THE EFFECTS OF A 160 KM RUN ON SELECTED ANTHROPOMETRIC, PHYSIOLOGICAL AND PSYCHOLOGICAL PARAMETERS

BY

CARMEN OLTMANN

THESIS

Submitted in fulfillment of the requirements for the Degree of Master of Science

Department of Human Movement Studies

Rhodes University, 1992

Grahamstown, South Africa

ABSTRACT

Twenty-one male subjects volunteered to participate in this study of the effects of an ultramarathon run under competitive conditions. Selected anthropometric measurements were made before and after the race. Blood samples were taken before, and within 10 min. of completing the race. Haematocrit and cortisol concentrations were analysed from each sample. Three ratings of perceived exertion (RPE) - local, central and overall, were obtained at two-hourly intervals throughout the race. The shortened form of the Eysenck Personality Inventory (EPI) was administered before the race. The Profile of Mood States (POMS) test was completed both before and immediately after the race.

The average running intensity was estimated to be about 32% $\dot{V}O_2max$, which would not have caused major lactate accumulation. Repetitive compressive forces as a result of heel-strike resulted in intervertebral disc fluid loss. This resulted in a significant ($p<0.05$) decrease in stature after the race. Mobilization of glycogen, triglycerides and protein for metabolism as well as fluid loss via sweat resulted in significantly decreased body mass after the race. Plasma volume and serum cortisol concentrations were significantly increased after the race compared to pre-race baseline levels. Local RPE responses were dominant throughout the entire race, followed by overall RPE.

Running 160 km had an effect on mood state. "Depression", "fatigue" and "confusion" (Profile of Mood States) were all significantly increased compared to pre-race scores. Sleep deprivation as a result of running through the night was probably an important contributing factor.

The anthropometric profile of the ultramarathon runners in this sample was more like that of the general population than elite marathon runners from the literature. The ultramarathon runners were more extroverted than elite marathon runners, but exhibited a similar Profile of Mood State before the race.

The stressor - running 160 km - resulted in a 'strain response'. This was evident in the decreased stature and mass, and increased serum cortisol, "depression", "confusion" and above all "fatigue".

ACKNOWLEDGEMENTS

I wish to extend my sincere thanks to the following for their assistance in this study :

Professor J Charteris for his guidance, support and patience as my supervisor.

Dr PA Scott for her continual support and advice.

The Washie runners and organisers, without whom this study would not have been possible.

Last, but by no means least, to my parents and family for being so tolerant and supportive.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER ONE

INTRODUCTION

Most of the emphasis of research conducted on runners has been on acute changes that occur during relatively short runs, but few studies have probed the changes that occur during ultra endurance running.

In general the physiological and psychological effects of running reflect transient changes in homeostasis. These changes may be perceived as medically 'abnormal' if taken out of the context in which they occur, just as apnoeic bradycardia appears abnormal unless seen in the context of the diving reflex.

In his early writings Selye used the term 'stress' to describe the "sum of all nonspecific changes (within an organism) caused by function or damage" or more simply, "the rate of wear and tear in the body" (Selye, 1976). Confusion concerning whether stress was a 'stimulus' as used in physics or a 'response' as initially used by Selye has long plagued the stress literature (Everly,1989). However, most researchers now distinguish between cause and effect by using the terms 'stressor' and 'response', where 'stressor' is causal. In the present study, therefore, running 160 km is deemed the stressor which in turn elicits a 'strain' response.

Another concept - 'fatigue' - has nebulous scientific and medical meanings. Often it refers to the inability to sustain an activity. The background of the author involved and the

task performed also seem to influence the definition of 'fatigue' used in a particular context.

In 1913 Frumerie suggested that fatigue is probably a result of prolonged mechanical stimulation of nerve endings in joints and tendons. In 1924 Hill and associates proposed a tissue acidosis and lactic acid accumulation model of fatigue. In 1947 Bartley and Chute divided fatigue into three categories:

(a) objective fatigue: where declines in work output were fatigue indicators

(b) subjective fatigue: where the feelings of discomfort were the indicators of fatigue

(c) physiological fatigue: where impairments at the tissue level were the indicators of fatigue.

The implications of this categorization are that fatigue represents a conflict between the demands of the task (stressor) and the aversion to the effort.

In 1914 Ash suggested that the final limit to performance is dependent on central rather than peripheral factors. According to Simonson (1971) this issue of central versus peripheral fatigue remains unsolved. It has been conventional to think of fatigue as a peripheral acidotic phenomenon but several authors have suggested that there is a definite central nervous system (CNS) component to fatigue (Mutch and Banister,1983 ; Banister et al., 1985). It is as yet unclear whether the CNS component is more important in the decline of motor function or the individual's perception of fatigue. According to Morgan and Pollock (1977) successful endurance

performance is governed both by the runner's physical capacity and willingness to tolerate discomfort associated with the hard physical work. It can therefore be expected that one's psychological state will influence one's performance. Morgan and Pollock (1977) also found that elite marathon runners tend to have unique psychological characteristics that differ from those of the normal population. The possibility exists that ultra marathon runners differ not only from the normal population but also from marathon runners.

When investigating physiological responses to an ultra-marathon, attention must be given to 'natural' fluctuations due to circadian rhythmicity and where possible baseline data must be collected in order to avoid false positive or negative findings (Everly,1989).

The most direct way of measuring the stress response is by measuring ACTH, the corticosteroids, and the catecholamines (Selye, 1976). Measuring cortisol concentration changes appears to be the most common method of determining ACTH and corticosteroid activity.

STATEMENT OF THE PROBLEM

The central purpose of this study was to determine the effects of running 160 km on selected anthropometric, physiological and psychological parameters, and to compare the anthropometry and psychological profiles of ultramarathoners to marathoners.

RESEARCH HYPOTHESES

This study investigates the following broad hypotheses:

1. No difference exists between stature or mass before and after the ultramarathon.
2. No difference exists between serum cortisol concentrations before an ultramarathon race and immediately afterwards.
3. No difference exists between local, central and overall Ratings of Perceived Exertion (RPE) (Ekblom and Goldbarg,1971) over the course of an ultramarathon.
4. No difference exists between Profile of Mood States (POMS) before the race and POMS after the race.

In addition, the ultramarathon runners in this sample were compared to marathon runners in the literature with respect to :

a. anthropometry,

b. Eysenck's Personality Inventory and

c. Profile of Mood States.

STATISTICAL HYPOTHESES

1. H_o : $\mu A_{before} = \mu A_{after}$

   H_a : $\mu A_{before} \neq \mu A_{after}$

   where 'A' represents the mean stature or mass before or after the ultramarathon.

2. H_o : $\mu E_{before} = \mu E_{after}$

   H_a : $\mu E_{before} \neq \mu E_{after}$

   where 'E' represents the mean serum cortisol concentration of all the subjects, before or after the race.

3. H_o : $\mu_l = \mu_c = \mu_o$

   H_a : $\mu_l \neq \mu_c \neq \mu_o$

   where 'μ_l', 'μ_c' and 'μ_o' refer to mean Ratings of Perceived Exertion (RPE) obtained at two-hourly intervals through the race; l being local, c being central and o being overall forms of RPE.

4. H_o : $\mu M_{before} = \mu M_{after}$

   H_a : $\mu M_{before} \neq \mu M_{after}$

   where' μ M' represents the mean POMS of all the subjects, before or after the race.

DELIMITATIONS

Data were collected on 21 male runners competing in the Washie 160 km race between Port Alfred and East London. The race started at 19h00 and competitors had 26 hours in which to complete the distance. Thus all subjects were exposed to the same 'geophysical' conditions although the duration of physical exertion varied. In other words all subjects were required to cover the same course but the time for completing the race varied substantially, ranging from 17 hours to 25 hours.

The runners were selected on availability provided they had completed at least one ultramarathon before. No runner was under any coercion to volunteer.

Since it was an actual race, the runners had peaked and tapered their training accordingly.

LIMITATIONS

This study adopted an holistic approach to ultra-marathon running, i.e. it was a general overview rather than a specific one. The laboratory situation is such that certain factors can be controlled. The tester can control the environment and the activity of the subjects, before during and after the experimental session, can be stipulated and controlled. However, '*in situ*' conditions such as pertained in this road race, involve much less control. The environmental conditions are beyond the tester's control, and because it is a 'race situation', so are the activities before, during and after the race.

The ideal situation would have been to test the runners at various set intervals during the race and during the recovery period. However, certain factors made this impossible :

(1) the race was run at night,
(2) the race was not run on a track or circular course,
(3) most runners left town after the race and
(4) taking numerous blood samples during the race may have affected performance negatively.

For these reasons the data were collected before and immediately after the race.

Each subject followed an individualised training programme: therefore there was no way of controlling state of training.

CHAPTER TWO

REVIEW OF THE LITERATURE

Introduction:

The marathon race commemorates the immortal run of an unknown soldier, fully armoured and 'hot from battle', to inform the Athenian capital that the invading Persians had been defeated on the plains of Marathon. Had the Athenians in their unguarded capital remained ignorant of the Persian defeat, they might have surrendered without resistance to a direct seaborne invasion : thus the reason for the run. Legend has it that, having delivered his news, the messenger died suddenly from what is considered to be a cardiac infarct. Thus in legend at least, the marathon race is linked with extreme physical exertion (Gardiner,1930).

Ultraendurance exercise is defined as exercise lasting longer than 4 hours (O'Toole and Douglas,1989). It is not a new phenomenon - in the middle to late 1800's 24-hour and 6-day races were common. The Greeks had a special name for participants in ultraendurance races - they called them 'hemerodromoi', meaning experts at covering long distances (Osler and Dodd,1979).

In 1925 Hill wrote that fatigue resulting from running is due to "...exhaustion of the material of the muscle or to incidental disturbances which may make a man stop before his muscular system reached its limits". Fatigue is a complex phenomenon which seems to consist of central and peripheral components (Green,1990). Fatigue seems to be dependent on elapsed time and the type of activity (Cameron,1974).

However, to simply think that fatigue is a function linking elapsed time and type of activity seems to be an oversimplification.

In vitro and in vivo methods have been used to investigate peripheral fatigue. Various invasive techniques have been used to determine:

(a) arterio-venous differences in O_2 concentrations;

(b) flux of substances;

(c) release of metabolites;

in order to find causes for such fatigue (Green,1990).

In 1973 Costill and associates suggested that the main cause of muscular fatigue caused by long distance running was the depletion of muscle glycogen in the slow twitch fibres. Since then other possible causes of fatigue have been suggested (Costill, 1974; Fitts, 1977). They include:

(1) lactic acid accumulation

(2) hypoglycaemia

(3) dehydration

(4) electrolyte loss

(5) hyperthermia

Fatigue may be a consequence of any one of these factors or combinations thereof.

(1) <u>Lactic acid accumulation</u>

Blood lactate increases exponentially with increasing exercise intensity (Katz and Sahlin, 1987). During light work, i.e. less than 50% of $\dot{V}O_2$ max, blood lactate remains constant or even declines (Owles, 1930; Graham, 1984). As

work load increases blood lactate also begins to increase.

(2) Hypoglycaemia

When Gunderson et al.(1983) tested changes in marathon runners, they found that the incidence of hypoglycaemia was low. In fact most runners had increased blood glucose concentrations at the completion of the race, or less than 25% less than pre-race values. Noakes and Carter (1976) measured blood glucose concentrations before and after a 160 km running race. They found that 11 of the 13 subjects had higher blood glucose concentrations at the finish, one subject had no change, and only one subject had experienced a decline. A possible reason for this is that free fatty acids (FFA) are utilised during such prolonged exercise, thereby sparing the muscle glycogen.

Proper carbohydrate ingestion during the 160 km race should also ensure that hypoglycaemia does not become the main limiting factor of performance.

(3) Dehydration

The sweat rate of runners depends on several factors : the environmental conditions, the intensity of running and the degree of acclimatization to the prevailing conditions (Burke and Read,1989). Fluid losses of 2 to 5 % may lead to impaired thermoregulation and performance (Brotherhood,1984). Therefore in sports of long duration it is critical that body fluid is replenished. Fluid replacement is dependent on the rate of gastric emptying, which depends on the following

factors : ambient temperature, the temperature of the fluid, the caloric content and osmolality of the fluid, the volume of fluid and the intensity of running (Murray,1987).

ACTH is released from the anterior pituitary gland into the circulatory system. The target organ of ACTH is the adrenal cortex. Here it has an effect on all three zona. The cells of each zona are stimulated and release their contents into the circulatory system. Thus glucocorticoids, mineralocorticoids and cortical adrenal androgenic hormones are released.

Aldosterone is one of the mineralocorticoids released. Its target tissue is the renal tubules, where it facilitates absorption of sodium and chloride. This causes fluid retention which will oppose dehydration (Guyton,1981).

(4) Electrolyte loss

Water and electrolytes are lost in sweat. However, there seem to be varying opinions as to whether electrolytes should be added to replacement fluids. According to the American Dietetic Association the Western diet supplies sufficient sodium, potassium, chloride and magnesium (Burke and Read,1989). Noakes and associates (1985) found that hyponatraemia developed in ultraendurance runners who ingested only water and no electrolytes.

(5) Hyperthermia

Hyperthermia may be a real threat to the runners. In order to minimize this threat, the race under study was held in

winter and started at 19h00, ensuring about 14 hours of relatively cool conditions.

In order to focus on the effects of running 160 km, three areas are here discussed : anthropometry, physiological parameters and psychological parameters.

ANTHROPOMETRY AND LONG TERM EXERTION

In 1940 Sheldon et al. was relating somatotypes to structure, function and temperament. It seemed there were distinct relationships between anthropometry on the one hand and mental and physical structure and function on the other. According to Morris and Jacobs (1950) it was probable that the somatotype was a reflection of the inter-relationships between structure and function of an individual.
Physique classification has been of interest as far back as 400 B.C. when Hippocrates proposed his 'phthisicus' and 'typus apoplecticus' dichotomy (Morris and Jacob,1950).
Somatotyping can be thought of as a numerical shorthand method of describing physique. A current somatotype method used is the one developed by Sheldon et al. in 1940 which is based on the assumption that every physique can be described in terms of three basic components: endomorphy, mesomorphy and ectomorphy. Each component has specific characteristics i.e. endomorphy applies to certain body characteristics which

differ from those of mesomorphy and ectomorphy. The Sheldon somatotype is thus concerned with body shape only : it is not influenced by size, and assesses aspects of physique that are assumed not capable of change. According to Sheldon et al. (1940) a person's somatotype therefore does not change with age, nutrition or state of training. The Sheldon somatotype method is subjective since it involves comparisons and ratings using the standard somatotype photographs.

A more frequently used adaptation of the somatotype method is the anthropometric method of Heath and Carter (1967). The Heath-Carter method expresses present morphological conformation in three primary components of physique which describe human variation. The first component (endomorphy) refers to the relative fatness. The second component (mesomorphy) refers to relative musculo-skeletal development. The third component (ectomorphy) in turn refers to relative linearity of individual physiques. Third component ratings are based on height/$\sqrt[3]{\text{weight}}$ ratios. The Heath-Carter method is more objective than the Sheldon method since it makes use of anthropometric measurements.

It has eliminated extrapolation of age and established a linear relationship between somatotype ratings and height /$\sqrt[3]{\text{weight}}$ ratios (Heath and Carter,1967). Low first component ratings signify physiques with little non-essential fat. Low second component ratings signify light skeletal frames and little muscular relief. High third component ratings signify linearity of the body as a whole.

The mean somatotype of the Olympic marathon runners who took

part in the 1968 U.S. Olympic marathon was 2.6;4.4;3.9 (Tanner,1964).

Carter and Heath (1990) calculated the mean somatotype of marathon runners of the 1960, 1968 and 1976 Olympic games to be 1.4;4.4;3.4.

Having a small and lean physique has been recognised as one of the most important attributes of a successful marathon runner since the late nineteenth century (Costill,1979 cited by Tanaka and Matsuura,1982). The small and lean physique implies a low percentage of body fat (%BF). Male marathon runners have been measured to have 6% to 8% body fat but lower levels have been recorded. A 3% body fat content has been estimated as being the lowest %BF and is known as essential body fat (Pollock et al., 1977).

Endurance runners derive an advantage from having slender muscles (Tanner, 1964) because slender muscles require less blood to perfuse them than bulky muscles. Bulky muscles would be counterproductive in endurance running since the entire body mass must be displaced horizontally for a long duration (Withers et al.,1986).

The initial fuel source for long distance running is carbohydrate. Later fatty acids are utilized via beta oxidation. The higher the % body fat the lower the ability to release free fatty acids (FFA) from adipose tissue, which implies a lower metabolic activity of a certain volume of adipose tissue. This then provides less lipid metabolites as energy substrate. The adipose tissue of trained distance runners thus has a more rapid turnover rate and a higher

metabolic activity, allowing for the utilization of the little fat that they do contain, for extra energy. It has also been shown that endurance runners have a somewhat higher blood flow to the splanchnic region, where important fat depots are situated. There seems to be a critical % body fat where having less is actually depriving the body of stored triglycerides. On the other hand having too much fat is detrimental because it means carrying excess weight (Burke and Read,1989).

For many ultramarathoners the limiting factor affecting performance is the supply of fuel to the muscles. When the supply of carbohydrates and FFA is insufficient protein is catabolized so that the amino acids can be used as substrate during gluconeogenesis. The gluconeogenesis occurs in the liver and the glucose is then transported to the active muscles (Guyton,1981; Ganong,1985).

Pollock et al., (1977) studied elite and good male distance runners and compared them to each other and to untrained, lean men. They found that the elite runners were shorter in stature and had less body mass and greater body density. The 20 elite runners had a mean height of 1.77 (±0.06) m. Their mean body mass was 63.06 (±4.80) kg and the mean body density was 1.08859 (±0.00749). The mean % body fat was 4.7 (±3.1)%. Tanaka and Matsuura (1982) studied male middle and long distance runners aged 16 to 22 years (x 19.0). The mean stature was 1.70 (±0.046) m. Their mean mass was 57.3 (±5.5) kg and the mean density was 1.076 (±0.003). The lower

age limit of 16 years may have been a reason for the lower mean statures and masses measured.

Skinfold estimates of body fat made on the 114 male marathon runners measured in 1968 (Costill,1972) showed that these athletes possessed a mean of 7.5% body fat. In fact several of the top finishers had less than 5% body fat. In order to assess % body fat for individuals, the body density has to be calculated first. This is done by adding the four skinfolds -biceps, triceps, subscapular and supra-iliac, and then using linear regression equations derived by Durnin and Womersley (1974).

PHYSIOLOGICAL PARAMETERS

CORTISOL

When serum cortisol concentrations are measured it is difficult to determine whether the observed changes are as a result of changes in secretion or simply reflect a modified distribution in the different pools (Dazord et al.,1972). Previous studies have reported changes in serum cortisol concentrations in response to acute and chronic exercise (Kraemer et al.,1989;Keul et al.,1981;von Glutz et al.,1978). Davies and Few (1973) reported that serum cortisol concentrations only increased when subjects were exercising at more than 65% $\dot{V}O_2$max for an hour. Exercise intensities below 65% $\dot{V}O_2$max generally showed a drop in serum cortisol concentrations. But the exercise was acute, lasting for less than one hour.

According to Few (1974) exercise increases the rate of uptake of cortisol by peripheral tissues. When there is an increase in work load above a certain critical level, it stimulates the adrenal cortex to secrete a massive amount of cortisol. Cortisol is not stored in the adrenal gland. The amount secreted is the amount produced (Haynes and Murad,1975). This increased serum level of cortisol also promotes continual uptake of cortisol by the peripheral tissues. But exactly what this critical work load is, is as yet unclear. There is also uncertainty about the relationship between intensity and duration of the work load.

Noakes and Carter (1976) measured a whole range of biochemical and haematological parameters on athletes running 160 km. They did not however measure cortisol. McKechnie et al., (1982) measured the metabolic response to a 90 km running race, but did not measure cortisol. Krebs et al., (1983) measured the acute and prolonged effects of marathon running on 20 blood parameters, but not cortisol.
According to Krebs et al. (1983) runners have a different blood chemistry to non-runners. Therefore, when blood results of endurance trained athletes are interpreted, they must be seen in the context of the changes that occur during endurance running, otherwise they may falsely indicate a diseased state.

Exercise causes an increase in sympathetic nervous activity and several hormones have been shown to increase in concentration in blood (Bunt,1986; Keast et al.,1988). An increase in sympathoadrenal activity is the most immediate widespread exercise response. The concentrations of catecholamines secreted increase proportionally with increased workload and duration (Keast et al.,1988). Bunt (1986) reported that catecholamines have an effect on cardiorespiratory function, thermoregulation, lipolysis, glycolysis and the production of glucose in the liver.

Cortisol is a steroid hormone produced by the adrenal cortex. A cortisol molecule contains 21 carbon atoms and is therefore referred to as a C21 steroid. Various hydroxyl (-OH) groups

are attached to the carbon atoms, at positions 11, 17 and 21 (Gower,1979).

The release of cortisol into the blood is controlled by adrenocorticotrophic hormone (ACTH). ACTH is released from the anterior pituitary gland and is dependent on corticotrophin releasing factor (CRF) which is produced in the hypothalamus. The hypothalamus, anterior pituitary gland and the adrenal cortex together constitute what is known as the hypothalamic - pituitary - adrenal axis.

Cortisol in blood is normally bound to a specific transport protein known as cortisol-binding-globulin (CBG) or transcortin, and to albumin. When cortisol is bound to protein it is inactive. Only free cortisol is active (Zilva and Pannall,1978; Gower,1979; Guyton, 1981). Under resting conditions plasma cortisol half life is 60 to 90 minutes and more than 96% of cortisol is bound to protein. This is a reversible process. The CBG binding sites are saturated when cortisol concentration is more than 552 $nmol.\ell^{-1}$ (McCarthy and Dale,1988).

This is an average value. When the amount of cortisol is increased it remains unbound i.e. free. It is this free cortisol that is biologically active. Therefore under resting conditions cortisol will be eliminated if the concentration is greater than 552 $nmol.\ell^{-1}$. However, the amount of CBG varies from person to person. Other plasma components and changes in body temperature can all affect CBG. In other words there is no simple relationship between the rate of secretion of cortisol and its concentration in the plasma

(McCarthy and Dale,1988). Also, serum cortisol concentrations are subject to circadian rhythmicity. At about 08h00 the level is at its highest and at about 20h00 it reaches its lowest level (about 110 nmol.ℓ^{-1}) (McCarthy and Dale,1988). The cortisol molecule forms a steroid - receptor complex with a receptor protein in the cytoplasm of cortisol sensitive cells. The complex then changes shape and moves through the cytoplasm to the nucleus. The cortisol then binds to the DNA in the nucleus so the RNA is transcribed and specific proteins are synthesized. Cortisol has an anabolic effect on tissues such as the liver. However, in tissues such as fibroblasts and lymphoid cells the proteins synthesized are catabolic (Haynes and Murad,1975).

Cortisol has the following effects:

(i) it opposes insulin and promotes gluconeogenesis

(ii) it enhances lipolysis

(iii)it stimulates protein breakdown

(iv) it helps to maintain extra-cellular fluid volume and therefore also blood pressure because it enhances the kidney's ability to excrete a water load.

At least 70% of the cortisol secreted is metabolized by the liver. The kidney excretes most of the metabolites of cortisol as 17-hydroxycorticosteroids, 17-ketogenic steroids and 17-ketosteroids. Biliary and faecal excretion is minimal (Guyton,1981).

One of the symptoms of an insufficiency of cortisol, eg Addison's disease, is weakness and fatigue resulting in a

decreased work capacity of skeletal muscle. However, abnormally high concentrations of cortisol, eg in Cushing's syndrome, may lead to atrophy of skeletal muscle with subsequent weakness and fatigue (Haynes and Murad,1975). Thus chronic abnormalities of cortisol, regardless of whether it is a deficiency or an abnormally high concentration, result in chronic weakness and fatigue.

Patients with Cushing's syndrome often have polycythaemia whereas Addison's disease patients usually present with a mild normocytic, normochromic anaemia. Thus cortisol has a tendency to increase haematocrit and haemoglobin concentrations. But this is a long term effect - over many months (Haynes and Murad,1975).

Patients with Addison's disease are often apathetic, depressed, irritable and some may even be psychotic. Treatment with cortisol is very effective and usually restores the behaviour and psyche. In fact some patients become euphoric, restless and insomniac. Patients suffering from Cushing's syndrome often have neurotic and psychotic spells. These spells usually disappear when Cushing's syndrome is effectively treated (Haynes and Murad,1975).

It is important to realize that increased levels of a hormone in serum does not necessarily mean that there was an increase in secretion of that hormone. The increase may also be due to a decrease in plasma volume (Bunt,1986). Therefore it was important in the present study to calculate the change in plasma volume.

PLASMA VOLUME

One of the most important factors involved in maintaining prolonged exercise is the body's ability to maintain sufficient plasma volume despite a situation of dehydration (Noakes,1988).

In order to maintain homeostasis the fluid intake must be equal to the output from the body. When the output is greater than the intake it is imperative to maintain plasma volume. Noakes and Carter (1976) assumed that their 160 km runners were dehydrated because they had lost weight and had increased concentrations of urea and creatinine and had increased haematocrits (Hct). They did not explain why certain other blood constituents varied in a 'haphazard manner'. Perhaps if they had measured or predicted plasma volume they might have come to a different conclusion.

In order to calculate plasma volume changes Hct had to be measured before and after the race. Exhaustive exercise has been shown to cause decreases in Hct when compared to initial levels (Szygula,1990). Intense short duration exercise results in a reduction of plasma volume which may mask the loss of erythrocytes (Szygula,1990). Thus if blood samples are taken immediately after the short duration exercise, there may seem to be no change or an increase in Hct. Reinhardt et al. (1983) reported that long distance running results in premature elimination of old erythrocytes from the circulation resulting in haemolysis and sequestration by the

spleen. This elimination results in a decreased Hct. After long duration exercise the haemolysis and erythrocyte sequestration by the spleen may not be effectively masked because the plasma volume may be recovering from its initial reduction (Szygula,1990).

The average life span of erythrocytes is about 120 days. After that they tend to be lysed in the spleen, liver or bone marrow (Guyton,1981). Robertson and associates (1988) suggested that long distance running may accelerate the aging process of erythrocytes causing them to be lysed prematurely. 'Foot-strike haemolysis' is the term used to describe the mechanical trauma the erythrocytes may undergo in the capillaries of the feet when the foot strikes the ground (Szygula,1990). According to Falsetti et al. (1983) the type of running shoe (hard- or soft-soled) and the surface one runs on can determine the amount of haemolysis that occurs during long distance running.

The contraction of large muscle groups for extended periods may cause erythrocytes in the capillaries to be compressed, which may lead to premature haemolysis (Szygula,1990).

Changes in blood viscosity, increased body temperature, high catecholamine concentrations and hypoglycaemia tend to decrease osmotic and mechanical resistance of erythrocytes which may cause haemolysis (Clement and Sawchuk,1984).

Approximately 60% of body mass is whole body fluid. This fluid can be compartmentalized anatomically and morphologically into :

(1) intravascular fluid : found inside the blood vessels i.e. plasma,

(2) interstitial fluid : found in the spaces between cells,

(3) intracellular fluid : found inside the cells (Guyton,1981).

Normally (1) and (2) are known collectively as extracellular fluid.

