: One way anova on selected baseline and post-match means between the subject groups at the 5% significance level.**

| Parameters | F-ratio | Sign. | Location of difference |
|---|---|---|---|
| pre- $T_{re}$ | | NS | |
| post- $T_{re}$ | | NS | |
| pre- BM | | NS | |
| pre- Hb | | NS | |
| pre- Hct | | NS | |
| pre- Na | 25.127 | .0000 | 1>2, 1>3 |
| pre- K | 8.219 | .0023 | 1>2, 1>3 |
| pre- Cl | | NS | |
| pre- Mg | | NS | |
| pre- Glucose | 12.084 | .003 | 1<3, 2<3 |
| pre- TPP | | NS | |

**Appendix C.8.1: Co-efficients of correlation of anthropometric and physical characteristics of the subjects (n=24).**

| | Correlation coefficient<br>Significance level | | | | |
|---|---|---|---|---|---|
| Variable | 2 | 3 | 4 | 5 | 6 |
| 1. Age (years) | .29<br>.1691 | .48<br>.0171 | .37<br>.0685 | -.12<br>.5512 | -.35<br>.0931 |
| 2. Mass (Kg) | — | .85<br>.0000 | .97<br>.0000 | -.93<br>.0000 | .38<br>.0600 |
| 3. Height (m) | | | .91<br>.0000 | -.59<br>.0021 | .04<br>.8323 |
| 4. BSA ($m^2$) | | | | -.85<br>.0000 | .27<br>.1880 |
| 5. BSA/mass | | | | | -.53<br>.0074 |
| 6. Body fat (%) | | | | | |

**Appendix C.8.2: Correlations coefficients of percent changes in blood volume compartments of groups two and three.**

| | Correlation coeeficient (n) p | | | |
|---|---|---|---|---|
| Variable | 2 | 3 | 4 | 5 |
| 1. BV | -.29<br>(13)<br>.3360 | .31<br>(14)<br>.2743 | .98<br>(14)<br>.0000 | .83<br>(12)<br>.0008 |
| 2. MCV | | -.99<br>(15)<br>.0000 | -.031<br>(15)<br>.9115 | .11<br>(13)<br>.6975 |
| 3. RCV | | | .10<br>(16)<br>.7054 | -.047<br>(14)<br>.8728 |
| 4. PV | | | | .85<br>(14)<br>.0001 |
| 5. TPP* | | | | |

(n): (sample size)
p: significance level
*: calculated from plasma volume changes

**Appendix C.9: Correlation coefficients of 13 variables with post-match $T_{re}$.**

| | Correlation (r) | Significance (p) |
|---|---|---|
| Group 1 | | |
| UTHRR (min) | -.3726 | .3636 |
| WTHRR (min) | .8401 | .0090* |
| OTHRR (min) | .7524 | .0312* |
| Body mass (kg) | -.1443 | .7331 |
| BSA ($m^2$) | -.1146 | .7870 |
| BSA/mass ($m^2$/kg) | .1569 | .7105 |
| Body fat (%) | .3921 | .3367 |
| Water ingested (ml) | .1864 | .6585 |
| Total dehydration (%) | .0307 | .9425 |
| Total swat loss (ℓ) | -.4574 | .2545 |
| ΔBV (%) | - | - |
| ΔPV (%) | - | - |
| TPP* (%) | - | - |
| Group 2 | | |
| UTHRR (min) | -.5469 | .1606 |
| WTHRR (min) | .5766 | .1346 |
| OTHRR (min) | - | - |
| Body mass (Kg) | .2283 | .5865 |
| BSA ($m^2$) | .1858 | .6596 |
| BSA/mass ($m_2$/kg) | -.2020 | .6314 |
| Body fat (%) | .6961 | .0552 |
| Water ingested (ml) | -.4846 | .2236 |
| Total dehydration (%) | -.3636 | .3760 |
| Total sweat loss (ℓ) | -.1076 | .7998 |
| ΔBV (%) | -.0120 | .9776 |
| ΔPV (%) | .0196 | .9632 |
| ΔTPP* (%) | -.0887 | .8345 |
| Group 3 | | |
| UTHRR (min) | .0111 | .9792 |
| WTHRR (min) | -.0097 | .9817 |
| OTHRR (min) | - | - |
| Body mass (kg) | -.5545 | .1538 |
| BSA ($m_2$) | -.4442 | .2702 |
| BSA/mass ($m_2$/Kg) | .5829 | .1294 |
| Body fat (%) | -.1176 | .7815 |
| Water ingested (ml) | .5454 | .1620 |
| Total dehydration (%) | 6053 | .1118 |
| TSL (L) | .3160 | .4458 |
| ΔBV (%) | -.6293 | .0946 |
| ΔPV (%) | -.6265 | .0965 |
| ΔTPP* (%) | -.6214 | .1878 |

*: calculated from PV changes

**Appendix C.10.1: Simple linear regression equations of physiological variables in the three subject groups.**

| Response variable | Equation | SE | r | n |
|---|---|---|---|---|
| $\dot{V}O_2$(Group one) | $\psi_1 = -18.283 + .266107X_1$ | 5.18 | 0.77 | 15 |
| $\dot{V}O_2$(Group two) | $\psi_1 = -3.52553 + .162785X_1$ | 4.93 | 0.67 | 33 |
| $\dot{V}O_2$(Group three) | $\psi_1 = -3.79722 + .183652X_1$ | 3.00 | 0.70 | 32 |
| $\dot{V}O_2$(Total Group) | $\psi_1 = -5.57484 + .185953X_1$ | 4.46 | 0.69 | 80 |
| **GROUP ONE** | | | | |
| Final $T_{re}$(˚C) | $\psi_2 = 37.6937 + .0164163X_2$ | 0.17 | 0.84 | 8 |
| Final $T_{re}$(˚C) | $\psi_3 = 38.1571 + .183673X_3$ | 0.21 | 0.75 | 8 |
| Water ingested($\ell$) | $\psi_4 = 1.37928 - 8.7596X_4$ | 0.09 | -0.87 | 8 |
| Water ingested($\ell$) | $\psi_5 = 1.35682 - .390521X_5$ | 0.13 | -0.74 | 8 |
| **GROUP THREE** | | | | |
| Water ingested($\ell$) | $\psi_6 = -135.241 + 256.868X_6$ | 76.4 | 0.72 | 8 |