There is constant interchange of fluid and the substances dissolved in the fluid, between these compartments. Volumes of fluid compartments change during the day and are affected by fluid intake, salt intake, sleep and sleep deprivation, and oedema (Ritschel,1986).

Blood consists of plasma, red blood cells (erythrocytes), white blood cells (leucocytes) and platelets (thrombocytes) (Ganong,1985). One can calculate the total blood volume from the following equation :

$$V_b = V_p \times 100 \div \left[100 - (0.87 \times Hct) \right] \quad .....(1)$$

where V_b = volume of blood

V_p = volume of plasma

Hct = haematocrit

Therefore

$$V_p = V_b \div \left[100 \div (100 - 0.87 \times Hct) \right] \quad .....(2)$$

The volume of blood (V_b) can be calculated according to the following equation, which is specific for adult males :

$$V_b = (0.3669 \times \text{stature}) + (0.03219 \times \text{mass}) + 0.6041 \quad ...(3)$$

where stature is expressed in m and mass in kg.

V_b can then be substituted into equation (2) so that the volume of plasma can be calculated.

When water is absorbed from the gastro-intestinal tract it enters the blood, thereby becoming part of the plasma. This fluid cannot accumulate in the blood so there are 4 ways in which the body can redistribute it:

(a) to the kidneys to be excreted,

(b) to the skin where it is lost as sweat,

(c) to the lungs where it is excreted during expiration,

(d) to interstitial fluid, from which it is retrievable (Ritschel,1986).

At the beginning of exercise water moves out of the vasculature into the interstitial space, probably because of increased hydrostatic pressure in the blood vessels and an increased osmotic pressure in muscle tissue (Szygula,1990). Therefore during short term exercise this leads to haemoconcentration. However, during extended exercise at lower intensities the water moves back into the vasculature because of the haemoconcentration and subsequent increased osmotic pressure in the vasculature (Szygula,1990).

Much of the research regarding fluid shifts has been done

using ergometers, particularly those involving cycling (Miles et al.,1983). There seems to be a linear relationship between the intensity of cycling and the amount of plasma lost (Senay et al.,1980). However during weight-bearing exercise such as running there seems to be a low correlation between exercise intensity and plasma efflux (Miles et al.,1983). Hill et al. (1987) examined plasma volume responses in an Olympic distance runner in five consecutive days of running. They found a plasma volume expansion which they did not associate with a decrease in sweating. Roecker et al. (1989) measured plasma volume in marathon runners and found a reduction in plasma volume immediately after the race but an increase during the recovery period. The plasma volume was returned to pre-exercise levels an hour after the marathon although no fluid was ingested. This indicates that there are endogenous mechanisms responsible for maintaining plasma volume. Some studies have shown an increase in plasma volume due to prolonged exercise (Astrand and Saltin,1964; van Beaumont et al.,1973). In these cases subjects were permitted to ingest fluid during the exercise. Thus it seems that if sufficient fluid is ingested during the exercise the plasma volume may not decrease.

Haemolysis:

Controversial results have been found on the effects of exercise on haemolysis, depending on whether trained or untrained subjects were used, the intensity and duration of exercise, and the time of sampling after exercising (Lijnen et al.,1988). Haemolysis can be identified when the following

occur : haptoglobin levels decrease, free haemoglobin concentrations increase, red cell filterability is impaired and red cell creatinine is increased (Szygula,1990). When the amount of haemoglobin that is released into the plasma after haemolysis exceeds the amount of haptoglobin, the excess haemoglobin is excreted by the kidney, resulting in haemoglobinuria (Miller,1990). The occurrence of haemoglobinuria as a result of haemolysis brought on by exercise has been of particular interest. Exhaustive exercise was shown to cause intense haemolysis of erythrocytes, and depending on the duration, resulted in marked changes in haematological parameters (Szygula,1990).

Leucocytosis:

There is increasing interest in the effect exercise has on the immune system. Of particular interest is the effect long distance running has on changes in immune function. Peters and Bateman (1983) found that marathon runners were significantly more susceptible to upper respiratory tract infections in the week following the marathon.

Various studies to date have shown that exercise results in an increase in the number of white blood cells (leucocytes) in the circulating blood (McCarthy and Dale,1988; Keast et al.,1988). This increase in leucocyte numbers is known as leucocytosis.

Leucocytosis ranging from 164% to 290% have been reported following endurance exercise (Moorthy and Zimmerman,1978; Davidson et al.,1987). The magnitude of the leucocytosis is dependent on both the intensity and duration of the exercise

(McCarthy and Dale,1988). Moorthy and Zimmerman (1978) suggested that the extent of the leucocytosis is inversely proportional to the degree of training. Nieman et al. (1989) found that the leucocytosis was less dependent on quantity of training or in fact performance ability, but rather on the frequency of training.

The leucocytosis has been positively correlated with increased concentrations in plasma cortisol, but the correlation with plasma catecholamine concentrations seems unclear (Moorthy and Zimmerman,1978). High concentrations of cortisol cause an increase in the number of certain leucocytes and a decrease in the number of others. Moorthy and Zimmerman (1978) found a high correlation between increased plasma cortisol concentration and circulating neutrophil counts under exercising conditions. There seems to be an increase in circulating neutrophils and a decrease in lymphocytes, eosinophils, basophils and monocytes (Guyton,1981). The neutrophils move from the bone marrow into the blood - often the neutrophils are still 'immature' (Haynes and Murad,1975). At the same time there is a decrease in the rate of removal of neutrophils. The decreased numbers of the other leucocytes is not because of the increased destruction but rather because of redistribution into certain tissues (Haynes and Murad,1975).

Neutrophils make up about 62% of all the leucocytes under resting conditions. The average life span of a neutrophil is about 12 hours (Ganong,1985). The bone marrow therefore produces billions of neutrophils per day. Neutrophils are

involved in the phagocytosis of bacteria, viruses and any other 'injurious agent' (Guyton,1981). They are attracted to an area of tissue damage by a process known as chemotaxis and move through the capillary pores by diapedesis (Ganong,1985). It seems that almost any factor that causes any type of tissue damage will cause an increase in circulating neutrophils (Guyton,1981). This increase in neutrophil numbers is known as neutrophilia. Cancers, acute haemorrhages and poisoning for example, all lead to neutrophilia. Thus the tissue damage does not necessarily have to be associated with inflammation.

Graphical representation of % increase in leucocyte count indicates that there is a massive increase in the first 15 minutes of exercise, which is consistent with the initial increase in catecholamine concentration (McCarthy and Dale,1988). Then there seems to be a steady increase, possibly as cortisol concentration increases.

Cortisol:

Cortisol can prevent inflammation, i.e. it can prevent local redness, heat, swelling and tenderness. Cortisol has the following effects :

(1) it stabilizes lysosomal membranes thereby decreasing the concentration of proteolytic enzymes,

(2) it decreases the permeability of capillaries,

(3) it decreases the migration of leucocytes to the inflamed area,

(4) it decreases phagocytosis because there are less leucocytes and therefore blocks increased release of

proteolytic enzymes,

(5) it suppresses the immune system by decreasing the antibody and sensitized leucocytes (Guyton,1981).

Cortisol has two effects on lipid metabolism. Firstly it causes mobilization of triglycerides from adipose tissue. Secondly it facilitates in lipolysis of the triglycerides mobilized from the adipose tissue (Guyton,1981). The triglycerides can then be catabolized and the energy utilized.

The ability to perform long lasting exercise depends largely on aerobic power and the supply of substrate to the working muscles. Cortisol stimulates gluconeogenesis in the liver, causing increasing circulating glucose concentrations (Wilks,1991). The gluconeogenesis occurs at the expense of free fatty acids and amino acids. The benefits of the extra energy are clear. However, when there is prolonged catabolism of muscle protein for this gluconeogenesis a negative nitrogen balance occurs together with a reduction in muscle strength and endurance (Wilks,1991). From this point of view the benefits of continuous cortisol secretion are questionable (Åstrand and Rodahl,1977). In fact, during long lasting exercise the blood levels of glucocorticoids and catecholamines may decrease to well below the initial levels. Often there is an increase before this decrease. Consequently the stress response of the pituitary - adrenocortical system has been divided into three phases :

(i) preliminary activation

(ii) subnormal activity

(iii) secondary activation (Viru,1983).

The preliminary activation occurs in response to the anticipatory stress and the initiation of exercise. The subnormal phase is associated with metabolic disturbances and the secondary activation phase is a direct response to the metabolic disturbances. The appearance of the subnormal activity phase depends on the state of the adrenal gland and on the level of fitness. Training elevates the functional stability of the pituitary - adrenocortical system. The subnormal phase is not a result of the exhaustion of the cells within the adrenal cortex that secrete cortisol. Rather, it is the result of a regulatory mechanism. It limits the duration of action of cortisol.

Consequently during prolonged exercise in the state of fatigue, the decrease in cortisol excludes the activity of the mechanism of mobilization of energy reserves and thereby avoid the fatal depletion of the body's plastic resources. Normally cortisol reaches its threshold level during intensities of 50 to 75% $\dot{V}O_2max$. However, long duration exercise can cause an increase in cortisol concentration even when the intensity is below 50% (Viru,1983).

Various studies have shown increased cortisol levels after long runs. Semple et al. (1985) tested marathon runners and found cortisol concentrations at the end of the race to be five times the initial pre-race levels. Dearman and Francis (1983) measured cortisol concentrations after a marathon and found cortisol levels to be 200% of pre-race levels. Keul et

al., (1981) observed after a 100 km run that cortisol levels were significantly increased.

Hypoglycaemia induced by long duration exercise is counteracted by high concentrations of serum cortisol concentrations. Hypoglycaemia affects the brain directly since the brain utilizes only glucose as a fuel source (Guyton,1981). Hypoglycaemia initially causes confusion and if not treated may lead to the loss of consciousness (Guyton,1981).

The brain has cortisol - binding receptors and therefore it has been postulated that cortisol may have a direct effect on the brain. In fact experimental chronic administration of cortisol has been found to increase brain excitability (Haynes and Murad,1975). Thus cortisol may reduce confusion even in circumstances of hypoglycaemia.

Circadian rhythmicity:

Circadian rhythmicity is an important element of human physiology that must be kept in mind when measurement of clinical indices, performance and mood are made (Moore-Ede et al.,1983). For example, body temperature is lowest in the early morning and reaches a peak in the early afternoon. In contrast, epinephrine and norepinephrine concentrations (and therefore alertness) normally reach a maximum at about midday.

Circadian rhythms also differ in the degree to which they are exogenously and endogenously controlled. Norepinephrine is largely exogenously determined, whereas body temperature and

epinephrine are endogenously determined.

Without time or light-darkness cues the endogenous circadian timing system adopts a 25-hour cycle (Oatley and Goodwin,1971).

Moore-Ede et al. (1983) proposed that under normal conditions physiologic systems receive signals from two 'pacemakers' : the first is the one that is driven by the body temperature, and the second is driven by the rest - activity cycle. The circadian rhythms observed in any one variable are therefore the net result of signals from both 'pacemakers'.

Ambient temperature, barometric pressure and a host of other physical measures all vary diurnally and can act as 'Zeitgebers' (a German neologism that means 'time-giver'). The most reliable index of environmental time is the regular diurnal alteration of light and darkness (Haus et al.,1983).

The impact of the endocrine system on human circadian rhythmicity, and of circadian rhythmicity on the endocrine system is largely unknown (Roelfsema,1987). The secretion of cortisol is thought to show both an endogenous and a sleep related component (Weitzman,1976). Although cortisol circadian rhythmicity does not seem to be affected significantly by sleep deprivation, the concentrations are.

Haematocrit and white blood cell count both follow circadian rhythms. Haematocrit is highest at about 08h00 and lowest at 24h00, whereas white cell count is highest at about 24h00 and lowest at 12h00 (Scales et al.,1988).

'Zeitgebers' are distorted during sleep deprivation, and this

may lead to disturbances in circadian rhythmicity. It is often suggested that the sleep deprived person is aroused by a brief burst of exercise. However, prolonged exercise causes a deterioration in performance and mood (Johnson et al.,1977).

Sleep deprivation:

The exact biological role of sleep is not clear. In normal human beings 'true insomnia' does not seem to last longer than 48 hours. In fact, according to the medical literature 'no-one has ever died of insomnia' (Karadzic,1972). Sleep deprivation research is complicated by the fact that there is no placebo condition and the subject always knows that he/she is being sleep deprived. Thus the results are always affected by the motivation of the subjects. Also, fatigue is always associated with sleep deprivation, and this makes it difficult to identify whether the results were due to insomnia or fatigue.

When athletes have to exercise for a long period of time without sleep, such as in the present study, this causes functional disturbances. There are however differences in sensitivity to sleep deprivation. According to Cappon and Banks (1960) neurotic extroverts are more vulnerable to sleep deprivation. In theory sleep deprivation has the following effects :

(1) it causes lapses in attention known as 'micro-sleep',

(2) it increases glycolysis which may eventually result in decreased activity of enzymes involved in aerobic metabolism,

(3) it increases plasma volume and causes increased peripheral distribution of blood volume,

(4) it decreases haematocrit and haemoglobin concentration,

(5) it has resulted in contradictory results regarding plasma cortisol concentrations,

(6) it increases rates of perceived exertion at any heart rate,

(7) it decreases cortical arousal and motivation, and causes a 'deterioration' in mood (Shephard,1984).

The above mentioned factors usually result in a decrease in performance.

PSYCHOLOGICAL PARAMETERS

The anthropometric and physiological characteristics of distance runners have been studied numerous times. However psychological indicators are also important, and studies linking all three domains are rare. In the past short-term and long-term benefits of running have been reported in clinical and normal populations (Berger and Owen,1988). In addition psychological indicators have been used to distinguish successful from unsuccessful athletes (Thomas et al.,1983).

Jones and Hardy (1989) cited an interesting approach to the relationship between sport and stress, as developed by Patmore in 1986. It assumes that the main motivational factor for the participation in sport is the human need to overcome stress. Ultramarathoners are subject to an arduous challenge, both physically and psychologically. It seems that successful ultramarathoners possess the physical capacity and the willingness to tolerate the discomfort associated with the hard work (Morgan and Pollock,1977). Because ultramarathon runners must persevere for an extended period of time, their psychological states can be expected to have an important impact on performance.

RATINGS OF PERCEIVED EXERTION (RPE)

Bartley and Chute's (1947) model of fatigue recognises three categories : objective fatigue, subjective fatigue and physiological fatigue. Subjective fatigue is best quantified by using Ratings of Perceived Exertion (RPE).

According to perceptual psychologists, perception is a process that is not associated with conscious awareness (Mihevic,1981). Perception of effort involves the integration of signals and feelings into a subjective estimate of effort that can be quantified (Birk and Birk,1987;Carton and Rhodes,1985). Perceived exertion is a complex phenomenon which seems to have both psychological and physiological input (Morgan,1973). Carton and Rhodes (1985) divided the factors that contribute - positively and negatively - to RPE into two categories :

1. physiological
2. non-physiological.

A brief discussion of the different factors and their effect on RPE follows.

1. Physiological factors :

a) Heart rate : Borg's (1970) rating scale yields high correlations with heart rate during exercise of varying intensities. But Mihevic (1983) found that this scale could not discriminate between people of varying "fitness" when the intensity of the exercise was low or moderate. Only during

high intensity exercise were there any significant differences in RPE.

b) Minute ventilation : Increases in minute ventilation and/or respiratory rate result in increased RPE. These are considered to be central signals. In fact dyspnoea is the only consciously monitored central signal (Robertson,1982).

c) Metabolic demand : an increased metabolic demand implies an increased energy expenditure. The intensity and duration of the activity determine whether the increased metabolic rate is achieved under aerobic or anaerobic conditions. Under aerobic conditions the increased metabolic demand is identified by an increase in $\dot{V}O_2$. Under anaerobic conditions the increase is identified by lactate accumulation (Ganong,1985). As $\dot{V}O_2$ increases and/or lactate accumulates, RPE seems to increase accordingly (Carton and Rhodes,1985).

d) Strain : the amount of strain that active muscles are under is related to the force and velocity of contractions. During exercise lasting less than 30s the strain due to the muscle contractions is dependent only on force and not on frequency of contraction. During exercise lasting longer than 30s both force and frequency of contractions become important (Cafarelli,1982). RPE increases as the amount of strain experienced increases (Carton and Rhodes,1985).

e) Blood lactate : as explained previously blood lactate accumulates when the metabolic demand is such that energy is converted under anaerobic conditions. Since lactate only starts accumulating at around 65% $\dot{V}O_2max$ the contribution of lactate to RPE is substantial at high intensity but minimal at low intensity exercise (Mihevic,1981). Both blood lactate and RPE increase when plotted against time during incremental exercise (Carton and Rhodes,1985).

f) Circulating catecholamines : when the sympathetic nervous system is stimulated, epinephrine and norepinephrine are released from the adrenal gland into the blood stream (Guyton,1981). Epinephrine and norepinephrine have almost the same effect as direct sympathetic stimulation, but the effect lasts much longer because the hormones are removed slowly from the blood (Ganong,1985). Thus, high circulating catecholamine concentrations correlate well with high RPE values. Perhaps catecholamines are more related to the perception of fatigue than to the perception of effort (Carton and Rhodes,1985).

2.Non-physiological factors in RPE :

a) Time of day : a fundamental property of living organisms is the phenomenon of circadian rhythmicity (Roelfsema,1987). It is as yet unclear whether RPE also has circadian rhythmicity. It seems that sub-maximal exercise RPE is independent of time of day, but that maximal exercise RPE

values are lower in the morning than in the afternoon (Hill et al.,1989).

b) Sleep deprivation : as mentioned previously, sleep deprivation and fatigue cannot be separated from each other (Shephard,1984). So the effect sleep deprivation has on RPE is always compounded by the effect fatigue has on RPE. However, sleep deprivation - and fatigue - have been shown to affect depression and confusion, which both influence RPE (Carton and Rhodes,1985).

c) Personality : personality type may affect how subjects perceive effort and indeed fatigue (Carton and Rhodes,1985). Often effort and the resulting fatigue are accompanied by pain. Although pain is a physiological response, the perception of pain varies from individual to individual. Extroverts seem to have a higher pain tolerance than introverts (Eysenck,1967;Morgan,1973;Rejeski,1985). Therefore under similar circumstances resulting in pain, extroverts tend to have lower RPE values than introverts. Also, extroverts seem to perceive exercise intensity as less strenuous than introverts (Carton and Rhodes,1985).

d) Psychological disposition : much research has involved physiological foundations of RPE but it seems that psychological dispositions are also important (Rejeski,1981; Borg,1982). Perceptual, cognitive and affective factors together with physiological factors form a complex

interrelationship (Morgan,1985). Cognitive strategies such as "association" and "dissociation" are common amongst distance runners (Morgan,1981).
"Association" involves concentrating totally on running and 'how the body feels'. "Dissociation" involves concentrating on anything other than running (Noakes,1988). RPE values tend to be lower when the runner is "dissociating".

e) Age : In males, $\dot{V}O_2max$ increases until about the age of 18, then it tends not to change much as a result of age until about the age of 30. Thereafter $\dot{V}O_2max$ declines at roughly 1% per annum (McArdle et al.,1986). Thus as people get older, they tend to produce increased RPE responses during maximal exercise (Carton and Rhodes,1985).

f) Environmental factors : when individuals are in an environment in which they gain heat from the environment, RPE increases. More blood has to be shunted to the skin in an effort to dissipate heat, and in the process blood flow to the active muscles tends to decrease, resulting in higher perceptions of effort (Carton and Rhodes,1985).
Pollution and increasing altitude both tend to increase RPE under any given work load because they both create a decrease in PO_2. Under conditions of a decreased PO_2, respiratory rate tends to increase and as mentioned previously respiratory rate is consciously monitored and has a direct effect on RPE.

g) Exercise intensity : as exercise intensity increases so do heart rate, $\dot{V}O_2$, and minute ventilation. Concomitantly there is an increased RPE (Pandolf,1978). Rejeski (1981) proposed that in exercise of near-maximum intensity, physiological inputs are the main sources of information for RPE. However, at prolonged submaximal exercise levels, there is an increased probability that psychological factors are the main cues for RPE.

h) Duration : during exercise lasting less than 30s the frequency of muscle contraction is less important than the force of contraction in mediating the strain felt in the muscle. However, when exercise lasts longer than 30s, both frequency and force of contraction become critical to the amount of strain in the muscle (Cafarelli,1982). Generally speaking, as duration increases, so does RPE, even during steady-state conditions (Carton and Rhodes,1985).

i) Motivation : according to Braun and Linder (1979) motivation is responsible for the initiation, direction and persistence of behaviour, and may therefore affect RPE. People perform best when they are motivated (Oxendine,1970). Rejeski (1981) found that RPE values were higher when subjects succeeded and lower when they failed.

Ekblom and Goldbarg (1971) suggested that a two-factor model of RPE be used :

(1) local RPE : in which feelings involving mainly the active muscles are essential, and

(2) central RPE : in which feelings involving mainly the cardiovascular and pulmonary systems are rated.

According to Pandolf (1982) the following variables are germane to the 'local RPE' category :

(i) local muscular discomfort or pain

(ii) blood lactic acid

(iii) mechanoreceptor stimulation

(iv) chemoreceptor stimulation

(v) proprioceptive factors

(vi) catecholamine secretion

(vii) local fatigue

(viii) Golgi tendon organ activity

The following variables fall into the 'central RPE' category:

(i) respiratory rate

(ii) minute ventilation

(iii) oxygen consumption

(iv) heart rate

(v) dyspnoea

(vi) hypoxia.

This classification must be used with reservation because it is difficult to measure many of the components.

According to Ekblom and Goldbarg (1971) when small muscle groups are being used the local RPE factor dominates. When large muscle groups are used, central RPE dominates. However,

research done by Pandolf (1982) indicates that local components are responsible for the most intense sensory stimuli and that this is independent of the size of the muscle mass involved.

This two-factor model can be adapted into a three-factor model, where the third factor is an overall rating, which may be independent of the other factors. Thus it is possible, for instance, to have a low local and central rating along with a high overall rating, and vice versa. The third rating takes factors other than local and central RPE into account, for instance psychological cues.

EYSENCK'S PERSONALITY INVENTORY (EPI)

A situation that creates strain in one person may not have the same effect in another. The individual's perception of a situation is dependent largely on personality dimensions (Jones and Hardy,1989).

Initially Eysenck developed a questionnaire consisting of 48 questions. However, this proved to be tedious, so it was reduced to a "shortened version" which has 12 questions. The questions are structured in such a way that the only possible answers are 'yes' and 'no'. Each 'yes' is marked +1 and each 'no', -1. Six questions relate to neuroticism-stability and six to extroversion-introversion. Thus the possible range of scores is from +6 to -6 (Eysenck,1958).

These psychological dimensions are thought to be enduring personality traits and therefore in the present study there

was no need for re-testing. While personality is considered to be relatively stable, 'mood' is a more transient state. According to Underwood et al. (1980) people are able to ignore changes in moods when asked to report on their typical behaviour during personality testing. Mood seems to have no "causal effect" on the determination of personality. Eysenck and Eysenck (1963) linked the extroversion-neuroticism dimension of personality with heredity. They also reported that the marathon runners had lower extroversion scores than their group of normal subjects. Morgan et al. (1988) found that a sample of elite US male distance runners possessed normal values for extroversion and neuroticism. Morgan and Costill (1972) found no significant correlation between performance in a marathon and extroversion and neuroticism. Morgan and associates (1988) studied elite US distance runners and found them to have extroversion and neuroticism scores that were well within the range for the population average. They also reported an inverse relationship between neuroticism and performance in long distance running. In other words, the better performing runners had lower neuroticism scores. Extroversion and running performance were not significantly correlated.

Johnson (1982) stated that there seemed to be no relationship between extroversion or neuroticism and one night's sleep deprivation. However, when 60 hours of sleep were 'lost', extroverts were affected more than introverts.

As established earlier, running 160 km is deemed the 'stressor' that will produce a 'strain' response.

Experiencing the 'strain response' is different from being in a state of arousal. The 'strain response' usually results because the demands of the situation are not the same as the ability to cope. Arousal on the other hand, is a continuum often referred to as wakefulness, vigour and alertness (Cox,1985). A challenge or demand may alter the state of arousal, but does not necessarily have to create a 'strain response' (Cox,1985). Arousal is a combination of drive, tension and activation (Landers,1980). Changes in drive, tension and/or activation result in changes in arousal. Yerkes and Dodson (1908) developed an "Inverted-U" theory of arousal and performance whereby an increase in arousal initially results in increased performance, but only to a certain point. Thereafter, performance decreases with an increase in arousal. Thus to optimize performance the level of arousal must also be optimal.

PROFILE OF MOOD STATES (POMS)

Colloquial use of the term 'mood' often implies a weak emotion (Mackay,1980). Moods do not refer to events, things or people - they are slowly changing 'states', not traits, which have no object reference (Ewart,1970). Moods are sensitive to the 'well-being' of the person and take into account information relating to the physiological and psychological status of the individual (Mackay,1980).
Exercise may be used as a 'stress reducing technique' (Berger,1984). The question that arises is : if a certain amount of exercise is beneficial, will twice as much be more beneficial?

Several studies have been conducted on the mood states of marathon runners. Morgan and Pollock (1977), using the Profile of Mood States (POMS) test developed by McNair and associates (1971), found that elite marathon runners had psychological characteristics that were different from the normal population. The POMS test measures "tension", "depression", "anger", "vigour", "fatigue" and "confusion". Morgan and Pollock (1977) found that elite runners had higher scores than normal college students in "vigour", and lower scores in the other five mood states. When these results were graphically illustrated they presented an "iceberg" shape, giving rise to the so-called "Iceberg Profile". Elite distance runners have consistently been found to possess this "iceberg profile" (Morgan et al.,1988). It has been shown,

however, that average runners also possess the "iceberg profile" (Thomas et al.,1983). An inverted "iceberg profile" is usually associated with poor mental health, or "burn-out" (Cockerill et al.,1991).

Moods, by definition, are emotional states that change. Moods can be affected by a number of personality and environmental factors. Therefore it is important to distinguish between POMS profiles obtained long before the competition and those obtained just before the start. According to Cockerill et al. (1991) it is necessary to determine the POMS profile as close to the start as possible.

It is important to realize that POMS is better suited to individual testing than to group testing (Cockerill et al.,1991). Individuals vary markedly in various emotions. For example, an increased level of "tension" seems to be a prerequisite for success in competitive running. In other words, the successful athlete may well be an 'active tension-seeker' which may differentiate him from his equally fit opponents. This would be indicated in the individual's POMS profile but could perhaps be 'lost' in the group profile.

In 1980 Wilson and associates chose 10 marathon runners, 10 joggers and 10 non-exercisers and measured their mood states according to the POMS test. They found that the marathon runners were less "depressed", less "angry", less "confused" but had more "vigour" than the joggers. There appeared to be no significant differences in "tension" or "fatigue" even though the marathon runners trained more often and ran further than the joggers. Both the marathon runners and the

joggers were significantly less "depressed", less "angry", less "confused" and possessed more "vigour" than the non-exercisers. However, these authors were unable to determine whether the differences were due to the amount of training done, different personality-types or as a result of differing expectations of and from running.

Tharion et al. (1988) tested entrants in the 1986 Massanutten Mountain Massacre 50-mile Trail Run in Virginia. They found these ultramarathoners to possess "iceberg profiles" similar to those that had been identified by Morgan and Pollock in 1977. POMS was tested before and after the event and then comparisons were made. This 50-mile (80 km) ultramarathon had a definite effect on all mood states except "anger" which was not changed significantly by the ultramarathon. The level of "tension" was significantly less after the race. Tharion et al. (1988) explained that this was probably due to the anticipation and unpredictability of the race. Once the race had been completed this "tension" had been alleviated.

The runners of this ultramarathon also experienced increased feelings of "depression" after the race. This was in contrast to Markoff et al. (1982) and Kavanagh et al. (1977) who found reductions in "depression" after running. The reason for this difference is probably the duration of the race which took 10 to 30 hours as opposed to workouts of less than an hour. Pre- and post-race levels of "vigour" were significantly different, where the post-race decrease in "vigour" was probably a direct consequence of the metabolic cost of running 80 km. Consequently, the level of "fatigue" was

significantly greater after the race. Metabolic cost and mental persistence probably accounted for the "fatigue" experienced. The ultramarathoners also reported feelings of "confusion" after the race. This is probably related to the increased feelings of "depression" as reported.

Tharion et al. (1988) also compared finishers to non-finishers. The only significant difference was that finishers experienced greater levels of "fatigue", having run further and for a longer period of time compared to the non-finishers.

Another factor that needs consideration is sleep deprivation, which could have a significant effect on mood states. Schomer (1990) cited Haslam (1984) as having shown that sleep deprivation has a greater psychological than physiological influence. The consequences of sleep deprivation and a lack of rest breaks cause deteriorating performance and therefore have a negative effect on mood states (Schomer,1990). This needs to be taken into account when comparing pre-race and post-race POMS scores.

CHAPTER THREE

MATERIALS AND METHODS

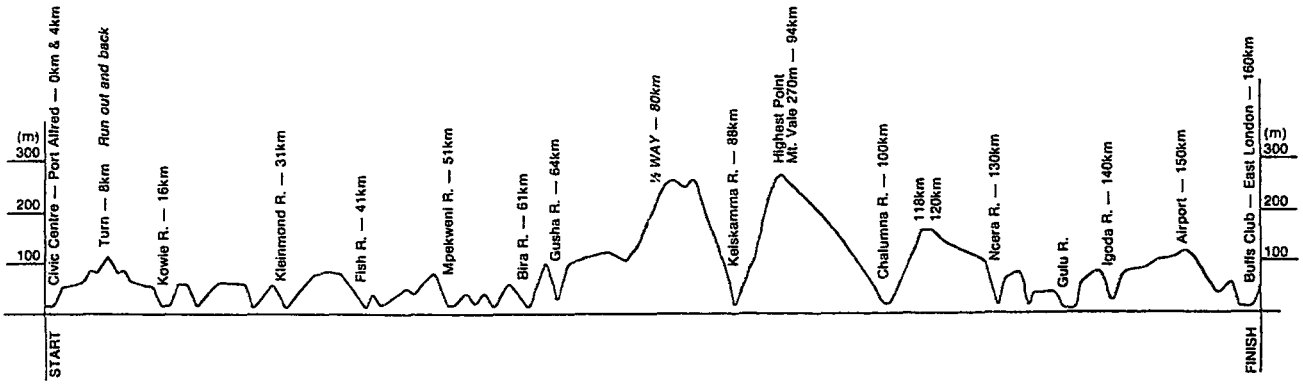
INTRODUCTION

Twenty one male Caucasian long distance runners volunteered to participate in this study. All were experienced ultramarathoners since one runner had completed 5 ultramarathons, 4 had completed between 5 and 10 and 16 had run more than 10. Each subject was required to complete an informed consent form (Appendix 1) before participating.

There were two testing sessions, one before the race and one within 15 minutes of completing the race.

ENVIRONMENTAL CONDITIONS

The Washie 160 km ultramarathon was held on 10 and 11 July 1992 and began at 19h00. The race started in Port Alfred and finished in East London. The following is a course profile of the race :



All subjects were exposed to the same overall environmental conditions although total time of exposure varied with performance.

INDIVIDUAL DIFFERENCES

There was no standardization of training, preparation, motivation, nutrient intake or clothing worn. Each subject was required to have his own seconds helping him through the race.

THE RESEARCH PROTOCOL

ANTHROPOMETRY

The following anthropometric measurements were obtained from each subject before the race :

stature (cm), body mass (kg), biceps skinfold (mm), triceps skinfold (mm), subscapular skinfold (mm), supra-iliac skinfold (mm), calf skinfold (mm), humeral bi-epicondylar diameter (cm), femoral bi-epicondylar diameter (cm), upper arm circumference (cm), calf circumference (cm).

After the race stature and body mass were again measured.

The specific procedures for each measurement are outlined in

detail below.

1. Stature : was measured using a Holtain stadiometer. Each subject was asked to position himself such that his bare feet, buttocks and upper back were in contact with the stadiometer. The arms were pendant and the head was in the Frankfurt horizontal plane. The vertex in the median sagittal plane was used to measure stature and was recorded in centimetres to the nearest millimetre (Tanner, 1964).
2. Body Mass : the subject was asked to remove all clothing except his shorts before being measured on the Seca scale. Body mass was measured in kilograms to the nearest gram.
3. Skinfold Measurements : Harpenden skinfold calipers were used to measure skinfolds in millimetres to the nearest 0.1 mm. The fold of skin was held between the thumb and the forefinger while the calipers were positioned to measure that fold. The dial was permitted to stabilize before taking the measurement.

   All measurements were made on the right side of the body. Every measurement was made three times. If the discrepancy between the readings was more the 3% then the measurements were made again.

Skinfold measurements were made following Tanner's (1964) general method - as outlined below.

a.) Biceps : a skinfold was taken anteriorly, superficial to the biceps femoris muscle midway between the acromion and the olecranon. The

subject was standing and the arm was pendant.

b.) Triceps : a skinfold was taken posterior to the previously measured biceps midway between the acromion and the olecranon.

c.) Subscapular : this skinfold was taken below the inferior angle of the scapula in an oblique plane (the plane extends inferiorly and laterally at approximately 45° to the horizontal). Again the subject was standing with his arms pendant.

d.) Supra-iliac : A skinfold was taken above the supra-iliac notch.

e.) Calf : the medial surface of the right calf was the site of this skinfold.

4. Bone Diameters : were measured using Holtain sliding calipers and were recorded in centimetres to the nearest millimetre.

a.) Humerus : with the forearm flexed at 90° the humeral bi-epicondylar width was measured and recorded.

b.) Femur : the subject was seated and the leg was flexed at 90° so that the femoral bi-epicondylar width could be measured and recorded.

5. Circumferences : two circumferences were measured using a Bendix measuring tape. Each measurement was made in centimetres to the nearest millimetre.

a.) Upper arm : the subject held his forearm at 90°

without flexing his biceps. The measurement was made where the circumference was maximum.

b.) Calf : with the subject seated and the leg at 90° to the thigh, the maximum circumference of the calf was measured.

The anthropometric measurements described above were recorded on data sheets (Appendix 2) and then used to derive the following data:

1. Heath - Carter somatotype (Carter,1980)

The first component (endomorphy) is estimated from the sum of the triceps, subscapular and supra-iliac skinfold thicknesses. The second component (mesomorphy) is assessed on the basis of two bone diameters (humerus and femur) and two circumference measurements (upper arm and calf), the latter two being corrected for the calf and triceps skinfold thicknesses. These two diameters and two corrected circumferences are evaluated relative to body height. The third component (ectomorphy) is assessed strictly on the basis of the height/$\sqrt[3]{\text{weight}}$ ratio. All the above data were analysed on a Heath - Carter somatotype rating form (Appendix 3).

2. Percent body fat (% BF)

In order to assess % body fat for individuals, the body density has to be calculated first. This is done by adding the four skinfolds - biceps, triceps, subscapular and supra-iliac, and then using the equations derived by Durnin and

Womersley (1974). Density is then calculated from the following linear regression equation which estimates density from the logarithm of skinfold thickness :

density = c - m x log skinfold

where 'c' and 'm' vary according to the regression equations depending on age and sex.

The formula used to calculate % body fat is the Siri equation (as cited by Durnin and Womersley,1974) :

$$\% \text{ body fat} = \left(\frac{495}{\text{density}} \right) - 450$$

which is specific for Caucasians.

BLOOD SAMPLING AND ANALYSIS

Two to three hours before the start of the race a blood sample was taken from each subject. Each subject was seated at the time. A total of 10 mℓ of blood was drawn from an antecubital vein using a 21-G needle and vacu-tubes. Red-topped tubes containing only a silicone coating were used. The tubes were stored at 4°C until the blood was spun down in a centrifuge and the serum was pipetted out into Eppendorf tubes.

Within 15 minutes after the completion of the race the second blood sample was taken following exactly the same procedures. Five mℓ were again stored at 4°C in red-topped tubes until the serum was separated. The other 5 mℓ were stored in

purple-topped tubes containing EDTA, as whole blood, at 4°C to be analysed for WBC count.
The haematocrit was determined using capillary tubes.

The blood samples were analysed by the South African Institute of Medical Research (SAIMR).
White blood cell counts were done using the SAIMR Coulter counter according to the standard technique.
Cortisol concentrations were analysed using the Radio-Immuno-Assay (RIA) technique, specifically the solid-phase assay in which the antibody is coated on the tube. ^{125}I was used as a label. Each sample was assayed in the same hormone system and by the same person to reduce inter-assay variation.
Haematocrit was determined using capillary tubes and the haematocrit chart.

PSYCHOLOGICAL QUESTIONNAIRES

All subjects completed two questionnaires about three hours before the start of the race, i.e. soon after arriving at the venue and before preparing to warm-up. The shortened version of the Eysenck Personality Inventory (EPI) (Eysenck,1958) was completed first. The EPI (Appendix 4) gives an indication of extroversion-introversion and neuroticism-stability.
Immediately after completing the EPI the runners were asked to complete the Profile of Mood States (POMS) questionnaire (McNair et al.,1971). The POMS test (Appendix 5) consists of a 65 - item adjective rating scale designed to assess six

mood states, namely "tension", "depression", "anger", "vigour", "fatigue" and "confusion" (McNair et al.,1971). Subjects were instructed to respond in terms of how they had been feeling for the past few hours.

Each adjective is scored from 0 (not at all) to 4 (extremely). Post-race POMS tests were administered within one hour after successfully completing the race. Subjects were asked to respond in terms of how they had been feeling during the race. A Total Mood Disturbance (TMD) score was calculated on both the pre- and post-race POMS. This was done by adding the five factors : "tension", "depression", "anger", "fatigue" and "confusion" and subtracting "vigour" from the sum (McNair et al.,1971).

RATINGS OF PERCEIVED EXERTION

After each subject had completed the psychological questionnaires the procedure for completing the Ratings of Perceived Exertion (RPE) forms was explained.

Each of the 21 runners was given an RPE instruction sheet, a Borg scale and a data sheet (Appendix 6) to be completed every two hours through the race. Three ratings were obtained. The first rating was associated with the sensation of strain in the exercising muscles i.e. the lower limb muscles. The second rating involved the feelings of effort associated with the cardio-pulmonary response, and the third rating required an overall or general evaluation of the

exertion experienced during the two-hour spell (Ekblom and Goldbarg,1971).
In reducing these data the RPE was expressed relative to percent running time rather than to actual running time or time of day because the finishing times varied between 17 hours and 9 min. and 25 hours and 33 min.

STATISTICAL ANALYSIS

The statistical analysis began with the computation of means and standard deviations.
Related "Student's" t-tests (Ferguson,1981) were computed on all the pre-race and post-race data in order to test if there were significant differences.

Analysis of Variance (ANOVA) (Ferguson,1981) was performed on RPE data in order to determine whether there were significant differences between local, central and overall RPE. In all cases the Tukey Test was used as a post hoc test where significant differences were found.
In all cases the 0.05 level of probability was used.

CHAPTER FOUR

RESULTS AND DISCUSSION

The major objective of this study was to identify the effect of running 160 km on certain anthropometric, physiological and psychological parameters. Twenty-one male runners completed the 160 km within the required time, namely 26 hours.

The data are organized and presented in seven sub-sections: subject performance characteristics; anthropometry; physiological parameters : plasma volume and cortisol; psychological parameters : rates of perceived exertion (RPE), Eysenck's Personality Inventory (EPI), Profile of Mood States (POMS).

Descriptive statistics were used to examine the results. Related t-tests were used to assess changes that had occurred during the run.

SUBJECT PERFORMANCE CHARACTERISTICS

The 21 males in this study were not all elite runners. The fastest finishing time was 17 hours and 9 min., at an average speed of 9.33 $km.h^{-1}$. The slowest runner completed the distance in 25 hours and 33 min., at an average speed of 6.26 $km.h^{-1}$. The 21 finishers exhibited a mean running time of 22 hours and 6 min. which is closer to the slowest time than the fastest. The average speed was 7.24 $km.h^{-1}$.

Admittedly this does not take into account the time spent resting.

The fastest ten finishers in the 1992 Comrades 'up run' (86.7km) completed the course in an average time of 5 hours and 50 min. Their average speed was 14.85 $km.h^{-1}$; more than twice the speed of the average runners in the present study. The last person to complete the 86.7 km Comrades marathon within the 11 hour time limit ran at an average speed of 7.89 $km.h^{-1}$. The running times of the 10 fastest Comrades runners, the slowest Comrades runner, the fastest of the 21 Washie runners and the slowest of the Washie runners are shown in Figure 1. The Washie is 1.85 times the distance of the 1992 Comrades marathon. Simple extrapolation of the Comrades times to the Washie times does not yield the actual Washie times.

Figure 2 indicates the running speeds of the fastest and slowest Comrades and Washie runners. The fastest of the Washie runners ran at an average speed closer to that of the slowest Comrades finishers than that of the 10 fastest Comrades runners. This is best illustrated in Figure 3 where the fastest 10 Comrades runners' mean speed is compared to the fastest Washie runner's average speed, and the last Comrades finisher's speed to the last Washie runner's average speed.

As running velocity increases so does oxygen consumption (Lewis et al.,1983). According to Menier and Pugh (1968), and Kagaya (1976) it is more economical to walk at speeds slower than 8 $km.h^{-1}$. Åstrand and Rodahl (1977) also support this.

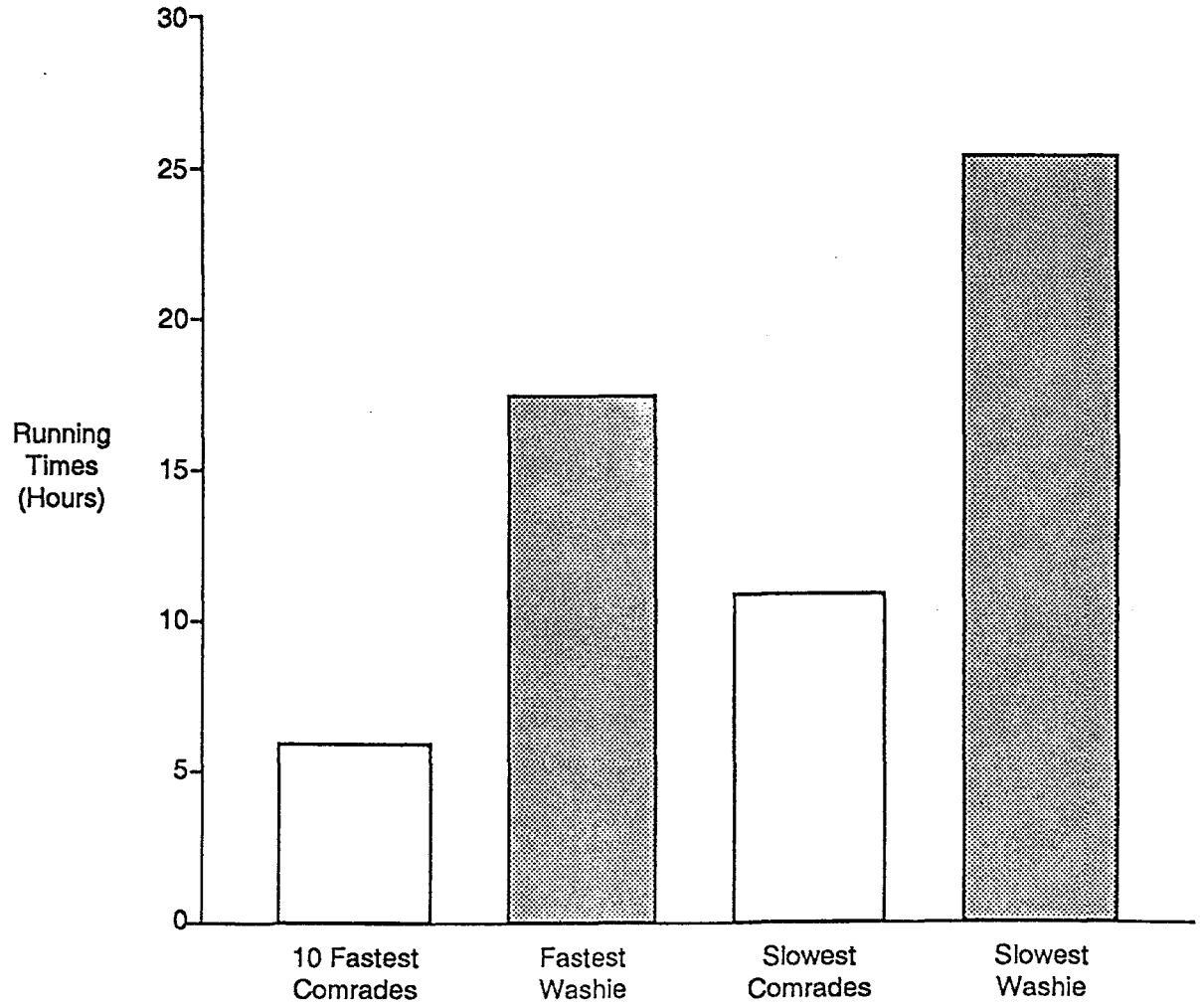


FIGURE 1: *RUNNING TIMES OF THE 10 FASTEST COMRADES RUNNERS, THE LAST COMRADES FINISHER AND THE FASTEST AND SLOWEST WASHIE RUNNERS IN THIS SAMPLE*

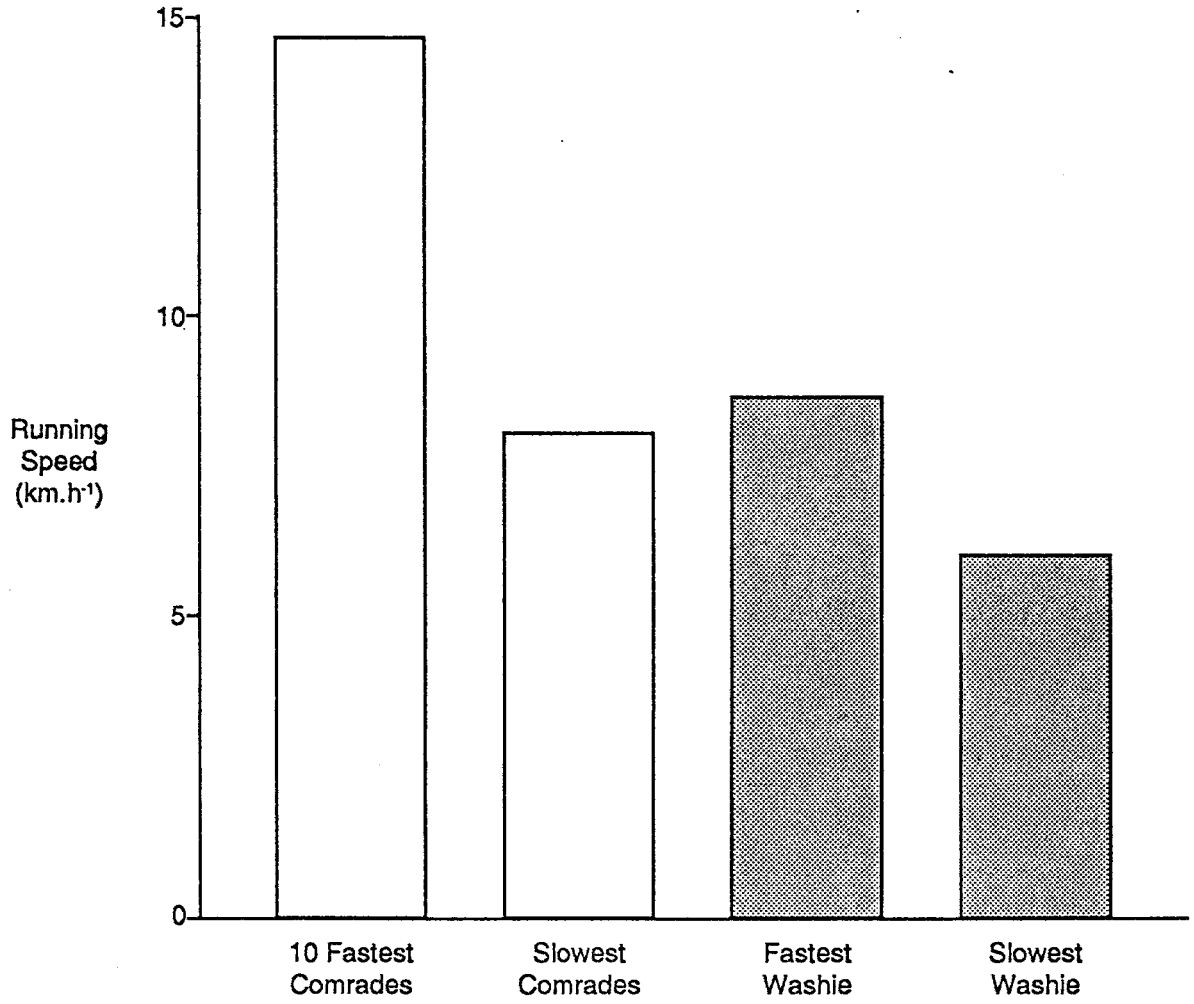


FIGURE 2: *RUNNING SPEEDS OF THE 10 FASTEST COMRADES RUNNERS (MEAN), THE LAST COMRADES FINISHER AND THE FASTEST AND SLOWEST WASHIE RUNNERS IN THIS SAMPLE*

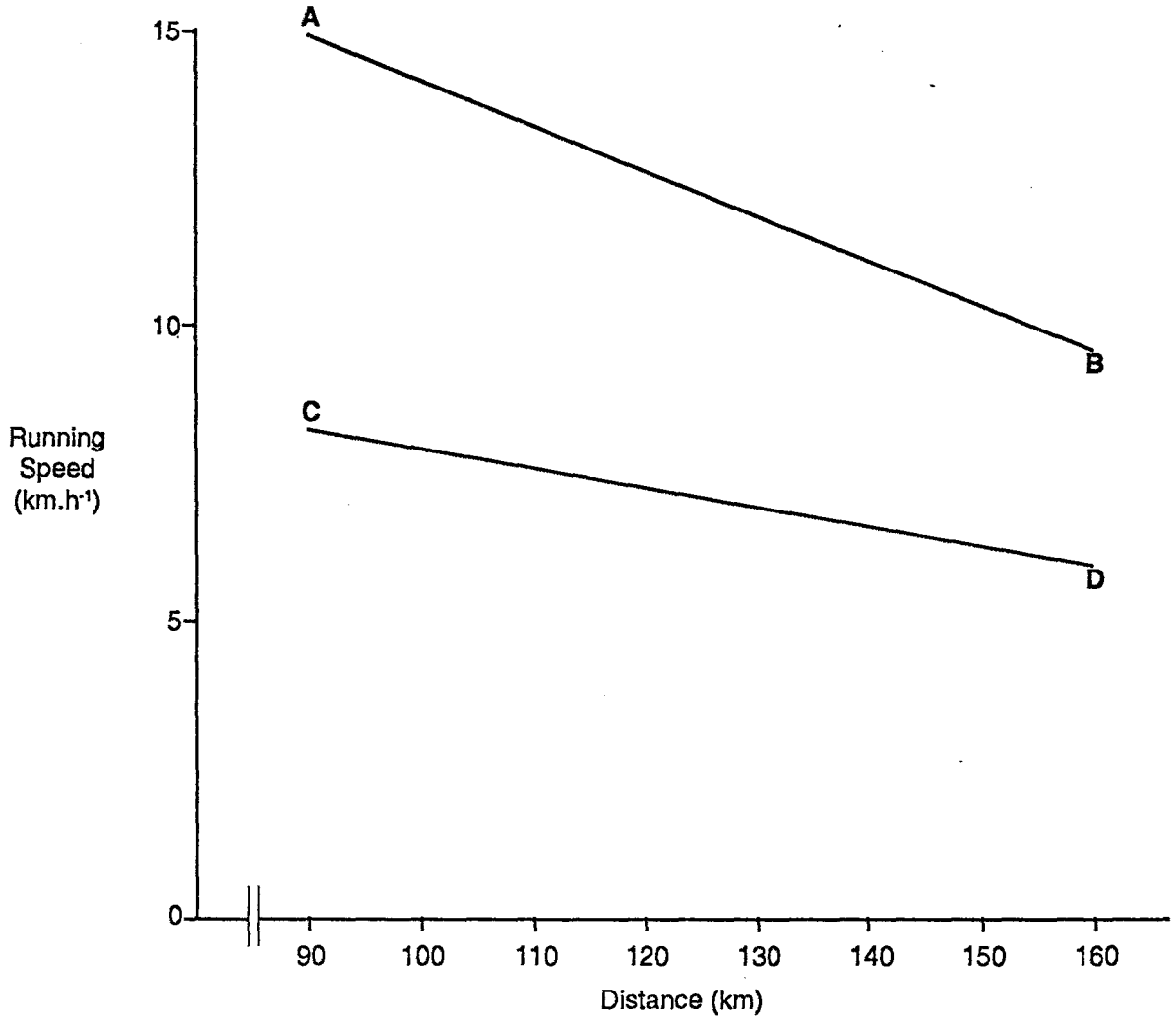


FIGURE 3: *RUNNING SPEEDS OF THE COMRADES FRONT RUNNERS (MEAN) (A), THE FASTEST WASHIE RUNNER (B), THE LAST COMRADES FINISHER (C) AND THE SLOWEST WASHIE RUNNER (D).*

Speeds of this order delineate the "walk-to-run" interface, which varies within a small range from individual to individual. For each individual it is the speed at which it is possible either to walk fast or run slowly.

McArdle et al. (1986) suggested that walking becomes more economical than running at a speed somewhere between 7 and 9 $km.h^{-1}$. In other words, the Washie runners as a group were running close to their 'walk-to-run' interface i.e. it may have been more economical if they had walked some of the way. However, averaging in this way is simply an estimate as it incorporates rest pauses and a variety of locomotor strategies adopted through the race. This raises the question as to what these differences in running speed mean in terms of energy expenditure and oxygen consumption. Since no direct measurements were taken, predictions were made on the basis of assumptions noted below.

Firstly, 1 MET may be assumed to approximate 1.6 $km.h^{-1}$ (Pate et al.,1991) when running on horizontal terrain. Since the Washie is not run only on horizontal terrain, this approximation underestimates the amount of energy required to run at a speed of 1.6 $km.h^{-1}$. Alternatively, it is widely accepted as a workable generalisation that 1 MET approximates 3.5 ml $O_2.kg^{-1}.min^{-1}$ (Pate et al.,1991). This is an average oxygen consumption based on an R value of 1 (McArdle et al.,1986).