$\psi_1$: $\dot{V}O_2$ ($ml.Kg^{-1}.min^{-1}$); $X_1$: HR (bpm)

The predictor variables $X_2$, $X_3$, $X_4$, $X_5$, $X_6$, represents:

$X_2$= min spent within the target HRR; $X_3$= min spent over the target HRR; $X_4$= water deficit ($\ell$); $X_5$= total dehydration (%); $X_6$= total dehydration (%);

SE= standard error of estimation; r= correlation coefficient; n= number of observations

**Appendix C.10.2: Simple linear regression equations of percent blood parameter changes.**

| Response varible | Equation | SE | r | n |
|---|---|---|---|---|
| | **GROUP TWO** | | | |
| BV | $\psi_1 = .0382519 + .623619X_1$ | 0.95 | 0.95 | 8 |
| BV | $\psi_2 = -.0671038 + .614026X_2$ | 0.36 | 0.99 | 8 |
| PV | $\psi_3 = -.154953 + .891351X_3$ | 1.67 | 0.94 | 8 |
| MCV | $\psi_4 = -.0764298 - .930937X_4$ | 0.10 | -0.99 | 8 |
| | **GROUP THREE** | | | |
| BV | $\psi_5 = -.560287 + .653629X_5$ | 0.49 | 0.96 | 8 |
| MCV | $\psi_6 = .140557 - 1.03395X_6$ | 0.11 | -099 | 8 |

The predictor varibles $X_1$, $X_2$, $X_3$, $X_4$, $X_5$, $X_6$, represents:

$X_1$ = PV changes calculated from Hb and Hct values

$X_2$ = protein changes calculate from PV changes

$X_3$ = protein changes calculate from PV changes

$X_4$ = mean corpuscular Hb changes

$X_5$ = PV changes calculated from Hb and Hct values

$X_6$ = mean corpuscular Hb changes

SE = standard error of estimation
r = correlation coefficient
n = number of observations

# APPENDIX D

## ABBREVIATIONS

| | |
|---|---|
| $(a\text{-}v)O_2$ | arteriovenous $O_2$ difference |
| AAP | American Academy of Pediatrics |
| ACSM | American Academy of Sports Medicine |
| ADH | antidiuretic hormone |
| ATP | adenosine triphosphate |
| AVD | active vasodilation |
| bpm | beats per minute |
| BSA | body surface area |
| BV | blood volume |
| °C | degrees celsius |
| CHO | carbohydrates |
| $Cl^-$ | plasma cloride |
| CO | carbon dioxide |
| CP | creatine phosphate |
| %ΔPV | percent plasma volume changes |
| EDV | end systolic volume |
| e.g., | *exempli gratia* (for example/for instance) |
| et al., | *et alii* (and others) |
| fig. | figure |
| $H^+$ | ion concentration |
| Hb | hemoglobin concentration |
| $HCO_3^-$ | bicarbonate |
| Hct | hematocrit |
| HDL | high density lipoprotein |
| HR | heart rate |
| HRR | heart rate range |
| HV | heart volume |
| i.e. | *id est* (which is to say/in other words) |
| $K^+$ | plasma potassium |
| Kg | kilogram |
| KJ | kilojoule |
| $\ell.hr^{-1}$ | liters per hour |
| $\ell$ | liter |
| LVM | left verticular muscle mass |
| MCHC | mean corpuscular Hb concentration |
| MCV | mean corpuscular volume |
| $Mg^{++}$ | plasma magnesium |
| min | minute |
| $Na^+$ | plasma sodium |
| NaCl | sodium cloride |
| PFK | phosphofructokinase |
| PH | acid-base balance |
| %HRmax | percentage of maximum heart-rate |
| post | after the match |
| pre | before the match |

| | |
|---|---|
| PV | plasma volume |
| R | response |
| RQ | respiratory quotient |
| SBF | skin blood flow |
| SDH | succinate dehydrogenase |
| SR | sweat rate |
| $T_{air}$ | ambient temperature |
| $T_c$ | body core temperature |
| $T_{db}$ | dry bulb temperature |
| $T_g$ | globe temperature |
| THRR | target HRR (60-85% of HRmax reserve) |
| THRR(O) | over the THRR (>85% of HRmax reserve) |
| THRR(U) | under the THRR (<60% of HRmax reserve) |
| THRR(W) | within the THRR (60-85% of HRmax reserve) |
| TPP | total plasma protein |
| $T_{re}$ | rectal temperature |
| $T_{set}$ | set point temperature threshold |
| $T_{sk}$ | skin temperature |
| $\bar{T}_{sk}$ | mean skin temperature |
| $T_{wb}$ | wet bulb temperature |
| $T_{ws}$ | weight sum temperature ($T_c+\bar{T}_{sk}$) |
| $\dot{V}O_2max$ | maximum volume of oxygen consumption.min$^{-1}$ |
| vs | versus |
| WBGT | wet bulb globe temperature index |
| yr(s) | year(s) |
| ZOF | zone of optimal function |

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