Using these assumptions the energy expenditure of level running can be quantifiably predicted. The fastest Washie runner thus ran at least at 5.83 MET, which is equivalent to

20.41 ml $O_2.kg^{-1}.min^{-1}$. If one were to conservatively assume a maximum oxygen consumption ($\dot{V}O_2max$) of 50 $ml.kg^{-1}.min^{-1}$, then this would approximate to 40.8% of $\dot{V}O_2max$. The 10 fastest Comrades marathon runners would have been working at 9.28 MET, which is 64.8% of a $\dot{V}O_2max$ of 50 $ml.kg^{-1}.min^{-1}$. The slowest Comrades finisher would have worked at 4.93 MET, which approximates 34.5% $\dot{V}O_2max$. Thus broadly speaking the running intensity of the fastest of the 21 Washie runners was closer to that of the slowest Comrades finisher than that of the 10 fastest Comrades runners. The slowest of the Washie runners worked at 3.91 MET (minimal estimate), or about 27.4% $\dot{V}O_2max$. The mean of all the Washie runners was 4.52 MET, about 31.68% $\dot{V}O_2max$. Since the anaerobic threshold of trained runners is about 75% $\dot{V}O_2max$ and for untrained male runners about 66% $\dot{V}O_2max$ (de Mello et al.,1985) it seems that running at an average of 32% $\dot{V}O_2max$ will not elicit a significant production of lactic acid.

In 1962 Bink developed an equation to demonstrate the relationship between general physical labour and acceptable energy to work for a certain time ($kcal.min^{-1}$) based on an average aerobic demand of 5.2 $kcal.min^{-1}$ for an 8h shift:

$$E = \frac{(\log 5700 - \log t)}{3.1} \times \text{aerobic capacity}$$

where E is the acceptable energy expenditure ($kcal.min^{-1}$) in order to ensure a working energy cost of 5.2 $kcal.min^{-1}$, and t is the working time in minutes. If a worker had an aerobic capacity of 15.1 $kcal.min^{-1}$ and worked for 8h, the acceptable energy expenditure is 34% of aerobic capacity.

Thus running 160 km in 25 hours and 33 min., as the slowest Washie runner did, would be like working for more than 3 eight-hour shifts without a break in a light factory job. Thus it seems that it is the TIME spent running rather than the intensity of running that is stressful.

Nine of the 21 finishers were "novices" (i.e. they had not run the Washie 160 km race previously). Twelve were "experienced" (i.e. had run the Washie at least once previously). Subjects were asked to predict their finishing times before the race. A related t-test was used to assess if there were any significant differences between the predicted and actual running times of either the novices or the experienced runners ($p<0.05$). Results showed that both groups were equally good at predicting their times.

The mean age of the 21 Washie runners was 38.76 (± 6.15) yr. According to Osler and Dodd (1979) most 160 km runners have had extensive experience at running shorter races and tend, therefore, to be older than the average marathon runner. Keul et al. (1981) investigated 7 male runners in a 100 km run in Switzerland and found their mean age to be 33.3 (± 3.5) yr. Noakes and Carter (1976) studied 7 males who completed 160 km and found their mean age to be 33.14 (± 4.96) yr. The average age of all the 1992 Comrades finishers was 35 years. In 1991 it had also been 35 years, and in 1990 it had been 34 years (Comrades Association South Africa,1992).

Intra-individual variation in certain factors was evident during the Washie run.

1. Body temperature : Runners wore whatever they wanted whenever they felt it necessary. Since all runners ran through the night, they all experienced the drop in ambient temperature. Some runners spent the entire day thereafter running as well. Body temperature will have varied depending on what the ambient temperature was at the time, what they were wearing and also on the pace at which they were running.

2. Stride length : It is possible to change the velocity of running in 3 ways :

(a) by changing the number of steps taken per unit time,

(b) by changing the stride length and

(c) by changing both (a) and (c) (McArdle et al.,1986).

Running fast i.e. high intensity short duration running, is usually achieved by increasing the stride length. It is possible to increase stride length in 3 ways :

(i) by extending the leg further

(ii) by increasing the angles of the thigh in the sagittal plane

(iii) by using more force (Hogberg,1952).

However (i) is uneconomical and (ii) increases pelvic oscillation and is therefore not beneficial. Using more force (iii) is the most efficient way of increasing stride length. There is, however, an optimal stride length for each individual which minimizes energy cost. According to McArdle et al. (1986) over-striding is more "costly" than under-

striding, at any given velocity. Thus it is not surprising that many long distance runners have developed a "shuffle". It was also evident that many runners, particularly the slower runners, began to "shuffle" towards the end of the run.

3. Body mass : Mass carried may affect foot-strike patterns which in turn may affect stride length by changing the below-knee excursions. All runners experienced a decrease in body mass during the race. Whether or not this itself mediated a change in running style is unknown.

4. Enzyme systems : These may be affected by long-duration running, since the body does not get a chance to replenish energy stores and substrates for the production of energy, such as it seems to do during sleep (Daniels,1985).

Inter-individual variation also existed. For example, there were differences in stride length, body mass, muscle fibre composition, anaerobic threshold, lactate tolerance, $\dot{V}O_2max$, food and fluid intake, state of training (McArdle et al.,1986). All these factors may have influenced the running speed and therefore the time taken to run 160 km.

ANTHROPOMETRY

Endurance running ability is determined by genetic factors as well as the effects of endurance training (Tanaka and Matsuura,1982). Clearly certain anthropometric attributes will exert important influences on performance.

Table I shows the relevant anthropometric data of the 21 male finishers. Their mean stature was 177.6 (± 6.4) cm before the race. Pollock et al. (1977) measured elite marathon runners. Their mean stature was 177.0 (± 5.0) cm which compares favourably with the Washie runners. Tanaka and Matsuura's (1982) athletes were shorter, with a mean of 170.1 (± 4.6) cm, but they were Japanese. The 1960 Olympic marathon runners whom Tanner (1964) measured had a mean stature of 170.5 cm. Costill (1972) surveyed all winners of the Boston marathon between 1897 and 1965 and found their average height to be 170.0 cm ; less than the Washie runners in this sample. From a plot of mean stature against running distance, (Figure 4) it is apparent that there is a tendency for height to decrease as running distance increases between 1500m and the standard marathon. However, when the mean stature of the Washie runners is also plotted it seems that there may be a 'U' trend; stature again increases for distances longer than 42 km. However, the 1500m and marathon runners were elite runners, while the Washie runners were not.

Table I: Selected anthropometric data of the Washie runners analysed.

Parameter	$\bar{x}$	s.d.
Stature (cm) :before	177.6	6.4
: after	176.6	6.3
Change in stature (cm)	1.03	0.4
Mass (kg) : before	77.4	11.4
: after	73.9	10.8
Change in mass (kg)	3.5	1.4
(%)	4.5	-
Density ($g.cc^{-1}$)	1.057	0.008
% Body fat (skinfold)	18.4	3.7
Fat weight (kg) : before	14.63	4.80
Lean body weight (kg) :before	62.77	6.64
Reciprocal Ponderal Index (RPI) $\frac{\text{Stature (cm)}}{\sqrt[3]{\text{mass (kg)}}}$	41.85	2.06
Body Mass Index (BMI) ($kg.m^{-2}$)	24.55	3.40
Body Surface Area (BSA) (m^2)	1.94	0.14

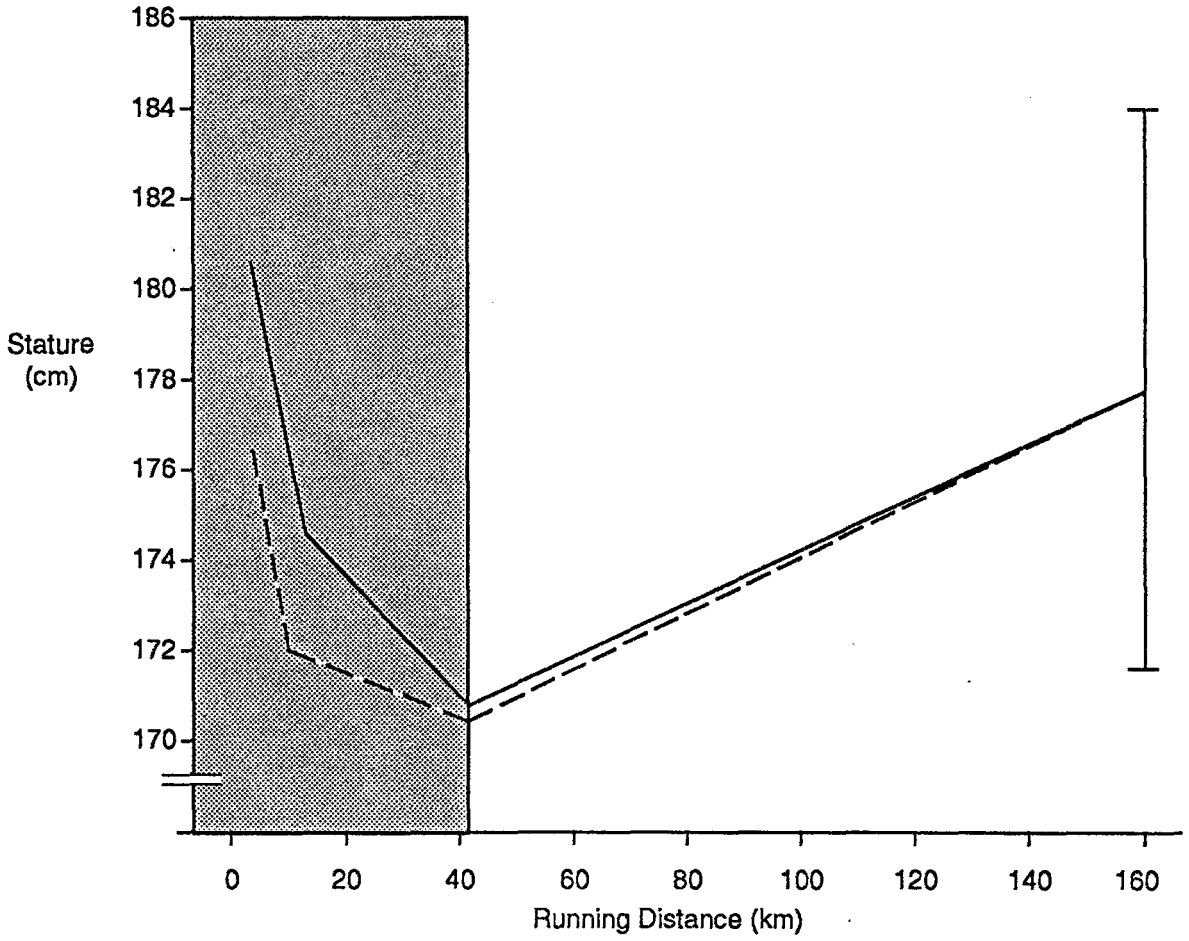


FIGURE 4: *MEAN STATURE OF OLYMPIC ATHLETES (LARSON, 1974) AND THE WASHIE RUNNERS.*

KEY: — — 1960 OLYMPIC ATHLETES: 1500m, 10000m and MARATHON RUNNERS

—— 1964 OLYMPIC ATHLETES: 1500m, 10000m and MARATHON RUNNERS

ELITE ATHLETES

Size-relative speed is that fraction of stature covered overground per second (Grieve and Gear,1966). The following equation is used to calculate relative speed if actual velocity is known :

$$\text{relative speed} = \frac{\text{velocity } (m.s^{-1})}{\text{stature } (m)}$$

Since the mean absolute speed of the 21 Washie runners was 2.01 $m.s^{-1}$ and their mean stature was 1.78 cm, their relative speed was calculated to be 1.13 statures per second. At any given speed shorter runners will take more steps than taller runners. Thus by making absolute speed relative to stature, the variability in cadence between subjects is minimized.
Stature of the subjects of the present study was again measured after the race. The mean change (in every case a decrease) was 1.03 (± 0.4) cm. A related t-test revealed significance at the $p<0.05$ level. The change in stature was probably as a result of repetitive compressive forces on the subjects' intervertebral discs.
Even in daily life the lumbar spine has large loads impressed on it. Flexion, extension and lateral bending all cause changes in pressure within the intervertebral discs (Ayoub and Mital,1989). According to Miller and associates (1986) the compressive forces on the lumbar spine during daily life can sometimes be up to 11 times body weight. When running, the heel-strike force is about twice body weight. When the weight is transferred onto the forefoot the force is 3 to 4 times body weight (Dickinson et al.,1985). Each heel strike

produces shock waves that travel up the leg to the rest of the axial skeleton. These vibrations gradually become less as they spread through the body. Helminen and associates (1987) calculated that jogging 32 km per week sends in excess of 1.7 million extra shock waves per year through the body. Fluid is forced out of the intervertebral disc whenever the compressive forces exceed the osmotic pressure inside the intervertebral disc (Troup et al.,1985).

The physiological advantages to long distance runners of having lower body mass are the result of the correlation which exists between body mass and energy expenditure at any given speed (Wyndham et al.,1971). The relationship between oxygen consumption (aerobic metabolism) and running speed is linear as long as the speed is less than about 6 $m.s^{-1}$ (Åstrand and Rodahl,1977). As the speed increases the relationship becomes exponential because energy supply is then supplemented by anaerobic means. This linear relationship between speed and energy cost during aerobic running suggests that the energy required to cover a certain distance is in fact independent of speed. However, it varies from subject to subject - probably because of different morphologies.

The human body consists of different kinds of tissues that have different densities. The density of fat is less than the density of lean tissue. Therefore for a given mass an obese person will have a greater volume than a non-obese person. For young and middle-aged adults fat has a density of

0.90 $g.cc^{-1}$ and lean tissue (non-fat) has a density of 1.10 $g.cc^{-1}$ (McArdle et al.,1986). It is therefore not sufficient to consider mass alone as an indicator of potential in endurance sport.

Figure 5 illustrates the tendency for mass to decrease as running distance increases from 1500m to the standard marathon. It should again be noted that these are means of elite (Olympic) runners. When the Washie runners' mean mass is plotted it seems to indicate a possible 'U' relationship, as was the case for stature (Figure 4). Figure 5 also shows a large standard deviation for the mass of the Washie runners. Thus some Washie runners had a mass similar to that of the 1500m runners.

Elite marathon runners tested by Pollock et al. (1977) were found to have a mean mass of 63.1 (± 4.8) kg. Tanaka and Matsuura (1982) found an even lower mean mass (57.3 kg) but these were young Japanese athletes. Tanner (1964) found that 1960 Olympic marathon athletes had a mean mass of 61.6 kg. The Washie runners were considerably heavier, with a mean of 77.4 (± 11.4) kg before the race. The 100 km runners measured by Keul et al. (1981) had a mean mass of 72.7 (± 4.9) kg. Although this is 4.7 kg lighter than the mean of the Washie runners, it too is considerably more than that of the elite marathon runners. Noakes and Carter's (1976) 160 km runners had a mean mass of 74.4 kg.

The mean density of the 21 Washie runners as determined by the anthropometric method (Durnin and Womersley,1974) was 1.057 $g.cc^{-1}$. This is closer to the density of lean body

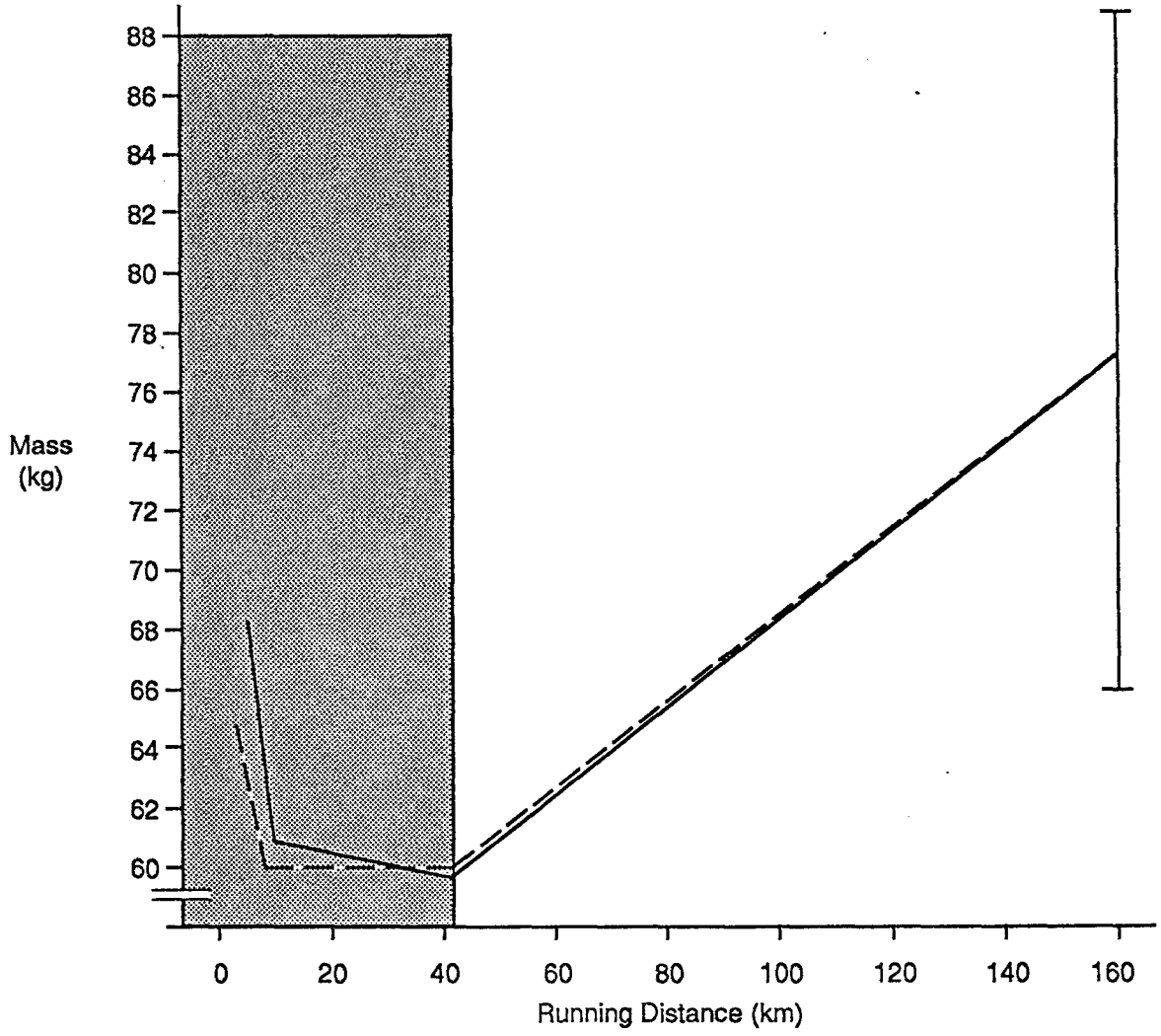


FIGURE 5: *MEAN MASS OF OLYMPIC ATHLETES (LARSON, 1974) AND THE WASHIE RUNNERS.*

KEY: — — *1960 OLYMPIC ATHLETES: 1500m, 10000m and MARATHON RUNNERS*

—— *1964 OLYMPIC ATHLETES: 1500m, 10000m and MARATHON RUNNERS*

▒ *ELITE ATHLETES*

tissue i.e. non-fat, than fat. In fact the mean fat weight and lean body weight were calculated to be 14.63 (± 4.8) kg and 62.77 (± 6.64) kg respectively. Lean body mass is an important factor because it is the active muscle tissue (Lewis et al.,1983).

The Body Mass Index (BMI) is used to assess weight relative to height. It is expressed as body mass (kg) / Height (m^2). The BMI of athletes taking part in the 1928, 1960, 1964, 1968 and 1972 Olympic games was calculated (Carter,1984a). Table II illustrates the different BMI values. As can be seen from Table II the BMI is greatest for sprinters and tends to decrease as the running distance increases. The 5000m and 10000m runners had the lowest BMI. However, the BMI does not seem to decrease further as distance increases - as may have been expected, but seems to increase again. The Washie runners' mean BMI was calculated to be 24.55 (± 3.4) $kg.m^{-2}$ which is closer to the BMI of the general population of the British, Americans, French and Germans than to the BMI of the Olympic runners, as illustrated below (Pheasant,1990) :

Nationality	Body Mass Index (BMI) ($kg.m^{-2}$)
British	24.47
American (USA)	25.45
French	24.88
German	24.04

Table II: Mean Body mass index (mass (kg) ÷ stature (m^2)) of Olympic athletes and the means for competitors in each of the distances (Carter,1984a).

Distance	1928	1960	1964	1968	1972	$\bar{x}$
100m,200m	21.71	22.40	22.84	22.21	22.52	22.34
800m,1500m	21.94	21.13	20.86	20.70	21.40	21.21
5000m,10000m	21.46	20.51	21.03	20.10	20.64	20.55
marathon	21.59	21.27	20.79	19.86	20.83	20.87

The Reciprocal Ponderal Index (RPI) gives an indication of linearity (Larson,1974). Weight is a three-dimensional expansion of the body. The RPI of the 1928, 1960, 1964, 1968 and 1972 Olympic Games athletes taking part in the various running events was calculated by Carter (1984a). Table III illustrates the RPI values. From Table III it becomes evident that Olympic sprinters have a lower mean RPI than the other Olympic runners. The 800m, 1500m,5000m and 10000m RPI values were similar but the marathon runners' mean RPI again decreased. The lower the RPI the more ponderous the athlete. Thus the Washie runners, who had a mean RPI of 41.85 (± 2.06) were even more ponderous than the Olympic sprinters, let alone the longer distance runners.

Winter (1978) differentiated between internal and external work. Internal work is the mechanical work done to make the body segments move; in this case to run. External work is done to overcome air and ground friction. As the gradient increases so does external work. Also, depending on running speed, it may take anything from 3.6 to 9% of total energy to overcome air resistance (McArdle et al.,1986). The effect that air resistance has on the energy cost of running depends on three factors :

(1) the density of air : this did not change much during the run because the Washie began and finished at sea level.
(2) wind velocity : it was not a particularly windy day or night. Runners experienced similar circumstances.
(3) body surface area (BSA) : changes from individual to

Table III: Reciprocal ponderal indices (RPI) of Olympic athletes in the various distances, and the means for each of the distances (Carter,1984a).

Distance	1928	1960	1964	1968	1972	$\bar{x}$
100m,200m	43.01	43.05	42.54	42.88	42.75	42.85
800m,1500m	43.01	44.03	44.82	44.13	43.59	43.92
5000m,10000m	43.27	44.80	43.78	43.96	43.72	43.91
marathon	42.52	42.94	43.31	43.94	43.38	43.22

individual. BSA is an effective measure of body size which incorporates both stature and mass. Body surface area (BSA) was calculated according to the formula proposed by DuBois and DuBois in 1916 :

$$BSA = \frac{71.84 \times \left[mass^{0.425} \text{ (kg)} \times height^{0.725} \text{ (m)} \right]}{10\ 000}$$

(Martin et al.,1984). The mean BSA was 1.94 (± 0.14) m^2, the largest was 2.23 m^2 and the smallest 1.53 m^2. Heat is lost from the body by 4 mechanisms :

(1) radiation : heat rays are radiated in all directions
(2) conduction : heat is passed on by conduction from the surface area to other objects, including air molecules
(3) convection : after heat has been conducted to the air molecules, currents take away the air molecules
(4) evaporation : for each gram of water that evaporates from the skin 0.58 calories of heat are lost.

Clothing traps air in contact with the skin. This reduces heat loss by conduction and convection. However, when clothing becomes wet the amount of heat loss increases because of the higher conductivity of water relative to air (Guyton,1981).

When running, the body is being displaced horizontally and thus the removal of heat via convection air currents increases. The amount of skin exposed to the air is a factor affecting convection. The greater the amount of skin exposed i.e. the greater the body surface area relative to volume, the greater the ability to lose heat by convection and in

fact evaporation. Obviously BSA is not the only factor affecting heat loss. Other factors include sweat rates of individuals, speed of running and the extent of vasodilation of peripheral blood vessels.

When the percentage of body fat was estimated from density (Durnin and Womersley,1974) the 21 Washie runners were found to have a mean of 18.4% fat. Tanaka and Matsuura's (1982) athletes had a mean % body fat of 10.6 (± 1.1). Costill et al. (1970) measured 114 runners that took part in the 1968 USA Olympic marathon trial race. These athletes had a mean of 7.5%. This is less than half that measured in the sample of Washie runners. According to Pollock et al. (1977) elite marathon runners have been found to have between 6 and 8% body fat. Thus the Washie runners in this sample had considerably more body fat than elite marathon runners.
The mean body mass of the Washie runners was again measured after the 160 km race. The mean mass then was 73.9 (± 10.8) kg. On average they had lost 3.5 (± 1.4) kg. This was a significant decrease in mass ($p<0.05$).

By definition somatotypes do not change with time or training since they are not based on size (Tanner,1964). The mean somatotype of the Washie runners was 3.1;4.8;2.1. This indicates that this sample of Washie runners was more endo-mesomorphic than meso-ectomorphic. The fairly high endomorphy rating is substantiated by the fairly high % body fat. The mesomorphy rating is consistent with the RPI of 41.85,

indicating above average body stoutness. de Garay et al. (1974) measured 20 of the top marathon runners in the 1968 Olympic marathon. Here the mean somatotype was 1.4;4.3;3.5. Thus the Washie runners studied were more endomorphic, slightly more mesomorphic and less ectomorphic . Carter (1984b) calculated the somatotypes of Olympic athletes taking part in the various running events. Table IV gives the results. There seems to be a trend : there is a decrease in mesomorphy and an increase in ectomorphy from the sprints to 10000m with a slight drop in endomorphic rating. There is slight reversal in marathon running and walking. Whether this reversal increases as distance increases beyond the marathon seems unclear. The Washie runners displayed greater endomorphic and mesomorphic ratings but lower ectomorphic ratings than the marathon runners and walkers. According to Withers et al. (1986) the main reason for somatotyping is to project a 'Gestalt" or overall impression of body type. This is best done when the somatotypes are plotted onto somatocharts - which provide pictorial clarity. However, it is a two-dimensional representation of three-dimensional phenomena (Bailey et al.,1982;Withers et al.,1986). Figure 6 is a somatochart illustrating the somatotypes of the Washie runners and the 1960 and 1968 Olympic marathon runners. Figure 7 is a somatochart indicating the somatotypes of the athletes of the 1948 to 1976 Olympic games as calculated by Carter (1984b). Somatotype dispersion distances (SDD) between the mean somatoplot of the Washie runners and the Olympic athletes participating in the various events were calculated

Table IV: Mean somatotypes of Olympic athletes participating in the various running distances, and the number of athletes in each sample (n). The athletes are from the 1948 to 1976 Olympic Games (Carter,1984b).

EVENT	SOMATOTYPE			n
	Endo-	Meso-	Ecto-	
100m,200m,110m hurdles	1.7	5.2	2.8	107
400m,400m hurdles	1.5	4.6	3.4	64
800m,1500m	1.5	4.3	3.6	56
3000m,5000m,10000m	1.4	4.2	3.7	58
marathon	1.4	4.4	3.4	32
20 km and 50 km walk	1.6	4.7	3.4	25

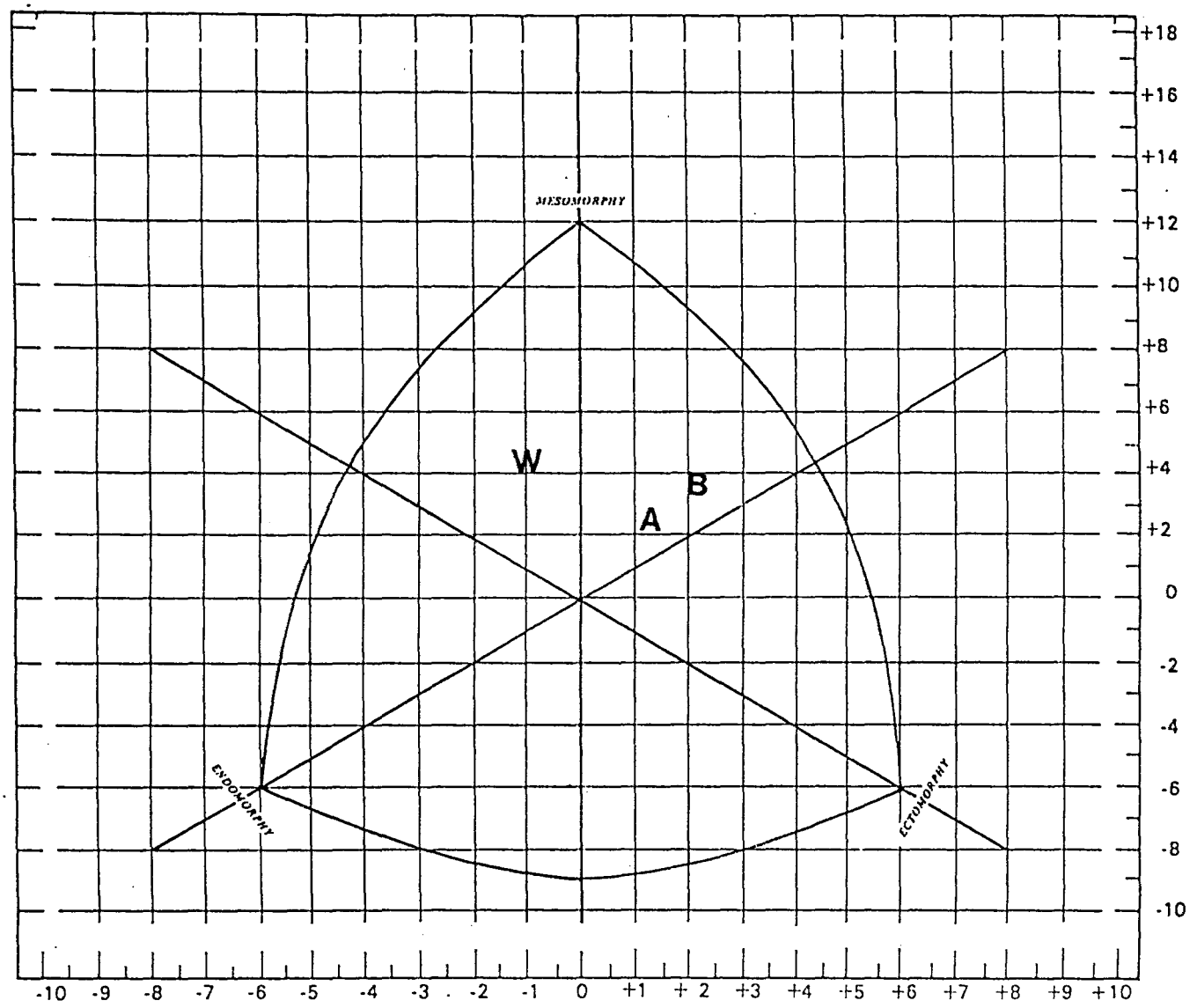


FIGURE 6: *A SOMATOCHART ILLUSTRATING THE SOMATOTYPES OF WASHIE RUNNERS AND OLYMPIC MARATHON RUNNERS.*

KEY: *W = WASHIE RUNNERS*
A = 1960 OLYMPIC MARATHON RUNNERS
B = 1968 OLYMPIC MARATHON RUNNERS

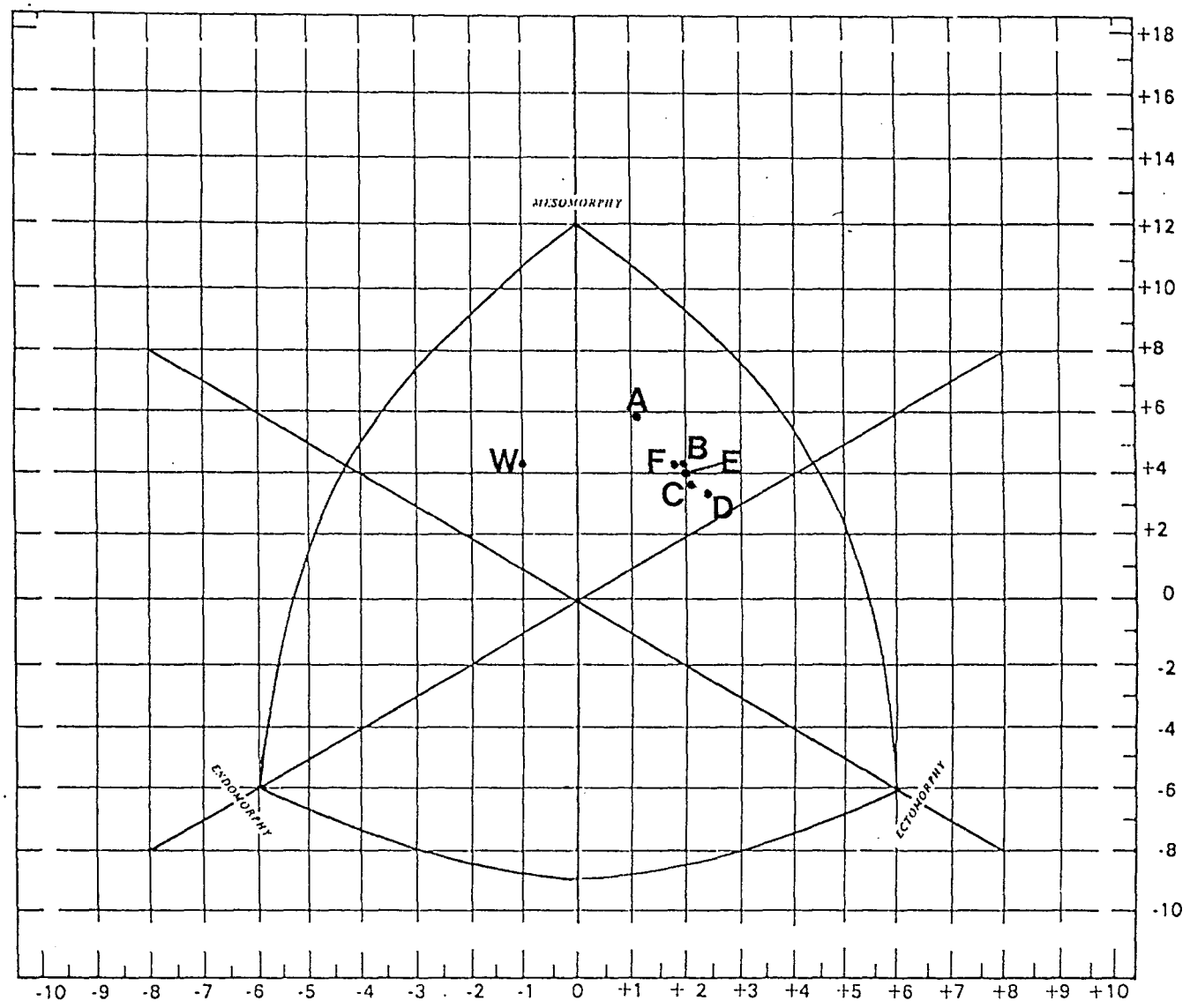


FIGURE 7: *A SOMATOCHART OF THE MEAN SOMATOPLOTS OF THE OLYMPIC ATHLETES WHO PARTICIPATED IN THE 1948 TO 1976 OLYMPIC GAMES (CARTER, 1984B) AND OF THE WASHIE RUNNERS IN THIS SAMPLE.*

KEY:
- *A = 100m, 200m, 110m hurdles*
- *B = 400m, 400m hurdles*
- *C = 800m, 1500m*
- *D = 3000m, 5000m, 10000m*
- *E = Marathon*
- *F = 20km & 50km walk*
- *W = Washie Runners*

according to the following equation :

$$SDD = \sqrt{3\,(x_1 - x_2)^2 + (y_1 - y_2)^2}$$

where x_1 and x_2 are the X co-ordinates and y_1 and y_2 the Y co-ordinates of the somatoplot (Carter,1990). The results were then tabulated (Table V). The somatotype dispersion mean (SDM) was the average distance of the 6 running events from the mean Washie runners' somatoplot and was calculated to be 4.92. From Table V can be seen that the Washie runners' somatotype has more similarity with the somatotype of the sprinters than with the marathon runners. This is in accordance with the RPI values that were obtained.

Table V : Somatotype dispersion distances (SDD) between the Olympic athletes as calculated by Carter (1984b) and the Washie runners.

Event	SDD
100m,200m,110m hurdles	3.31
400m,400m hurdles	5.02
800m,1500m	5.29
3000m,5000m,10000m	5.82
marathon	5.21
20 km and 50 km walk	4.85
Washie runners	4.92

PHYSIOLOGICAL PARAMETERS

Haematocrit and serum cortisol concentration were measured before and after the race. A white blood cell count (WBC) was made from blood drawn after the race.

PLASMA VOLUME CHANGES

The plasma volume of the Washie runners studied was greater after the race than before. The mean plasma volume before the race was 2.92 (± 0.31) ℓ and after the race 3.00 (± 0.28) ℓ. There was a significant increase in plasma volume as measured after the race ($p<0.05$). This phenomenon of increased plasma volume is known as 'plasma volume expansion' (Ritschel,1986). There seem to be different plasma volume responses depending on the intensity and duration of the exercise. The 160 km runners here studied ran at an estimated average intensity of about 32% $\dot{V}O_2max$, which seems to allow the haemoconcentration that may have developed during the early stages of the run to correct itself. Fluid is therefore retrieved from the interstitial space to make up any deficit in plasma volume. This 'retrieval' of fluid probably occurs because osmotic shifts are established : proteins returning from the tissue back to the plasma will create a colloid osmotic pressure in the blood that will 'attract' fluid from the interstitial space (Ritschel,1986). There seems to be some controversy about how these proteins actually move back to the vasculature during prolonged exercise (Myhre et al.,1985).

Sleep deprivation has been reported to increase plasma volume. However, it also seems to increase the peripheral distribution of blood so the increased plasma volume does not create massive changes in blood pressure (Shephard,1984).

CORTISOL

Pre- and post-race serum cortisol concentrations measured in the present study are illustrated in Figure 8. The 21 male Washie runners in this sample had a mean pre-race value of 385.1 (± 146.5) nmol.ℓ^{-1} i.e. less than the concentration at which cortisol binding globulin (CBG) is saturated. These pre-race samples were taken between 15h00 and 18h00 which is more or less the time of day when normal resting cortisol concentration is at its lowest. Haynes and Murad (1975) found that the circadian rhythm for cortisol is at its lowest concentration at about 16h00. This rhythm is, however, not continuous but intermittent. It has been found that major fluctuations occur especially during sleep (Hellman et al.,1970).

The Washie runners' post-race mean was 1001.3 (± 298.5) nmol.ℓ^{-1}. This post-race level is 2.6 times the pre-race concentration and almost twice the amount at which CBG binding sites are saturated. Thus about 449 nmol.ℓ^{-1} was free active cortisol in the plasma. This post-race increase was significant ($p<0.05$). The post-race blood samples were taken within 10 minutes of each runner's completion of the race i.e. between 11h30 and 20h45.

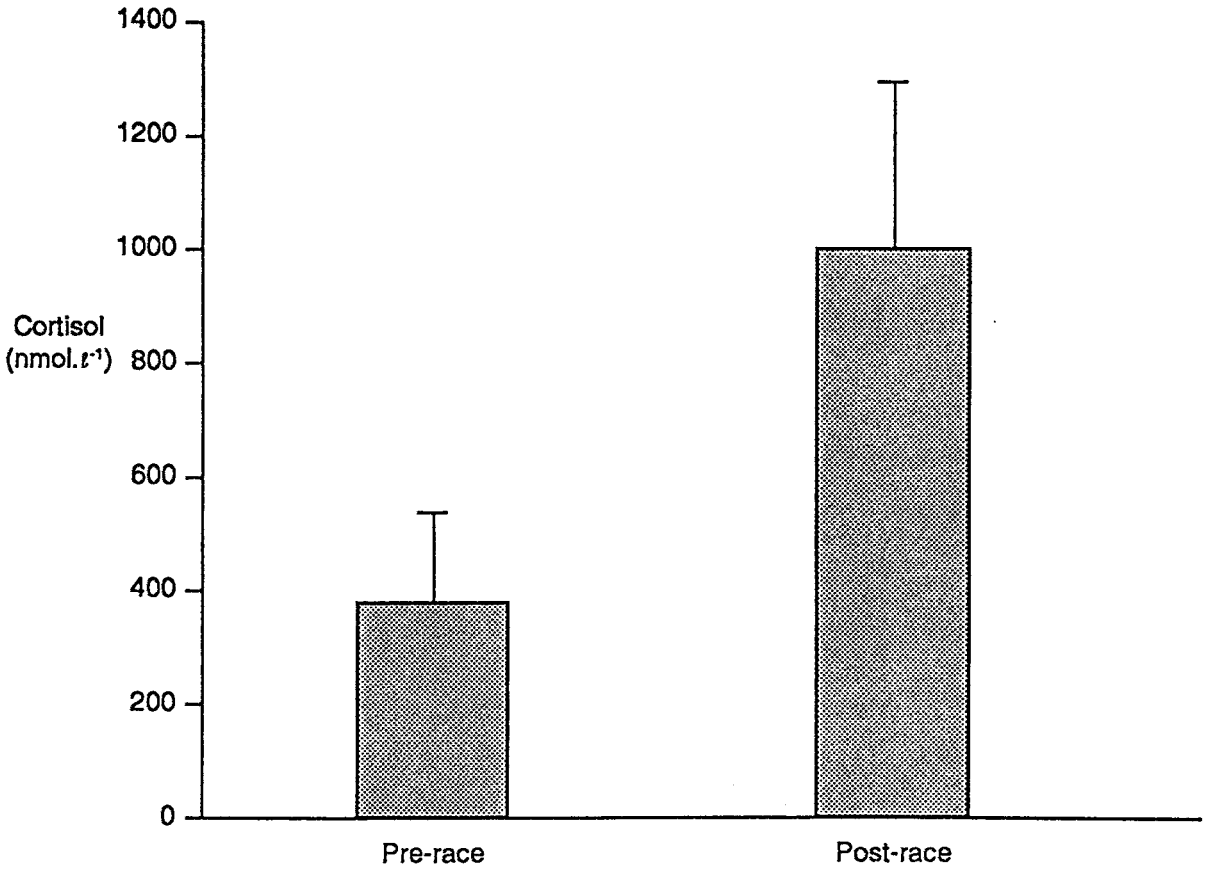


FIGURE 8: *MEAN SERUM CORTISOL CONCENTRATIONS OF THE 21 MALE WASHIE RUNNERS BEFORE AND AFTER THE 160km RACE*

von Glutz et al. (1978) and Keul et al. (1981) measured pre- and post-race cortisol concentrations of 7 runners taking part in a 100 km race. The mean cortisol concentration before the race was 264.5 (SEM=160) nmol.ℓ^{-1} and after the race 783.7 nmol.ℓ^{-1} (SEM=187.6) : a 2.96-fold increase. This finding is in agreement with other studies concerning long distance exercise (Hartley et al.,1972;Maron et al.,1975).

Cortisol promotes gluconeogenesis in the liver and peripheral tissues. In the peripheral tissues cortisol mobilizes amino acids which are then transported to the liver where they are the substrates for the production of glucose. In the liver, cortisol also causes increased synthesis of some of the enzymes involved in gluconeogenesis from amino acids. But the synthesis of these enzymes is not an acute response - it requires a few hours to become effectual. The acute effect of cortisol in the liver involves the hepatic mitochondria: cortisol affects the first reaction of forming glucose from pyruvate, whereby carboxylate pyruvate is changed to oxaloacetate. The importance of gluconeogenesis to the Washie runners becomes evident when one considers the duration of the race and the type of substrate being used for energy. Initially glucose is used, then more and more triglycerides are catabolized. But there has to be a certain amount of glucose in the plasma because the brain can only metabolize glucose. Therefore gluconeogensis will produce glucose when stores of glycogen have been depleted, and severe hypoglycaemia can be averted.

The subjects of this study showed a mean WBC count of 18.4 x $10^9.\ell^{-1}$ (± 3.9) after the race. Since the normal resting WBC count is anything between 4 to 11 x $10^9.\ell^{-1}$, and since most runners showed plasma volume expansion, this seems to indicate leucocytosis. Unfortunately no differentiated WBC count tests were made, only total WBC, so it was not possible to determine which type of leucocyte dominated. However, the leucocytosis seems to indicate that running 160 km is indeed a stressor that warrants such a response.

It is also important to remember that total WBC cycles over a 24h period : it seems to be highest at night and lowest during the day (Scales et al.,1988).

It seems that leucocytosis is inversely related to the state of training i.e. there is an increased leucocytosis in less trained athletes (McCarthy and Dale,1988). Unfortunately there was no way of assessing the Washie runners' level of training.

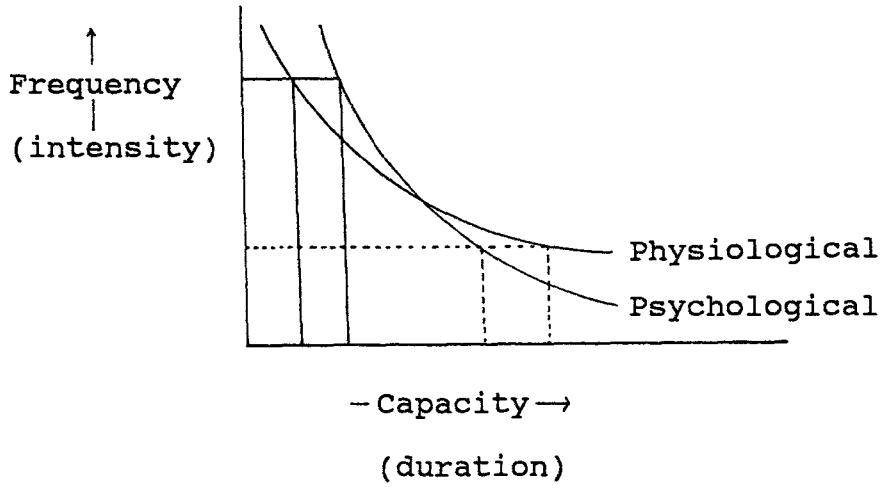
Cortisol can prevent inflammation, as discussed previously. The shock waves created during heel strike are transmitted through the body and affect the muscles, bones, joints and cartilage (Helminen et al.,1987). Baetzner (1920) explained that strenuous and repetitive sports like ultra endurance running, causes 'Sportschaden' - overstrain injuries. Repeated heel strikes may result in local tissue damage which may result in an inflammatory response (Lijnen et al.,1988). High concentrations of cortisol in the plasma may therefore reduce the inflammatory response and the discomfort

associated with it.

Cortisol is involved in maintaining normal glucose concentrations in the blood by gluconeogenesis. Thus it counteracts hypoglycaemia and possible confusion resulting from it.

PSYCHOLOGICAL PARAMETERS

In industry, where people work manually, a relationship between physiological and psychological fatigue criteria has been developed. This is best illustrated graphically :



(After Ayoub and Mital,1989).

In manual materials handling, when a task is repeated often i.e. at a high frequency, the main limitation to continuing the task (capacity) at that intensity for an extended period of time, is physiological fatigue. During low frequency tasks the duration for continuing the task (capacity) increases,

the duration for continuing the task (capacity) increases, but eventually the limitation to continuing for an even longer period, is psychological factors rather than physiological ones.

If this relationship was to be adapted to running, 'frequency' could be interpreted as running speed, and 'capacity' as the ability to continue running at that pace. Then according to the above-mentioned relationship, it becomes evident that the greater the speed the lower the capacity i.e duration, and vice versa. Also, at high running speed i.e. high intensity, physiological fatigue would limit performance before psychological criteria. However, when running speed/intensity was 'low', psychological criteria could account for the inability to continue at that intensity, before physiological criteria. Applying this principle to the present study, since running 160 km took an average of about 22 hours for the Washie runners in this sample, it was low intensity - long duration activity. Thus according to the relationship outlined by Ayoub and Mital (1989), psychological criteria could account for fatigue before physiological criteria.

RATINGS OF PERCEIVED EXERTION (RPE)

The Washie runners were asked to indicate an RPE score at two- hourly intervals until they completed the race. To account for differences in running times, these ratings were

then expressed relative to % running time. Figure 9 indicates the mean local, central and overall RPE.

One-way repeated measures Analyses of Variance (ANOVA) (Ferguson,1981) were performed on the data. Table VI contains the local, central and overall RPE scores and running times where the ANOVA showed differences to be significant ($p<0.05$).

Local RPE increased significantly ($p<0.05$) from 10 to 40 % running time. Then, between 40 and 70 % running time the local RPE did not change. Thus between 40 and 70 % running time there is a 'plateau effect'.

Figure 9 also illustrates the central RPE. The one-way repeated measures ANOVA (see Table VI) revealed that the only significant differences ($p<0.05$) existed between 10 and 90% and between 20 and 90 %. There was large variability which was probably as a result of there being no specific cues for central RPE.

The overall RPE data are also displayed in Figure 9. It follows a similar trend to local RPE because the local RPE is the main 'driving force' of overall RPE. The overall data were also subjected to a one-way ANOVA (Table VI). As for local RPE, a 'plateau effect' exists, between 50 and 80 % running time.

Thus it seems that local RPE values increased significantly ($p<0.05$) during the first 40 % of the race, and overall RPE in the first 50 %. Then both levelled-out to form a plateau, only to again increase significantly until completion of the race.

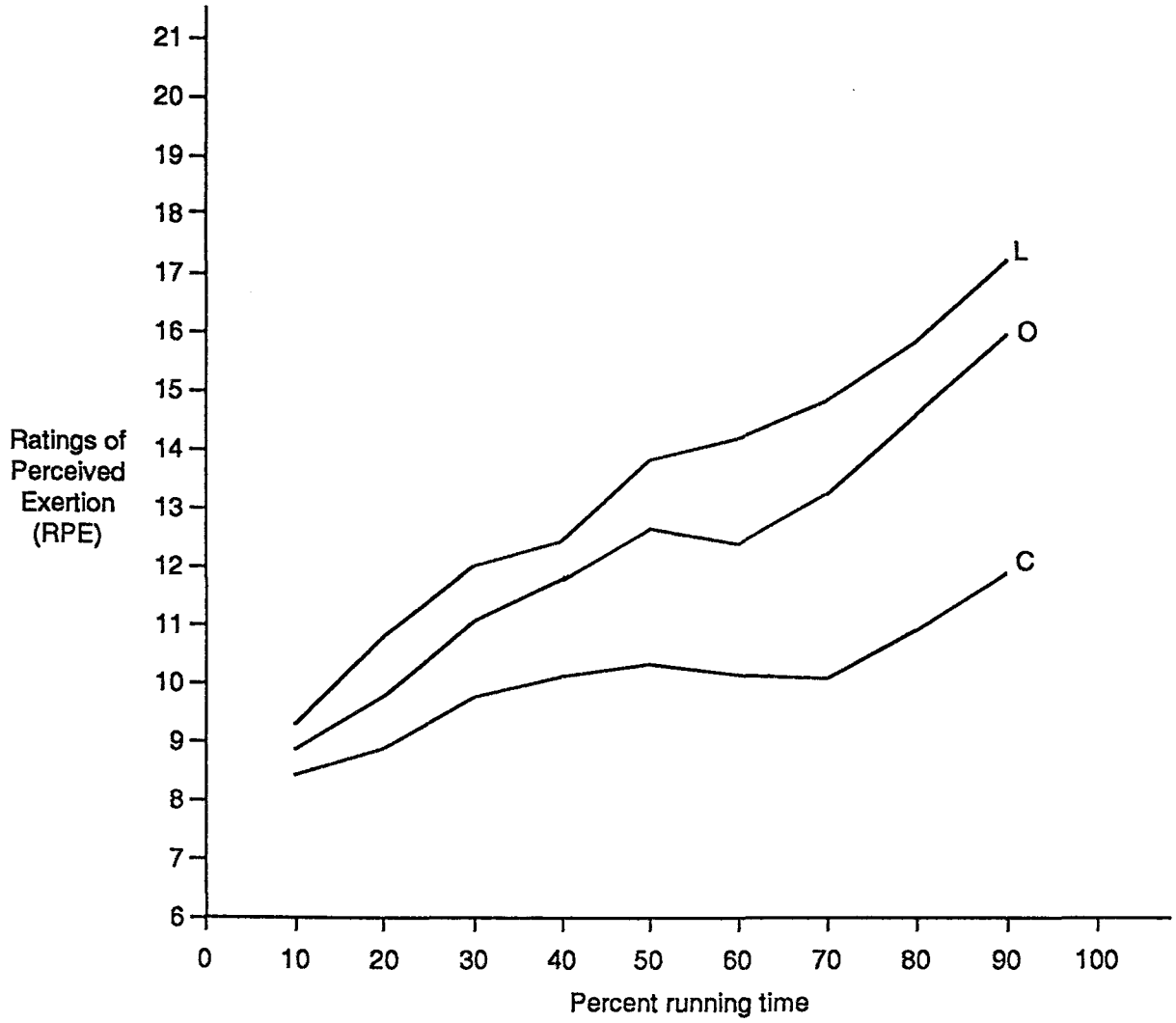


FIGURE 9: MEAN LOCAL (L), CENTRAL (C) AND OVERALL (O) RATINGS OF PERCEIVED EXERTION (RPE) OF THE 21 WASHIE RUNNERS RELATIVE TO PERCENT RUNNING TIME

Table VI : Local, central and overall ratings of perceived exertion (RPE) and percent running times where the repeated measures ANOVA showed differences to be significant ($p<0.05$).

RPE FACTORS	PERCENT RUNNING TIME (%)
LOCAL RPE	10 - 40, 50, 60, 70, 80, 90
	20 - 50, 60, 70, 80, 90
	30 - 80, 90
	40 - 80, 90
	50 - 90
	60 - 90
CENTRAL RPE	10 - 90
	20 - 90
OVERALL RPE	10 - 50, 60, 70, 80, 90
	20 - 70, 80, 90
	30 - 80, 90
	40 - 90
	50 - 90
	60 - 90

The 'plateau effect' corresponds to the time of day when cortisol's normal circadian rhythm is increasing to peak at about 08h00 (McCarthy and Dale,1988). As mentioned previously, cortisol enhances gluconeogenesis, opposes the effect of insulin and promotes the mobilization and lipolysis of triglycerides. Thus increased concentrations of cortisol ensure adequate energy supply, and prevent hypoglycaemia. This may have resulted in less 'strain' being perceived in the legs, hence the 'plateau effect' in local and overall RPE.

Although overall RPE follows a similar trend to local RPE it is not identical. Overall RPE is not simply the sum of local and central RPE and is therefore not mid-way between local and central RPE.

Figure 10 is an interaction plot that illustrates the fact that local RPE is the dominating factor. Central RPE did not change as dramatically as local or overall RPE. From Figure 10 it becomes apparent that initially all three factors are similar : at 10 % running time the differences between local, central and overall RPE are minimal. As running time increases the differences between local and central, and central and overall RPE become more pronounced, as illustrated by the 'V' becoming narrower. Thus local RPE is the main factor affecting perception of effort.

Figure 11 shows the regression lines of RPE and % running time for the three RPE factors. The best prediction equations for these factors were linear (see Figure 11). Local and

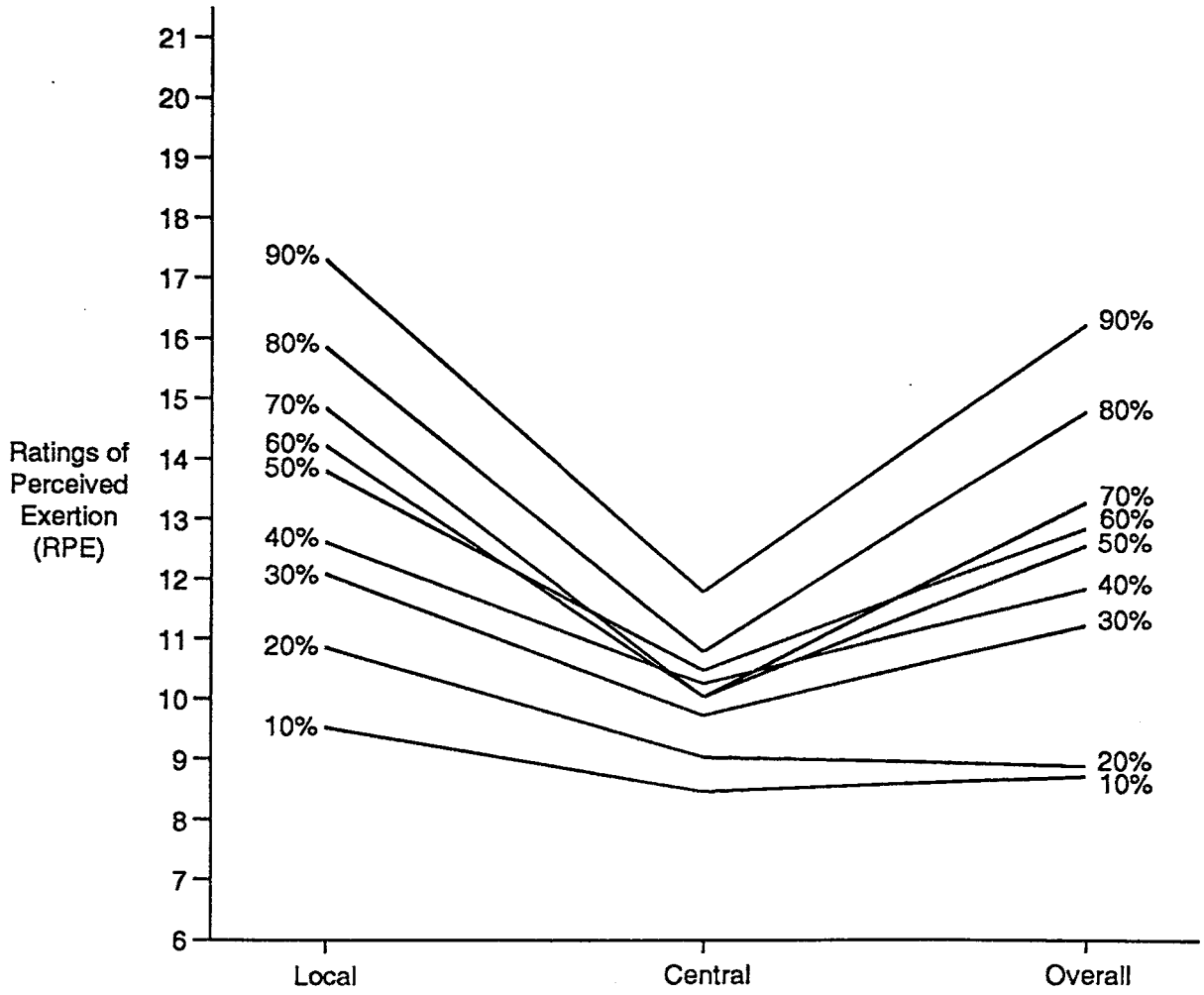


FIGURE 10: INTERACTION PLOT OF LOCAL, CENTRAL AND OVERALL RATINGS OF PERCEIVED EXERTION (RPE) OF THE WASHIE RUNNERS AT 10 TO 90% RUNNING TIME

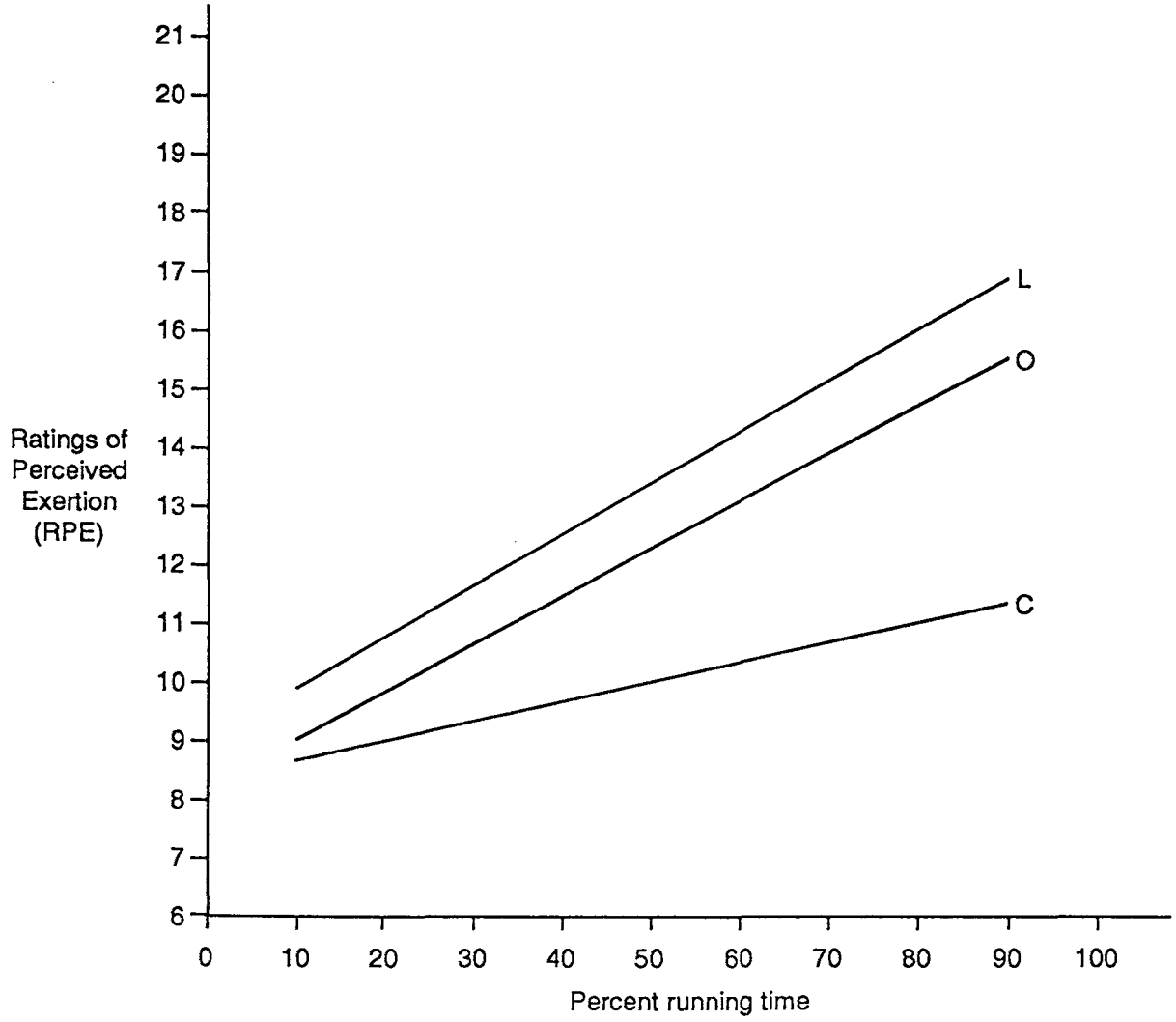


FIGURE 11: *REGRESSION FIT FOR LOCAL, CENTRAL AND OVERALL RATINGS OF PERCEIVED EXERTION (RPE) RELATIVE TO PERCENT RUNNING TIME.*

Key: *L = Local*
C = Central
O = Overall

L: y = 0.09x + 8.98
C: y = 0.03x + 8.32
O: y = 0.08x + 8.16

overall RPE regression lines are almost parallel. Thus if one measured overall RPE it would be possible to make a fairly accurate estimate of the local RPE at that running time, for these runners at least. Local RPE could be predicted from the following equation :

local RPE = overall RPE + (0.01x + 0.82)

where x is % running time.

A comparison was made between the five fastest and the five slowest runners in this sample to see if RPE were any different in these two extreme groups.

Figure 12 illustrates the mean local RPE of the fastest and slowest finishers. The fastest runners exhibited lower perceived exertion ratings than the slowest runners, throughout the race.

After one-way ANOVAS had been performed on the data, it became evident that the fastest runners did not experience the 'plateau effect' in any of the RPE factors, but the slowest runners did.

In Figure 13 the five fastest Washie runners also rated the central factor lower than did the five slowest runners sampled. A two-way ANOVA performed on the data showed that there was no significant difference between the central RPE of the fastest or the slowest Washie runners in this sample. A one-way ANOVA showed that there was no significant difference in central RPE at any given % running time for either group. Again there is large variability which is probably because there were no direct cues for rating central exertion.

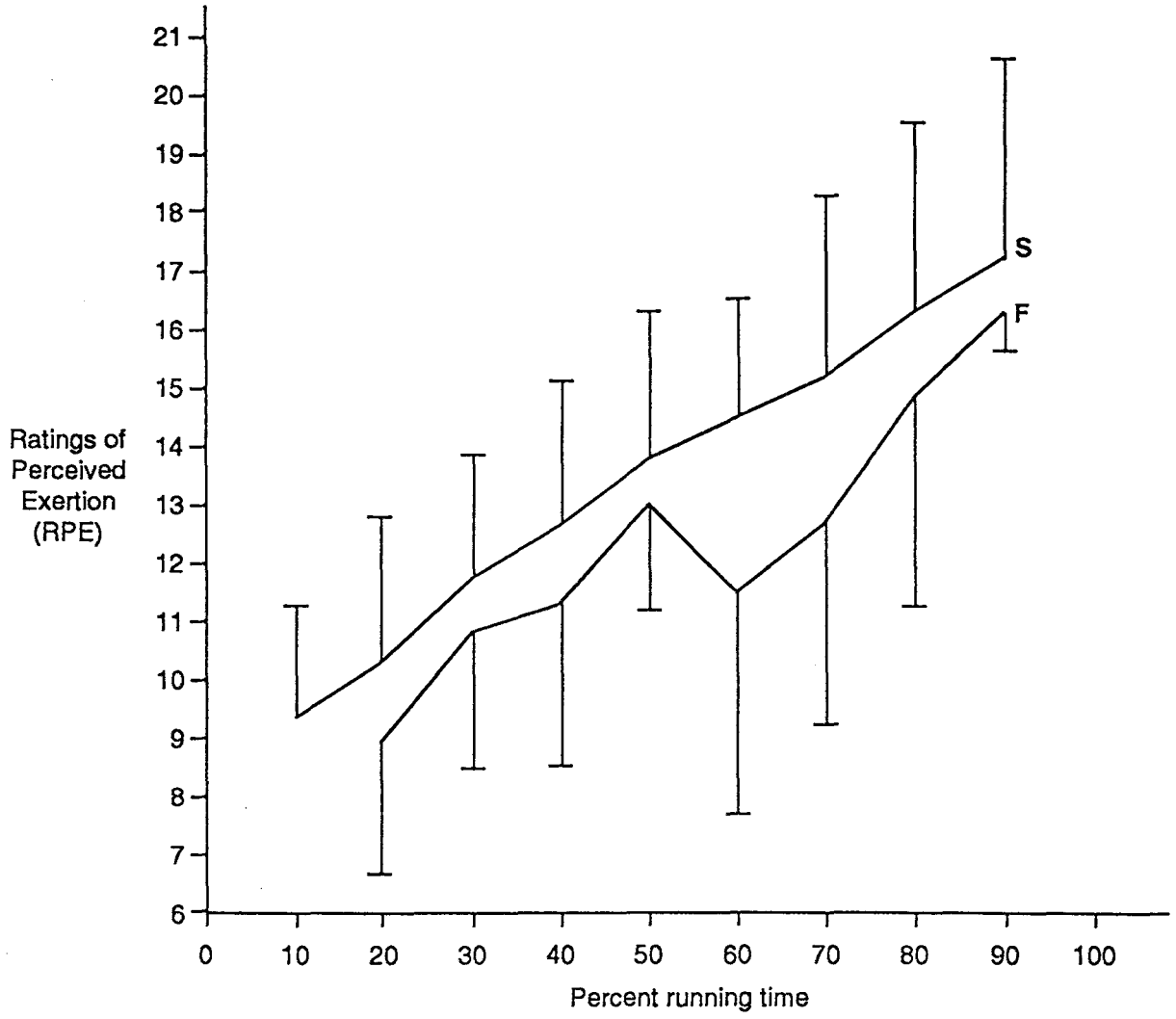


FIGURE 12: *MEAN LOCAL RATINGS OF PERCEIVED EXERTION (RPE) OF THE 5 FASTEST (F) AND 5 SLOWEST (S) WASHIE RUNNERS IN THIS SAMPLE RELATIVE TO PERCENT RUNNING TIME.*

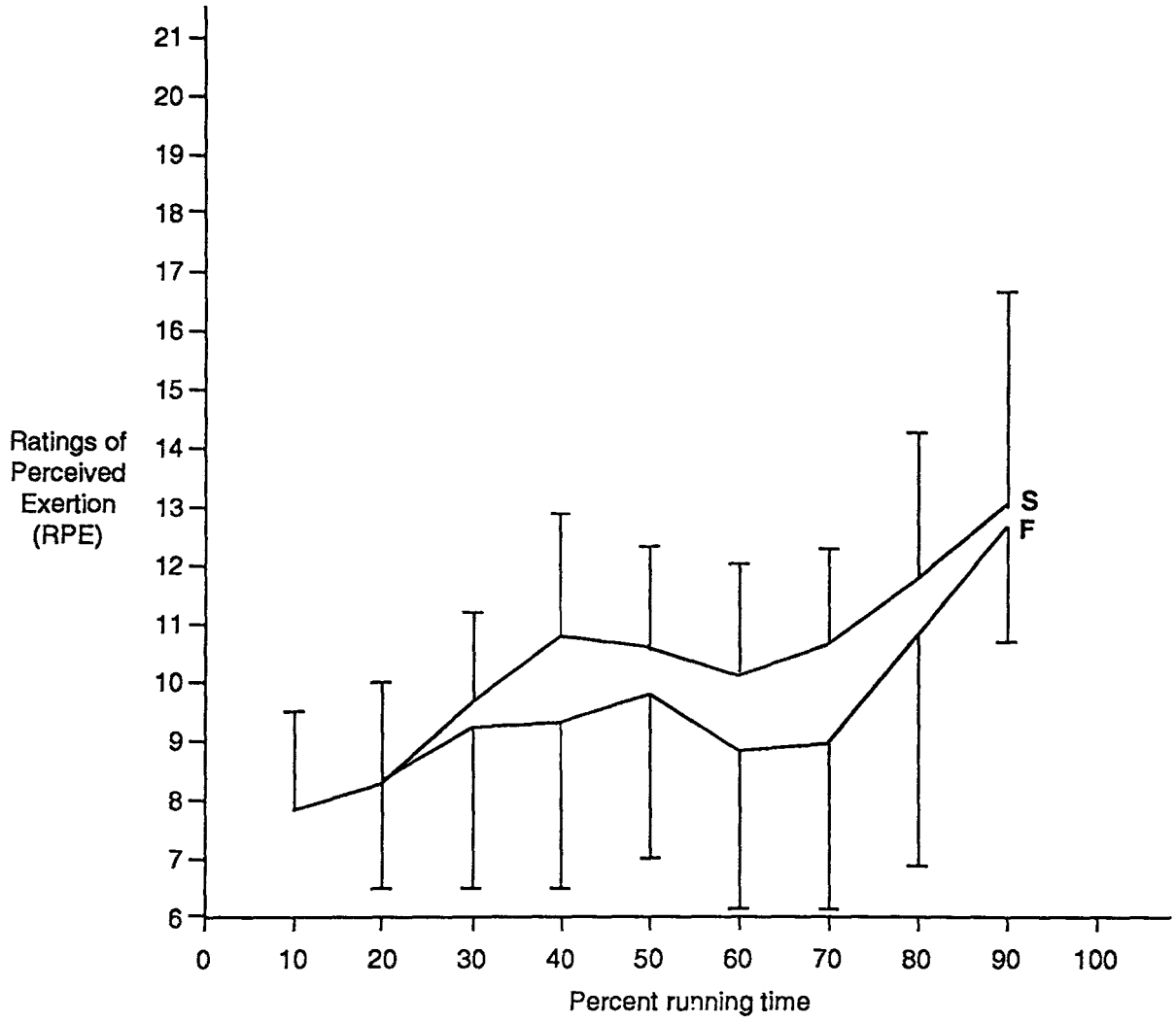


FIGURE 13: *MEAN CENTRAL RATINGS OF PERCEIVED EXERTION (RPE) OF THE 5 FASTEST (F) AND 5 SLOWEST (S) WASHIE RUNNERS IN THIS SAMPLE RELATIVE TO PERCENT RUNNING TIME.*

The difference in overall RPE between the fastest and slowest runners increased as the running time increased (see Figure 14). The theory of Ayoub and Mital (1989) states that as the duration of working or exercising increases beyond a certain point the psychological factors affecting fatigue start to dominate over the physiological factors. Overall RPE indicates the 'general well-being' of the individual regarding perception of effort. Therefore it may include psychological factors, for example boredom. As percent running time increases, psychological factors may play an increasing role in overall RPE.

A two-way ANOVA indicated no significant differences in the way either group rated overall exertion. Only the slowest group exhibited the 'plateau effect' between 50 and 70 % running time.

In a study done by Pandolf (1982) local components played a more important role in the perception of effort than central components. The results of the present study support Pandolf's findings.

Sleep deprivation has been shown to increase RPE at any intensity (Shephard,1984). One of the effects of sleep deprivation became evident when post-race "depression" and "confusion" (POMS scale) were found to be significantly higher than pre-race levels. These higher levels in "depression" and "confusion" may have affected overall RPE. Sleep deprivation also tends to decrease motivation

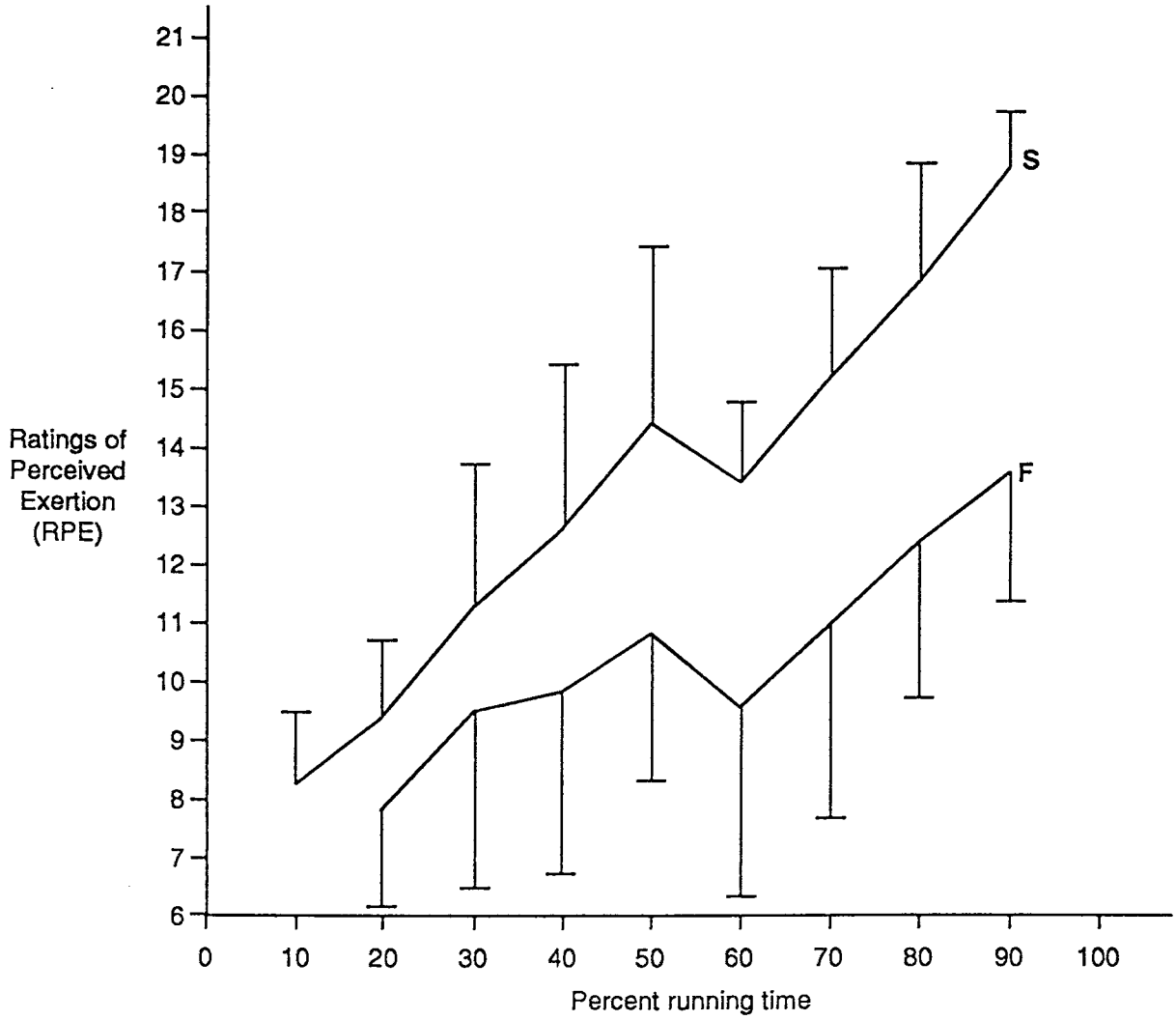


FIGURE 14: *MEAN OVERALL RATINGS OF PERCEIVED EXERTION (RPE) OF THE 5 FASTEST (F) AND 5 SLOWEST (S) WASHIE RUNNERS IN THIS SAMPLE RELATIVE TO PERCENT RUNNING TIME.*

(Shephard,1984) which may cause the subject to have feelings of "having had enough". The feelings of sleepiness and subsequent lapses of attention known as 'micro-sleep', could add to these feelings and thereby cause the overall RPE to dominate over local and central RPE.

Thus the present research provides support for 'local' dominance over 'central'.

Strong correlations between RPE and blood lactate were reported by Carton and Rhodes (1985). When energy has to be supplemented by means of anaerobic metabolism central cues become the main driving force in RPE. Unfortunately no blood lactate measurements were made during this study but since the runners seem to have run at an average of about 32% VO_2max, lactate should not have been increased significantly. However, according to the results obtained in this study, the extreme duration of exercise has a similar affect on local and central RPE as high-intensity exercise does. Thus it seems that Mihevic's (1981) proposal that "...as the intensity of activity increases, the sensory input from local cues becomes gradually more influential" is not only dependent on intensity but also on duration.

Pandolf et al. (1972) stated that RPE during prolonged work initially indicates the adaptation to the work but later it becomes not a perception of effort but of fatigue. Thus the ratings of perceived exertion depicted in Figure 9 may have been ratings of perceived fatigue.

Morgan (1981) suggested that cognition influences RPE. Since all the Washie runners were experienced road runners, they

probably had certain expectations of what effect running 160 km would have on them. This may have influenced how they rated local, central and overall exertion. According to Rejeski (1981) the effect cognition has on RPE is greater at submaximal exercise, especially when the duration is long. During exercise of such long duration the importance of 'dissociative' strategies becomes obvious. By 'dissociating' the subject may suppress internal sensations such as discomfort and/or pain and thereby continue running. 'Micro-sleep' caused by sleep deprivation may be a form of 'dissociation'. The 'dissociative' strategies may all lower local, central and overall RPE.

THE EYSENCK PERSONALITY INVENTORY (EPI)

It takes a certain 'type' of person to run 160 km. Therefore some assessment of personality must be obtained in order to fully understand and interpret the various responses of the subjects.

Eysenck's personality inventory (EPI) is a universally accepted means of assessment of personality. The shortened version (1958) was specifically developed to facilitate applications in diverse circumstances.

On this scale Eysenck dichotomises between "neuroticism" and "stability".

Results of the present study demonstrated that the Washie runners tended towards neuroticism. The mean score on the Neuroticism scale was +1.4 (± 4.5).

Morgan and Costill (1972) reported a neuroticism score of +0.5 in marathon runners they studied. This is interesting because according to Ellison and Freischlag (1975) long distance running demands persistence which would appear to be associated with stability rather than neuroticism. Thus it is interesting to note that the Washie runners as a group were more neurotic than stable. However, it is not clear whether stability is developed during training or whether it is an innate quality.

According to Eysenck (1960) personality has a biological origin and is dependent on the level of neural activity. The continuum between neuroticism and stability depicts the autonomic nervous system's excitatory ability with the

hypothalamus playing a dominant role (Eysenck,1960).

The Eysenck scale also dichotomises between "extroversion" and "introversion". The mean EPI score for extroversion for the Washie runners studied was +4.1 (± 1.8). These results are in contrast with those of Morgan and Costill (1972) whose marathon runners were more introverted than extroverted. The continuum between extroversion and introversion depicts the central nervous system's excitatory and inhibitory functions with the reticular activating system (RAS) being the main controlling centre. The RAS is in the midventral part of the medulla and midbrain. It contains ascending and descending components which are important in the regulation of sensory input, consciousness, arousal level and endocrine secretion. There are also centres that regulate heart rate, blood pressure and respiration. Part of the RAS supplies the cortex, part goes to certain nuclei of the thalamus and part goes to the spinal cord (Barr and Kiernan,1987;Ganong,1985). During sleep the activity of the RAS is decreased but almost any type of sensory stimulus can activate the system and therefore arouse the person. This is known as arousal reaction. Some sensory stimuli are more potent than others - the most potent being pain and proprioceptive impulses (Guyton,1981). During wakeful times the 'degree of wakefulness' can vary i.e. there are differing levels of arousal. The inverted-U-hypothesis illustrating the relationship between arousal and performance was developed by Yerkes and Dodson in 1908. The hypothesis has engendered

controversy for 80 years, an indication of its profound impact, but it continues to be used in various performance situations (Kerr,1985). This hypothesis states that initially performance increases as arousal increases but that at some point further increases in arousal become detrimental to performance. This point has been called the "...point of optimal arousal" (Kerr,1985); it varies from individual to individual and from situation to situation. Moderate levels of arousal are usually best for high performance in sport (Oxendine,1970). Although the Inverted-U-Theory has been criticised (Kerr,1985), it seems to do well in practice. Unfortunately the level of arousal of the Washie runners was not assessed. However, throughout the race there will have been an optimal level of arousal at which performance could be maintained.

While the neuroticism and extroversion scale results have been discussed independently in order to establish an overall personality profile, it is necessary to note the interaction of the two. The relationship between neuroticism-stability and extroversion-introversion is best displayed graphically. Figure 15 shows the mean of the 21 Washie runners studied, along with those of marathon runners studied by Morgan and Costill (1972), South African interprovincial hockey players (Scott,1990), and normal adults. The hockey players, being involved in team sport, showed expected high extroversion and low neuroticism. In order to function optimally as a team, the individual members are expected to be fairly stable

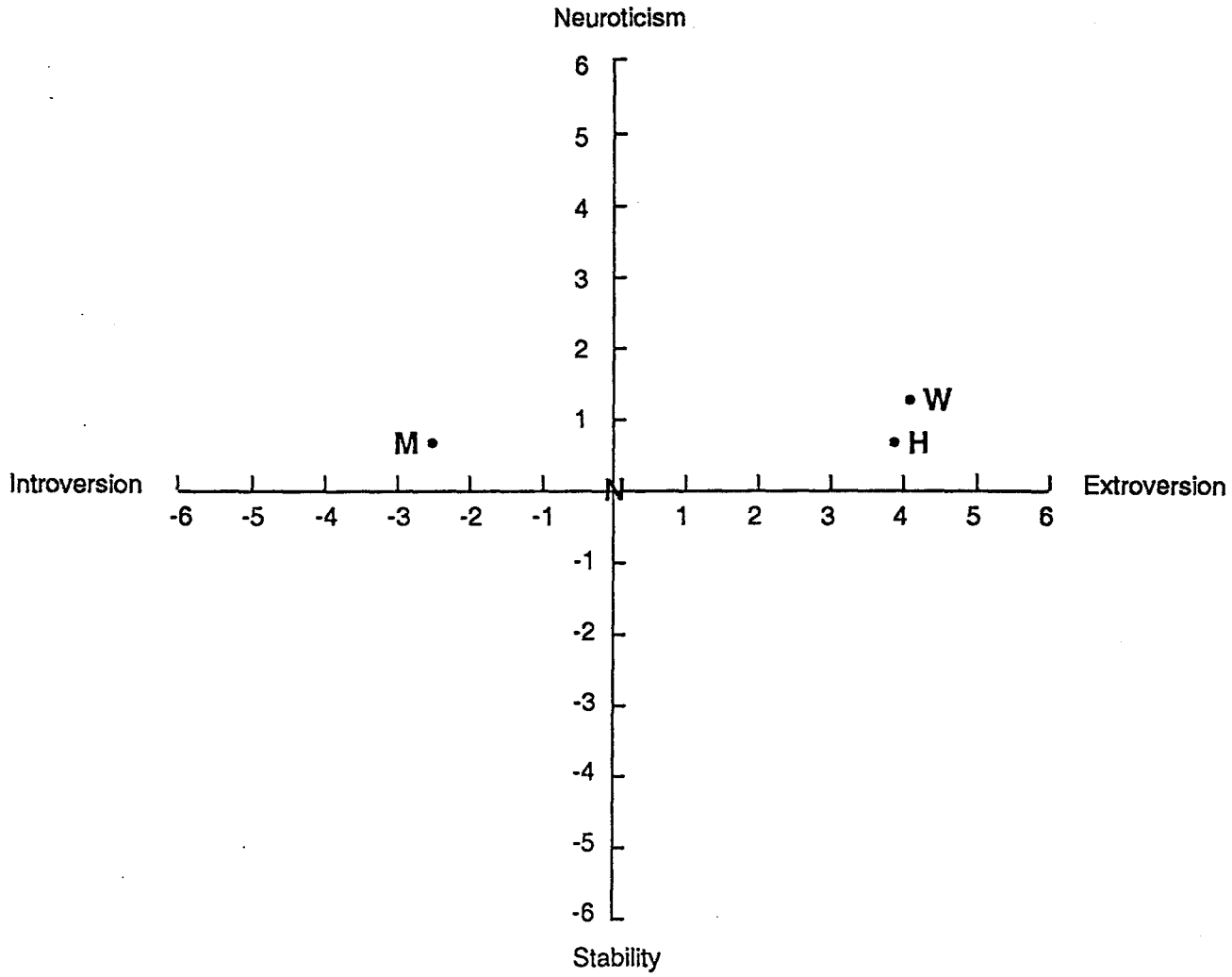


FIGURE 15: *MEAN EYSENCK PERSONALITY INVENTORY (EPI) SCORES OF THE WASHIE RUNNERS (W), NORMAL ADULTS (N), MARATHON RUNNERS ACCORDING TO MORGAN AND COSTILL (1972) (M) AND SOUTH AFRICAN MALE HOCKEY PLAYERS (SCOTT, 1990) (H).*

because the team has to function as a unit.

According to Kirkcaldy (1980) neuroticism and extroversion are often related. In other words if someone is found to be neurotic, the possibility that that person is also extroverted is, according to Kirkcaldy more than 50%. Morgan and Costill's (1972) marathon runners were neurotic and introverted, which does not mean they were abnormal, simply that Kirkcaldy's findings are a generalization.

The degree of neuroticism seems to correlate with the sport and with proficiency in it (Thomas et al.,1983).

Eysenck (1967) postulated that extroverts were less sensitive to sensory stimuli than introverts. This is apparently due to 'cortical inhibition' (Kirkcaldy,1980). It could thus be argued that extroverts tend to have a higher pain threshold than introverts. As the runners were working at less than 50% $\dot{V}O_2max$ there probably was not a major accumulation of lactic acid. However the effect of running 160 km on the active muscles and joints will in all probability have resulted in varying degrees of pain being experienced by the runners. Local RPE substantiates this : as the race progressed the runners perceived greater 'strain' in their lower limbs (Figure 9). The higher the pain threshold the less the discomfort experienced while running 160 km.

Ellison and Freischlag (1975) reported that there appears to be an association between having a high pain tolerance and a low level of arousal. It could be argued that this is because pain induces arousal, which in turn affects performance positively or negatively (Brewer et al.,1990).

Pain as a form of arousal may affect performance negatively because pain perception reduces the amount of attention that the person can focus on the performance (Brewer et al.,1990). As has been mentioned previously, using "dissociative" strategies is important in long distance running. The runner will "dissociate" in order to suppress the perception of pain and discomfort. As the pain increases beyond the pain threshold it may however become more difficult to "dissociate".

The perception of pain may be different depending on the task being performed i.e. on the 'type' of pain, and on whether it is acute or chronic pain. Pain fibres associated with chronic pain terminate in the reticular area of the brain stem and in the intralaminar nuclei of the thalamus (Ganong,1981). Both the reticular area and the intralaminar nuclei of the thalamus are part of the reticular activating system (RAS). Therefore when these chronic pain fibres are excited, the RAS is excited, which in turn affects arousal. This is how pain can induce arousal. When arousal (RAS activity) is low, the standard of performance is generally low. When arousal (RAS activity) increases, performance increases to a certain extent, then decreases. This is the Inverted-U-Theory as described earlier.

Neurotic extroverts may experience less discomfort due to 'cortical inhibition', but they may experience a greater 'drop-off' in motivation and decreased co-ordination as a result of being more sensitive to sleep deprivation.

According to Shephard (1984) neurotic and extroverted people are particularly sensitive to sleep deprivation. Theoretically sleep deprivation causes a decrease in cortical arousal, weakens motivation and impairs co-ordination (Schomer,1990). Thus running through the night as the Washie runners did, resulted in sleep deprivation, which will have decreased arousal and motivation, and impaired co-ordination. The decreased arousal and motivation will have resulted in higher RPE scores, particularly overall RPE because it becomes more difficult to continue running when motivation decreases. Also, the impaired co-ordination will have increased local RPE and have resulted in altering stride length. Since it is more economical to "under-stride" (McArdle et al.,1986), the runners will have started "shuffling".

Locke et al. (1981) define goal-setting as "...that which an individual is trying to accomplish, it is the object or aim of an action". Most goal-setting requires getting something done in a certain time and involves getting it done according to a certain standard. A more difficult goal requires more effort and attention than an easier goal. More demanding goals often also require more knowledge about the task and its environment, and more skill. The goal setting of the Washie runners in this sample varied considerably. Trying to finish in the first 10 for instance, is a more demanding goal than simply wanting to finish within the required time. Other psychological factors which need to be considered are motivation and the degree of competitiveness. The

'achievement motivation' theory of Atkinson and Feather (1966) depends on three factors : the probability of achieving a goal (expectancy), how 'desperately' the person wants to achieve the goal (motive strength), and the incentive for succeeding. Motive strength and the incentive for succeeding may be two different factors e.g. motive strength may be intrinsic and incentive for succeeding extrinsic. The motive strength of the runners in the present study was not ascertained and therefore there was no way of knowing whether their motivational drives were high or low. The incentive for succeeding was probably mostly intrinsic i.e. the desire to complete the distance for personal sense of achievement. There were no extrinsic motivators in the form of money. Most of the runners in this sample ran to achieve personal goals, not to win. Winning and losing are not synonymous with success and failure (Roberts and Duda,1984). Success and failure depend on the individual's interpretation of the outcome. Therefore a similar outcome may be 'success' to one person and 'failure' to another (Roberts and Duda,1984).

PROFILE OF MOOD STATES (POMS)

Moods reflect the physiological 'well-being' of the individual, how the individual's environment is perceived, and the amount of success recently experienced (Maddi,1968). Thus the time period of a particular mood state is dependent on all three factors.

It is generally accepted that runners exhibit a common mood profile, in that when they are compared to non-runners, the runners tend to exhibit less "tension", "fatigue" and "confusion" , and more "vigour" (Tharion et al.,1988). This is the resting "mood profile", depicted in the classical "Iceberg Profile" presented by Morgan and Pollock in 1977. However, many factors may affect the mood profile. A mood profile obtained days or weeks before an event will probably differ from that obtained just prior to the event (Cockerill et al.,1991). The main reason for this is that moods fluctuate in response to situational demands and their effects on the basic personality factors of the individual differ(Cockerill et al.,1991). The mean running time of the Washie runners in this sample was 22 hours and 6 min. Thus the mean time between pre- and post-race POMS testing was 23 hours since each runner had to attend the pre-race briefing 45 min. before the start of the race.

Table VII gives the pre- and post-race raw scores of the 21 Washie runners in this sample. Figure 16 is a graphical representation of the POMS scores.

Table VII : Raw score means ($\bar{x}$) and standard deviations (s.d.) for the Profile of Mood States (POMS) tests of the 21 Washie runners before and after the race.

Variable	Profile of Mood States (POMS)			
	Pre-race		Post-race	
	$\bar{x}$	s.d.	$\bar{x}$	s.d.
Tension	16.5	7.4	14.4	9.1
Depression	8.2	8.9	14.3	14.6
Anger	7.9	8.4	9.2	9.9
Vigour	20.6	4.9	17.9	5.7
Fatigue	4.8	5.2	19.7	4.4
Confusion	6.6	3.8	10.2	7.0
Total Mood Disturbance	23.5	28.0	49.9	41.6

Bars indicate significant differences ($p<0.05$).

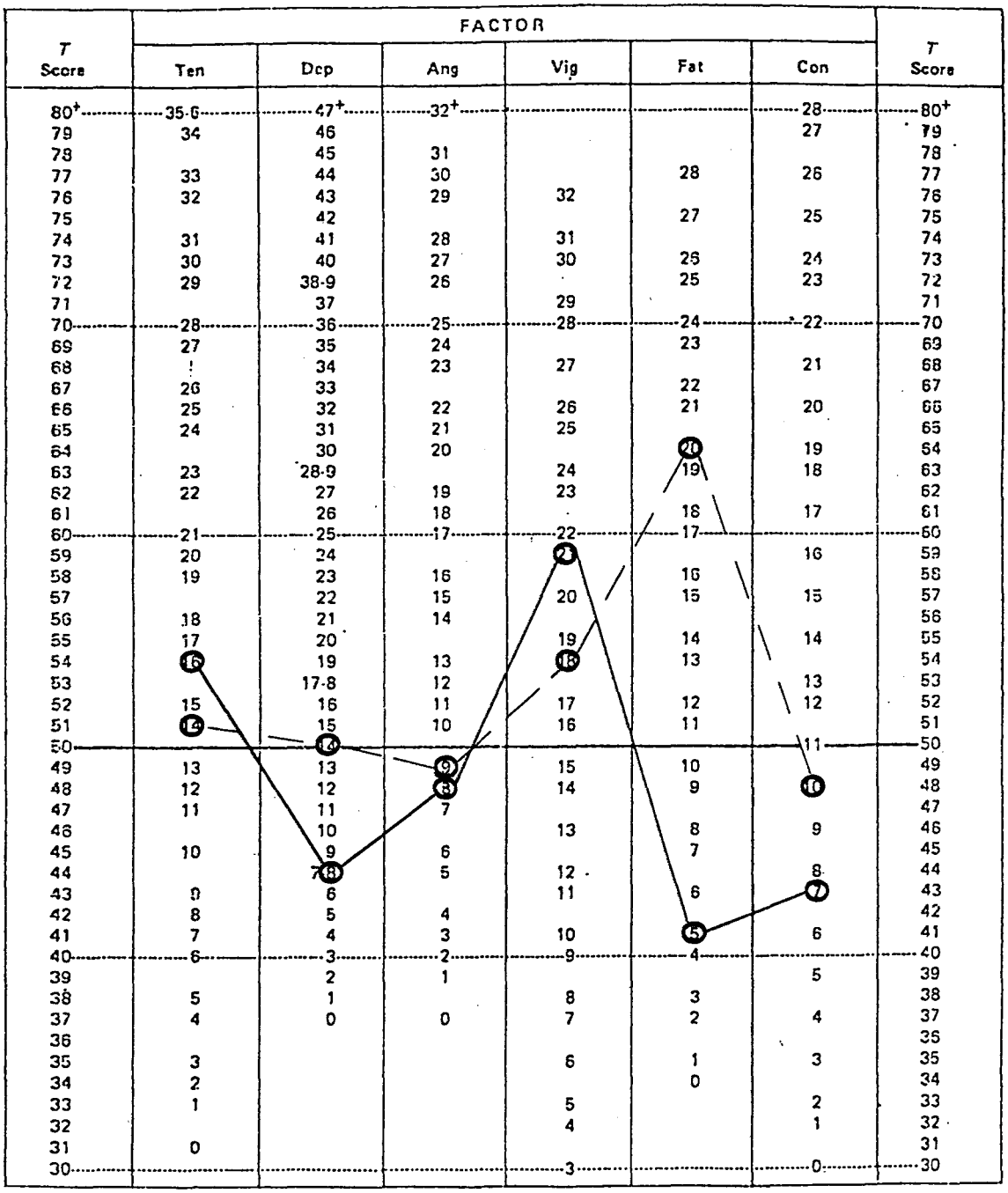


FIGURE 16: *PROFILE OF MOOD STATES (POMS) PROFILE SHEET OF THE 21 WASHIE RUNNERS BEFORE (————) AND AFTER (— — — — —) THE RACE.*

After running 160 km the following mood factors : "depression", "fatigue" and "confusion" were significantly increased (related t-test,$p<0.05$). Interestingly neither "tension", "anger" nor "vigour" were changed significantly, yet it has been well documented that there tend to be significant reductions in "tension" after both acute and chronic exercise (Markoff et al.,1982; Tharion et al.,1988; Scott,1990). According to Cockerill et al. (1991) an elevated level of "tension" is necessary for success in any competitive sport. However, at some point an increased tension may become detrimental to performance. In other words tension may follow a similar trend to that suggested by the 'Inverted U theory' of arousal (Yerkes and Dodson,1908), where either too little or too much tension may hinder performance rather than promote it.

Tharion et al. (1988) measured pre- and post-race POMS scores of runners in 50 mile (80 km) and 100 mile (160km) cross-country events in Virginia. They recorded a mean pre-race raw "tension" score of 6.98 and a post-race score of 7.0. Related t-test analysis showed no significant difference ($p<0.05$) between the pre- and post-race "tension" scores of these cross country runners. These are almost half the scores of the Washie runners, who in both pre- and post-race testing revealed elevated "tension" scores.

Running 160 km is a daunting prospect ; the unpredictability of the race, each runner's performance and weather are all probable factors which may have lead to the Washie runners having such elevated tension scores. One would, however, have

expected this to have changed once the race was over. Perhaps the instructions to answer the questionnaire after the race relative to how each runner had felt during the race influenced the results. If they had been instructed to answer only according to how they felt on completion of the race, the results may well have been different, not only for tension, but for all other factors.

"Anger" levels of the 21 Washie runners were similar to those recorded by Tharion et al. (1988), and these scores showed no significant change pre- and post-race ($p<0.05$). This is in agreement with the findings of Tharion et al. (1988) and Cockerill et al. (1991). In other words the 21 runners as a group were neither more or less angry with themselves or anyone else after the race than before the race. The relief of finally finishing may have had an influence on what is considered a 'negative' mood state. There was no significant difference between the pre- and post-race "vigour" scores ($p<0.05$). In fact the pre-race scores were surprisingly low. The normal 'spike' in the 'Iceberg Profile' was not as pronounced as expected. Perhaps the fact that the race started at 19h00 was a contributing factor i.e. the effect of being awake and active for a full day waiting in anticipation of the start of the race may have had a negative effect on "vigour". Many of the runners had to drive to the start of the race at Port Alfred during the day and were thus not as 'fresh' at 19h00 as they may have been if the race had started in the morning. However these results are similar to those of Tharion et al. (1988) who also found no significant

difference between pre- and post-race "vigour". This was in contrast to the "fatigue" factor where there was a significant increase in post-race "fatigue" ($p<0.05$). The Washie runners in this sample had a mean pre-race "fatigue" score of 4.8 (± 5.2). Individual scores may have differed according to different activity patterns during the waking hours before the race. The post-race fatigue score was 19.7 (± 4.4), almost 4 times the pre-race value. As mentioned previously, the runners were performing at significantly less than 50% $\dot{V}O_2max$. Thus it was not the intensity of the activity that was the main contributing factor: rather one must argue that the main causal factors must be the duration of the race and the sleep deprivation resulting from running through the night. One may have expected a concomitant decrease in vigour with the increase in fatigue. Clearly at the completion of a 160 km run, the effect of the continuous physical demands being compounded by sleep deprivation must result in "fatigue" being experienced.

"Depression" and "confusion" both increased significantly ($p<0.05$). This was probably as a result of sleep deprivation, since all runners ran through the night. Schomer (1990) stated that sleep deprivation of just one night may have marked effects on mood states in runners resulting in irritability and increased fatigue. Some runners may even experience illusions and hallucinations, although this is more common during longer sleep deprivation (60 to 200 hours). In theory, sleep deprivation causes a decrease in cortical arousal, it impairs co-ordination and weakens

motivation (Shephard,1984). The decreased cortical arousal may have led to confusion, and if there had been weakened motivation this may have contributed to the increased level of depression found after the race. Instead of being in a state of euphoria for having run 160 km successfully, the runners as a group, demonstrated an elevation in the "depression" and "confusion" factors. This seems to be reflected by the overall rating of perceived exertion (RPE) since this factor takes into account not only local and central cues but also the psychological 'well-being' at the time. In other words the overall RPE may be interpreted as indicating increasing fatigue - both physiologically and psychologically.

A Total Mood Disturbance (TMD) score is calculated by adding the scores of tension, depression, anger, fatigue and confusion and subtracting vigour (McNair et al.,1971). The 21 Washie runners in this sample had a mean TMD score of 23.5 before the race. This compares favourably with the pre-race TMD value of Morgan et al. (1988), namely 21.2, and of Thomas et al. (1983), namely 24.4. The five factors that are summed may be negative influences on performance when excessive, yet a certain amount may be 'positive'. The effect of excessive "vigour" on the other hand is positive on performance. Since the pre-race vigour scores were fairly low, the pre-race scores of the other five mood states must have been within the normal ranges since the TMD score is average.
The mean post-race TMD score of the Washie runners was 49.9,

which is approximately twice the pre-race TMD. The main contributors to the increase in post-race TMD were the significant increases in "depression", "fatigue" and "confusion", with no significant change in "vigour". The difference in TMD before and after the race was significant, as expected, although Tharion et al. (1988) found no significant difference between pre-race and post-race TMD scores. Unfortunately neither Morgan et al. (1988) or Thomas et al. (1983) measured post-race POMS.

CHAPTER FIVE

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

AIMS OF THE STUDY

This study sought to examine selected anthropometric, physiological and psychological parameters related to ultramarathon running. Particular attention was paid to making it an holistic investigation of ultramarathon running. The questions addressed aimed firstly at determining whether or not there were significant differences between pre- and post-race data obtained and the relevance of these differences in relation to running 160 km. Secondly, the aim was to explain significant differences between ultramarathon runners and marathon runners.

The following test hypotheses were developed in respect of the above questions :

Hypothesis one : That no difference exists between stature or mass before and after the ultramarathon.

Hypothesis two : That there is no difference between pre- and post-race serum cortisol concentrations relative to a 160 km run.

Hypothesis three : That there is no difference in the ratings of local, central and overall ratings of perceived exertion.

Hypothesis four : That there is no difference between POMS scores measured before and after the race.

In addition to these hypotheses, several comparisons between the ultramarathon runners of this study and marathon runners reported in the literature were made.

METHODS

Twenty-one male subjects volunteered to participate in this study (mean age 38.76 ± 6.15 years; stature 177.6 ± 6.4 cm; body mass 77.4 ± 11.4 kg). They all ran the Washie 160 km road race. Several anthropometric measurements were performed before the race, the mean values being: body density 1.057 ± 0.008 and percentage body fat 18.4 ± 3.7 %.

Stature and mass were again measured after the race to determine whether any significant changes had occurred.

Baseline blood samples were taken before the race. Serum cortisol concentration and haematocrit (Hct) were obtained from the blood. Within 10 min of finishing, each subject again had a blood sample taken. Again serum cortisol concentration and Hct were obtained, as well as a white blood cell count (WBC).

Ratings of perceived exertion (RPE) were obtained every 2 hours during the race. Local, central and overall RPE ratings were monitored.

Two psychological tests were completed before the race. They

were the shortened Eysenck Personality Inventory (EPI) and the Profile of Mood States (POMS) test. The POMS test was also completed after the race.

The results were analysed using single-variable statistics, "students" t-tests and Analysis of Variance tests (ANOVAS). A 0.05 level of probability was chosen.

RESULTS

1) The mean predicted running intensity was found to be well below the anaerobic threshold intensity. The anaerobic threshold of trained male runners is about 75% $\dot{V}O_2$max (de Mello,1985) and the Washie runners were running at an average intensity of about 32% $\dot{V}O_2$max. This probably did not elicit a major production of lactic acid. Thus it seems that it was the duration rather than the intensity of running that was stressful.

2) Before the race the mean stature was 177.6 (± 6,4) cm and the mean mass 77.4 (± 11.4) kg. Immediately after the race the mean stature was 176.6 (± 6.3) cm indicating a significant ($p<0,05$) decrease as a result of repetitive compressive forces on the subjects' intervertebral discs. The mean body mass after the race was significantly less at 73.9 (± 10.8) kg; a decrease of 4.5 %.

3) The physiological parameters measured before and after the race showed significant changes. Plasma volume was greater immediately after the race. This phenomenon is known as 'plasma volume expansion' (Ritschel,1986). The nature of the relationships between intensity, duration and plasma cortisol concentrations is not clear (McCarthy and Dale,1988). In this study cortisol concentrations were significantly increased after the race. These findings are consistent with those of several authors who tested cortisol changes in exercise of differing intensity and duration (Hartley et al.,1972; von Glutz et al.,1978; Keul et al.,1981). The leucocytosis observed appears to have been as a result of elevated plasma cortisol concentrations.

4) Throughout the race local ratings of perceived exertion (RPE) were always higher than overall ratings, which in turn were always higher than central ratings. As running time increased local and overall RPE appeared to increase in an almost linear fashion. Between 40 and 70 % running time there seemed to be a 'plateau effect' in local RPE, and between 50 and 80 % running time overall RPE displayed the 'plateau effect', where the increase in RPE was not significant. Central RPE did not change significantly as running time increased.

In previous work, Pandolf (1982) stated that local components play a more important role in the perception of effort than central components. The results of the present study support the above conclusion.

5) Pre- and post-race Profile of Mood States (POMS) differed significantly ($p<0.05$). The mood factors "depression", "fatigue" and "confusion" were significantly increased after the race ($p<0.05$). Sleep deprivation was probably an important contributing factor.

6) The anthropometric profiles of the 21 Washie runners were different to those of marathon runners reported in the literature. The Washie runners were taller (177.6 ± 6.4 cm) and heavier (77.4 ± 11.4 kg) before the race than marathon runners measured by Tanner (1964), Costill (1972), and Pollock et al. (1977). The mean body mass index (24.55) showed the Washie runners in this sample to be more like the general population than marathon runners. Also, the Washie runners were more ponderous (RPI 41.85) than Olympic sprinters, let alone Olympic marathon runners. The Washie runners were found to have greater percent body fat (18.4 ± 3.7 %) than the marathon runners of Costill et al. (1970), Pollock et al. (1977) and Tanaka and Matsuura (1982). The Washie runners' somatotype (3.1;4.8;2.1) also differed from those reported for marathon runners. They were more endomorphic and mesomorphic and less ectomorphic than the marathon runners. The somatotype dispersion distance indicated that the Washie runners' somatotypes were more like those of elite sprinters than marathon runners.

7) The personality parameters investigated in this study (introversion - extroversion and neuroticism - stability)

differed from those found in marathon runners (Morgan and Costill,1972). The Washie runners had similar neuroticism scores (+1.4 ± 4.5) to marathon runners but they were significantly ($p<0.05$) more extroverted (+4.1 ± 1.8) than the marathon runners. Extroversion has been linked with having a higher pain threshold (Eysenck,1967). The effect running has on active muscles and joints appeared to result in varying degrees of mechanical pain experienced. The fact that the Washie runners were more extroverted and therefore probably had a higher pain threshold, was probably an important contributing factor to being able to run for such an extended period of time.

8) The Washie runners exhibited a similar Profile of Mood States (POMS) to marathon runners. The Washie runners also exhibited the classical "Iceberg Profile" presented by Morgan and Pollock (1977).

CONCLUSIONS

1) Running 160 km resulted in significant decreases in stature and mass.

2) The 'stressor' - running 160 km - resulted in a 'strain response' as indicated by an increase in serum cortisol concentrations.

3) The use of three forms of RPE as a means of quantifying subjective fatigue identified that local RPE was always the dominant factor, whereas central RPE did not change much as the race progressed. Overall RPE was influenced largely by the dominant local RPE.

4) Running 160 km resulted in major changes in certain mood factors, notably "depression", "fatigue" and "confusion".

5) The anthropometric profiles of the ultramarathon runners in this sample were distinctly different from those of marathon runners tested elsewhere, even when allowance for ethnic differences, in some cases, is made. The ultramarathon runners were taller, heavier and more ponderous than marathon runners.

6) Ultramarathon runners are more extroverted than marathon runners according to Eysenck's Personality Inventory.

7) Ultramarathon runners appear to exhibit similar mood profiles (according to the Profile of Mood States test) to marathon runners.

In light of these results the following conclusions can be drawn :

Hypothesis one : Rejection of the null hypothesis.
The findings in this study lead one to tentatively accept the Alternative Hypothesis ($p<0.05$) as follows : that there is a difference in stature and mass before and after the ultramarathon.

Hypothesis two : Rejection of the null hypothesis.
The findings in this study lead one to tentatively accept the Alternative Hypothesis ($p<0.05$) as follows : that there is a difference between serum cortisol concentrations before and after the race.

Hypothesis three : Rejection of the null hypothesis.
The findings in this study lead one to tentatively accept the Alternative Hypothesis as follows : that there is a difference between local, central and overall RPE.

Hypothesis four : Rejection of the null hypothesis.
The findings of this study support tentative acceptance of the Alternative Hypothesis ($p<0.05$) as follows : that there is a difference in Profile of Mood States (POMS) before the race and after the race.

In addition to the hypotheses certain comparisons were made between ultramarathon runners and marathon runners from the literature :

a. ultramarathon runners have different anthropometric profiles to marathon runners,
b. ultramarathon runners are more extroverted (according to the EPI test) than marathon runners , and
c. ultramarathon runners have similar pre-race mood profiles (according to POMS) to marathon runners.

RECOMMENDATIONS

1) Since running intensity had to be predicted, future researchers would be advised to monitor this parameter carefully. Actual measurement of $\dot{V}O_2max$ and anaerobic threshold would also be beneficial.

2) In this study cortisol was only analysed from blood taken before and after the race. It is suggested that in future research blood samples are drawn during the race at certain intervals.

3) Future researchers may also concentrate on examining the recovery period.

REFERENCES

Note: Asterisked citations are secondary sources. These were not directly consulted and are referenced as fully as primary sources, indicated in brackets, permit.

* Ash IE (1914). Fatigue and its effects upon control. **Archives of Psychology**, 4 : 1 - 61 (see Hockey,1983).

Åstrand P-O and Rodahl K (1977). **Textbook of Work Physiology.** New York : McGraw-Hill Book Company.

Åstrand P-O and Saltin B (1964). Plasma and red cell volume after prolonged severe exercise. **Journal of Applied Physiology**, 19 : 829 - 832.

Atkinson JW and Feather NT (1966). **A Theory of Achievement Motivation.** New York : Wiley.

Ayoub MM and Mital A (1989). **Manual Materials Handling.** London: Taylor and Francis.

* Baetzner W (1926). Sportschaeden am Bewegungsapparat. **Klinische Wochenschrift**, 5 : 653 - 654 (see Helminen et al.,1987).

Bailey DA, Carter JEL and Mirwald RL (1982). Somatotypes of men and women. **Human Biology**, 54 (4) : 813 - 828.

Banister EW, Rajendra W and Mutch BJC (1985). Ammonia as an indicator of exercise stress - implications of recent findings to sports medicine. **Sports Medicine**, 2 : 34 - 46.

Barr ML and Kiernan JA (1987). **The Human Nervous System**, 5th edition. Philadelphia : J.B. Lippincott Company.

* Bartley SH and Chute E (1947). **Fatigue and impairment in man**. New York : McGraw - Hill (see Hockey,1983).

Berger BG (1984). Stress reduction through exercise : the mind-body connection. **Motor Skills : Theory into Practice**, 7 (2) : 31 - 46.

Berger BG and Owen DR (1988). Stress reduction and mood enhancement in four exercise modes : swimming, body conditioning, Hutha yoka, and fencing. **Research Quarterly for Exercise and Sport**, 59 (2) : 148 - 159.

* Bink B (1962). The physical working capacity in relation to working time and age. **Ergonomics**, 5 (1) : 25 - 28 (see Ayoub and Mital,1989).

Birk TJ and Birk CA (1987). Use of ratings of perceived exertion for exercise prescription. **Sports Medicine**, 4 (1) : 1 - 8.

Borg G (1970). Perceived exertion as an indicator of somatic stress. **Scandinavian Journal of Rehabilitation Medicine,** 2 : 92 - 98.

Borg G (1982). Psychological bases of perceived exertion. **Medicine and Science in Sport and Exercise,** 14 (5) : 377 - 381.

Braun JJ and Linder DE (1979). **Psychology Today,** 4th edition. New York : Random House.

Brewer BW, Van Raalte JL and Linder DE (1990). Effects of pain on motor performance. **Journal of Sport and Exercise Psychology,** 12 (4) : 353 - 365.

Brotherhood JR (1984). Nutrition and sports performance. **Sports Medicine,** 1 (5) : 350 - 389.

Bunt JC (1986). Hormonal alterations due to exercise. **Sports Medicine,** 3 (5) : 331 - 345.

Burke LM and Read RSD (1989). Sports nutrition. **Sports Medicine,** 8 (2) : 80 - 100.

Cafarelli E (1982). Peripheral contributions to the perception of effort. **Medicine and Science in Sports and Exercise,** 14 (5) : 382 - 389.

Cameron C (1974). A theory of fatigue. In AT Welford (Ed). **Man under Stress**. London : Taylor and Francis Ltd.

Cappon D and Banks R (1960). Studies in perceptual distortion. **Archives of General Psychiatry**, 2 : 346 - 349.

Carter JEL (1980). **The Heath-Carter Somatotype Method.**San Diego: San Diego University Press.

Carter JEL (1984a). Age and body size of Olympic athletes. **Medicine and Sports Science**, 18 (1) : 53 - 79.

Carter JEL (1984b). Somatotypes of Olympic athletes from 1948 to 1976. **Medicine and Sports Science**, 18 (1) : 80 -109.

Carter JEL and Heath BH (1990). **Somatotyping : development and applications**. Cambridge : Cambridge University Press.

Carton RL and Rhodes EC (1985). A critical review of the literature on ratings of perceived exertion. **Sports Medicine**, 2 (3) : 198 - 222.

Clement DB and Sawchuk LL (1984). Iron status and sports performance. **Sports Medicine**, 1 (1) : 65 - 74.

Cockerill IM, Nevill AM and Lyons N (1991). Modelling mood states in athletic performance. **Journal of Sports Sciences**, 9 (2) : 205 - 212.

Comrades Association South Africa (1992). **Official results.** Published under the auspices of Unidata South Africa.

Costill DL (1972). Physiology of marathon running. **Journal of the American Medical Association**, 221 (9) : 1024 - 1029.

Costill DL (1974). Muscular exhaustion during distance running. **Physician and Sportsmedicine**, 2 (1) : 36 - 41.

Costill DL, Bowers R and Kammer WF (1970). Skinfold estimates of body fat among marathon runners. **Medicine and Science in Sport**, 2 (1) : 93 - 95.

Costill DL, Gollnick PD, Jansson E, Saltin B and Stein E (1973). Glycogen depletion in human muscle fibres during distance running. **Acta Physiologica Scandinavica**, 89 : 374 - 383.

Cox T (1985). The nature and measurement of stress. **Ergonomics**, 28 (8) : 1155 - 1163.

Daniels JT (1985). A physiologist's view of running economy. **Medicine and Science in Sports and Exercise**, 17 (3) : 332 - 338.

Davidson RJL, Robertson JD, Galea G and Maughan RJ (1987). Haematological changes associated with marathon running. **International Journal of Sports Medicine**, 8 (1) : 19 - 25.

Davies CTM and Few JD (1973). The effects of exercise on adrenocortical function. **Journal of Applied Physiology**, 35 : 887 - 891.

Dazord A, Saez J and Bertrand J (1972). Metabolic clearance rates and interconversion of cortisol and cortisone. **Journal of Clinical Endocrinology and Metabolism**, 35 (1) : 24 - 34.

Dearman J and Francis KT (1983). Plasma levels of catecholamines, cortisol and beta-endorphins in male athletes after running 26.2, 6 and 2 miles. **Journal of Sports Medicine**, 23 (1) : 30 - 38.

de Garay AL, Levine L and Carter JEL (1974). **Genetic and anthropological studies of Olympic athletes.** New York : Academic Press.

de Mello JJ, Cureton KJ, Boineau RE and Singh MM (1985). Effects of state of training and gender on ratings of perceived exertion at the lactate threshold. **Medicine and Science in Sports and Exercise**, 17 (2) : 198.

Dickinson JA, Cook SD and Leinhardt TM (1985). The measurement of shock waves following heel strike while running. **Journal of Biomechanics**, 18 : 415 - 422.

Durnin JVGA and Womersley J (1974). Body fat assessed from total body density and its estimate from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. **British Journal of Nutrition**, 32 : 77 - 97.

Ekblom B and Goldbarg AN (1971). The influence of training and other factors on the subjective ratings of perceived exertion. **Acta Physiologica Scandinavica**, 83 : 399 - 406.

Ellison K and Freischlag J (1975). Pain tolerance, arousal, and personality relationships of athletes and non-athletes. **Research Quarterly**, 46 (2) : 250 - 255.

Everly GS (1989). **A Clinical guide to the treatment of the Human Stress Response**. New York : Plenum Press.

Ewart O (1970). The attitudinal character of emotion. In MB Arnold (ED). **Feelings and Emotions**. New York : Academic Press.

Eysenck HJ (1958). A short questionnaire for the measurement of two dimensions of personality. **Journal of Applied Psychology**, 42 (1) : 14 - 17.

Eysenck HJ (1960). **The Structure of Human Personality**, 2nd edition. London : Methuen and Co.

Eysenck HJ (1967). **The Biological Basis of Personalty.** Springfield: CC Thomas.

* Eysenck HJ and Eysenck SBG (1963). **Manual for the Eysenck Personality Inventory.** San Diego : Educational and Industrial Testing Service (see Morgan and Costill,1972).

Falsetti HL, Burke ER, Feld RD, Frederick EC and Ratering C (1983). Haematological variations after endurance running with hard- and soft-soled running shoes. **The Physician and Sports Medicine**, 11 (8) : 118 - 127.

Ferguson GA (1981). **Statistical Analysis in Psychology and Education**, 5th edition. New York : McGraw-Hill.

Few JD (1974). Effect of exercise on the secretion and metabolism of cortisol in man. **Journal of Endocrinology**, 62 : 341 - 353.

Fitts RH (1977). The effects of exercise training on the development of fatigue. **Annals of the New York Academy of Sciences**, 301 : 424 - 430.

* Frumerie K (1913). Ueber das Verhaeltnis der Ermuedungsgefuehls zur CO_2 - Abgabe bei statischer Muskelarbeit. **Skandinawische Archive fuer Physiologie**, 30 : 409 (see Hockey,1983).

Ganong WF (1985). **Review of Medical Physiology.** Los Altos : Lange Medical Publishers.

Gardiner EN (1930). **Athletes of the Ancient World** Oxford : Clarendon Press.

Gower DB (1979). **Steroid Hormones.** London : Croom Helm Ltd.

Graham TE (1984). Mechanisms of blood lactate increase during exercise. **The Physiologist,** 27 (4) : 299 - 303.

Green HJ (1990). Manifestations and sites of neuromuscular fatigue. In : AW Taylor, PD Gollnick, HJ Green, CD Ianuzzo, EG Noble, G Metivier and JR Sutton (Eds). **Biochemistry of Exercise VII.**Champaign : Human Kinetics Books.

Grieve DW and Gear RJ (1966). The relationships between length of stride, step frequency, time of swing and speed of walking for children and adults. **Ergonomics,** 5 (9) : 379 - 399.

Gunderson HM, Parliman JA, Parker JA and Bell G (1983). Membrane permeability changes as a fatigue factor in marathon runners. In HG Knuttgen, JA Vogel and J Poortmans (Eds). **Biochemistry of Exercise.** Champaign : Human Kinetics Publishers Inc.

Guyton AC (1981). **Textbook of Medical Physiology.** Philadelphia: W.B. Saunders Company.

Hartley LH, Mason JW, Hogan RD, Jones LG, Kotchen TA, Mongey EH, Wherry FE, Pennington LL and Rickets PT (1972). Multiple hormonal responses to graded exercise in relation to physical training. **Journal of Applied Physiology**, 33 : 602.

Haus E, Lakatua DJ, Swoyer J and Sackett-Lundeen L (1983). Chronobiology in haematology and immunology. **The American Journal of Anatomy**, 168 : 467 - 517.

Haynes RC and Murad F (1975). Adrenocorticotropic hormone; adrenocortical steroids and their synthetic analogs; inhibitors of adrenocortical steroid biosynthesis. In A Goodman Gilman, LS Goodman and A Gilman (Eds). **The Pharmacological Basis of Therapeutics**, 6th edition. New York: MacMillan Publishing Co. Inc.

Heath BH and Carter JEL (1967). A modified somatotype method. **American Journal of Physical Anthropology**, 27 (1) : 57 - 74.

Hellman L, Nakada F, Curti J, Weitzman ED, Kream J, Roffwarg H, Ellman S, Fukushima DK and Gallagher TF (1970). Cortisol is secreted episodically by normal man. **Journal of Clinical Endocrinology**, 30 : 411 - 422.

Helminen HJ, Kiviranta I, Tammi M, Saamanen A-M, Paukkonen K and Jurvelin J (1987). **Joint loading - Biology and Health of Articular Structures.** Bristol : Wright.

* Hill AV (1925). The physiological basis of athletic records. **Lancet**, 2 : 481 - 486 (see Noakes,1988).

Hill DW, Cureton KJ and Collins MA (1989). Effect of time of day on perceived exertion at work rates above and below the ventilatory threshold. **Research Quarterly for Exercise and Sport**, 60 (2) : 127 - 133.

Hill DW, Hill JS, Grisham SC and Zauner CW (1987). Plasma volume response to exercise on five consecutive days. **Journal of Sports Medicine and Physical Fitness**, 27 (1) : 6 - 10.

* Hill AV, Long CNH and Lupton H (1924). Muscular exercise, lactic acid, and the supply and utilization of oxygen. Parts I - III. **Proceedings of the Royal Society of London**, 96 : 438 - 478 (see Mutch and Banister,1983).

Hockey R (1983). **Stress and fatigue in human performance.** Chichester : John Wiley and Sons.

Hogberg P (1952). Length of stride, stride frequency, 'flight' period and maximum distance between the feet during running with different speeds. **Arbeitsphysiologie**, 14 : 431 - 436.

Johnson LC (1982). Sleep deprivation and performance. In WB Webb (Ed). **Biological Rhythms, Sleep, and Performance.** Chichester : John Wiley & Sons Ltd.

Johnson LC, Naitoh P, Moses MJ and Lubin A (1977). Variations in sleep schedules. **Waking and Sleeping**, 1 (1) : 133 - 137.

Jones JG and Hardy L (1989). Stress and cognitive functioning in sport. **Journal of Sport Sciences**, 7 (1) : 41 - 63.

Kagaya H (1976). Cardiorespiratory responses to optimal speed of walking and to 'metabolic intersection' speed of walking and running. In F Landry and WAR Orban (Eds). **Exercise Physiology.** Quebec : Symposia Specialists.

Karadzic VT (1972). Physiological changes resulting from total sleep deprivation. In VT Karadzic (Ed). **Sleep : Physiology, Biochemistry, Psychology, Pharmacology, Clinical Implications. First European Congress on Sleep Research.** Basel : Karger.

Katz A and Sahlin K (1987). Effect of decreased oxygen availibility on NADH and lactate contents in human skeletal muscle during exercise. **Acta Physiologica Scandinavica**, 131 : 119 - 127.

Kavanagh T, Shephard RJ, Tuck JA and Qureshi S (1977). Depression following myocardial infarction : the effects of distance running. **Annals of the New York Academy of Sciences**, 301 : 1029 - 1038.

Keast D, Cameron K and Morton AR (1988). Exercise and the immune response. **Sports Medicine**, 5 (4) : 248 - 267.

Kerr JH (1985). The experience of arousal : a new basis for studying arousal effects in sport. **Journal of Sports Sciences**, 3 (3) : 169 - 179.

Keul J, Kohler G, von Glutz G, Luethi U, Berg A and Howald H (1981). Biochemical changes in a 100 km run : carbohydrates, lipids, and hormones in serum. **European Journal of Applied Physiology**, 47 : 181 - 189.

Kirkcaldy BD (1980). An analysis of the relationship between the psychophysiological variables connected to human performance and the personality variables extroversion and neuroticism. **International Journal of Sport Psychology**, 11 (4) : 276 - 289.

Kraemer WJ, Fleck SJ, Callister R, Shealy M, Dudley GA, Maresh CM, Marchitelli L, Cruthirds C, Murray T and Falkel JE (1989). Training responses of plasma beta-endorphin, adrenocorticotropin, and cortisol. **Medicine and Science in Sports and Exercise**, 21 (2) : 146 - 153.

Krebs PS, Scully BC and Zinkgraf SA (1983). The acute and prolonged effects of marathon running on 20 blood parameters. **The Physician and Sportsmedicine**, 11 (4) : 66 - 73.

Landers DM (1980). The arousal - performance relationship revisited. **Research Quarterly for Exercise and Sport**, 51 (1) : 77 - 90.

Larson LA (1974). **Fitness, Health, and Work Capacity**. New York : MacMillan Publishing Co., Inc.

Lewis SF, Taylor WF, Graham RM, Pettinger WA, Schutte JE and Blomqvist CG (1983). Cardiovascular responses to exercise as functions of absolute and relative work load. **Journal of Applied Physiology**, 54 (5) : 1314 - 1323.

Lijnen P, Hespel P, Fagard R, Lysens R, Van den Eynde E, Goris M, Goossens W, Lissens W and Amery A (1988). Indicators of cell breakdown in plasma of men during and after a marathon race. **International Journal of Sports Medicine**, 9 : 108 - 113.

Locke EA, Shaw KN, Saari LM and Latham GP (1981). Goal setting and task performance : 1969 - 1980. **Psychological Bulletin**, 90 (1) : 125 - 152.

Mackay CJ (1980). The measurement of mood and psychophysiological activity using self report techniques. In I Martin and PH Venables (Eds). **Techniques in Psychophysiology.** Chichester : John Wiley & Sons Ltd.

Maddi S (1968). **Personality Theories : a comparative analysis.** Chicago : Dorsey Press.

Markoff RA, Ryan P and Young T (1982). Endorphins and mood changes in long distance running. **Medicine and Science in Sports and Exercise**, 14 (1) : 11 - 15.

Maron MB, Horvarth SM and Wilkerson JE (1975). Acute blood biochemical alterations in response to marathon running. **European Journal of Applied Physiology**, 34 : 173.

Martin AD, Drinkwater DT and Clarys JP (1984). Human body surface area : validation of formulae based on a cadaver study. **Human Biology**, 56 (3) : 475 - 488.

McArdle WD, Katch FI and Katch VL (1986). **Exercise Physiology - Energy, Nutrition, and Human Performance.** Philadelphia : Lea & Febiger.

McCarthy DA and Dale MM (1988). The leucocytosis of exercise. **Sports Medicine**, 6 (6) : 333 - 363.

McKechnie JK, Leary WP and Noakes TD (1982). Metabolic responses to a 90 km running race. **South African Medical Journal**, 61 : 482 - 484.

McNair DM, Lorr M and Droppleman LF (1971). **Profile of Mood States Manual**. San Diego : Educational and Industrial Testing Service.

Menier DR and Pugh LGCE (1968). The relation of oxygen intake and velocity of walking and running, in competition walkers. **Journal of Physiology**, 197 : 717 - 721.

Mihevic PM (1981). Sensory cues for perceived exertion : a review. **Medicine and Science in Sports and Exercise**, 13 (3) : 150 - 163.

Mihevic PM (1983). Cardiovascular fitness and the psychophysics of perceived exertion. **Research Quarterly for Exercise and Sport**, 54 (3) : 239 - 246.

Miles DS, Sawka MN, Glaser RM and Petrofsky JS (1983). Plasma volume shifts during progressive arm and leg exercise. **Journal of Applied Physiology : Respiratory, Environmental and Exercise Physiology**, 54 (2) : 491- 495.

Miller BJ (1990). Haematological effects of running - a brief review. **Sports Medicine**, 9 (1) : 1 - 6.

* Miller JAA, Schultz AB, Warwick DN and Spencer DL (1986). Mechanical properties of lumbar spine motion segments under large loads. **Journal of Biomechanics**, 19 (1) : 79 - 84 (see Ayoub and Mital,1989).

Moore-Ede MC, Czeisler CA and Richardson GS (1983). Circadian time-keeping in health and disease. **New England Journal of Medicine**, 309 (8) : 469 - 476.

Moorthy AV and Zimmerman SW (1978). Human leucocyte response to an endurance race. **European Journal of Applied Physiology**, 38 : 271 - 276.

Morgan WP (1973). Psychological factors influencing perceived exertion. **Medicine and Science in Sports and Exercise** , 5 (2) : 97 - 103.

Morgan WP (1981). Psychophysiology of self awareness during vigorous physical activity. **Research Quarterly for Exercise and Sport**, 52 (3) : 385 - 427.

Morgan WP (1985). Psychogenic factors and exercise metabolism: a review. **Medicine and Science in Sports and Exercise**, 17 (3) : 309 - 316.

Morgan WP and Costill DL (1972). Psychological characteristics of the marathon runner. **Journal of Sports Medicine and Physical Fitness**, 12 (1) : 42 - 46.

Morgan WP, O'Connor PJ, Ellickson KA and Bradley PW (1988). Personality structure, mood states, and performance in elite male distance runners. **International Journal of Sport Psychology**, 19 (4) : 247 - 263.

Morgan WP and Pollock ML (1977). Psychological characterization of the elite distance runner. **Annals of the New York Academy of Sciences**, 301 : 382 - 403.

Morris RW and Jacobs LM (1950). On the application of somatotyping to the study of constitution in disease. **South African Journal of Clinical Sciences**, 1 (4) : 347 - 370.

Murray R (1987). The effects of consuming carbohydrate-electrolyte beverages on gastric emptying and fluid absorbtion during and following exercise. **Sports Medicine**, 4 (5) : 322 - 351.

Mutch BJC and Banister EW (1983). Ammonia metabolism in exercise and fatigue : a review. **Medicine and Science in Sports and Exercise**, 15 (1) : 41 - 50.

Myhre LG, Hartung GH, Nunneley SA and Tucker DM (1985). Plasma volume changes in middle-aged male and female subjects during marathon running. **Journal of Applied Physiology**, 59 (2) : 559 - 563.

Nieman DC, Berk LS, Simpson-Westerberg M, Arabatzis K, Youngberg S, Tan SA, Lee JW and Eby WC (1989). Effects of long-endurance running on immune system parameters and lymphocyte function in experienced marathoners. **International Journal of Sports Medicine**, 10 (5) : 317 - 323.

Noakes TD (1988). **Lore of Running.** Cape Town : Oxford University Press.

Noakes TD and Carter JW (1976). Biochemical parameters in athletes before and after having run 160 kilometers. **South African Medical Journal**, 50 : 1562 - 1566.

Noakes TD, Goodwin N, Rayner BL, Branken T and Taylor RKN (1985). Water intoxication : a possible complication during endurance exercise. **Medicine and Science in Sports and Exercise**, 17 (3) : 370 - 375.

Oatley K and Goodwin BC (1971). The explanation and investigation of biological rhythms. In WP Colquhoun (Ed). **Biological Rhythms and Human Performance.** London : Academic Press.

Osler T and Dodd E (1979). **Ultramarathoning - the next challenge.** Mountain View : World Publications Inc.

O'Toole ML and Douglas PS (1989). Introduction : the ultra-endurance triathlete : physiologic and medical considerations. **Medicine and Science in Sports and Exercise,** 21 (5) : S198 - S199.

* Owles WH (1930). Alterations in the lactic acid content of the blood as a result of light exercise and associated changes in the CO_2-combining power of the blood and in the alveolar CO_2 pressure. **Journal of Physiology**, 69 : 214 - 237 (see Åstrand and Rodahl,1977).

Oxendine JB (1970). Emotional arousal and motor performance. **Quest**, 13 : 23 - 32.

Pandolf KB (1978). Influence of local and central factors in dominating rated perceived exertion during physical work. **Perceptual and Motor Skills**, 46 (3) : 683 - 698.

Pandolf KB (1982). Differentiated ratings of perceived exertion during physical exercise. **Medicine and Science in Sports and Exercise**, 14 (5) : 397 - 405.

Pandolf KB, Cafarelli E, Noble BJ and Metz KF (1972). Perceptual responses during prolonged work. **Perceptual and Motor Skills**, 35 (3) : 975 - 985.

Pate RR, Blair SN, Durstine JL, Eddy DO, Hanson P, Painter P, Smith LK and Wolfe LA (1991). **Guidelines for Exercise Testing and Prescription.** Philadelphia : Lea & Febiger.

Peters EM and Bateman ED (1983). Respiratory tract infections: an epidemiological survey. **South African Medical Journal**, 64 : 582 - 584.

Pheasant S (1990). **Bodyspace, Anthropometry, Ergonomics and Design.** London : Taylor and Francis.

Pollock ML, Gettman LR, Jackson A, Ayres J, Ward A and Linnerud AC (1977). Body composition of elite class distance runners. **Annals of the New York Academy of Sciences**, 301 : 297 - 309.

Reinhardt WH, Staubli M and Straub PW (1983). Impaired red cell filterability with elimination of old red blood cells during a 100-km race. **Respiratory, Environmental and Exercise Physiology**, 54 (3) : 827 - 830.

Rejeski WJ (1981). The perception of exertion : a social psychophysiological integration. **Journal of Sport Psychology**, 3 (4) : 305 - 320.

Rejeski WJ (1985). Perceived exertion : an active or passive process ? **Journal of Sport Psychology**, 7 (4) : 371 - 378.

Ritschel WA (1986). **Handbook of Basic Pharmacokinetics.** Hamilton : Drug Intelligence Publications Inc.

Roberts GC and Duda JL (1984). Motivation in sport : the mediating role of perceived ability. **Journal of Sport Psychology**, 6 (3) : 312 - 324.

Robertson RJ (1982). Central signals of perceived exertion during dynamic exercise. **Medicine and Science in Sports and Exercise**, 14 (5) : 390 - 396.

Robertson JD, Maughan RJ and Davidson RJL (1988). Changes in red cell density and related indices in response to distance running. **European Journal of Applied Physiology and Occupational Physiology**, 57 : 264 - 269.

Roecker L, Kirsch KA, Heyduck B and Altenkirch H-U (1989). Influence of prolonged physical exercise on plasma volume, plasma proteins, electrolytes and fluid-regulating hormones. **International Journal of Sports Medicine**, 10 (4) : 270 - 274.

Roelfsema F (1987). The influence of light on circadian rhythms. **Experimentia**, 43 (1) : 7 - 13.

Scales WE, Vander AJ, Brown MB and Kluger MJ (1988). Human circadian rhythms in temperature, trace metals, and blood variables. **Journal of Applied Physiology**, 65 (4) : 1840 - 1846.

Schomer HH (1990). Mood states of two athletes during a world 24-hour relay record performance. **South African Journal for Research in Sport, Physical Education and Recreation,** 13 (1) : 59 - 68.

Scott PA (1990). **The Development of a Psychological Scale for evaluating self-perceived Strain,** unpublished PhD thesis : Stellenbosch University South Africa.

Selye H (1976). **The stress of life.** New York : McGraw - Hill Book Company.

Semple CG, Thomson JA and Beastall GH (1985). Endocrine responses to marathon running. **British Journal of Sports Medicine,** 19 (3) : 148 - 151.

Senay LC, Rogers G and Jooste P (1980). Changes in blood plasma during progressive treadmill and cycle exercise. **Journal of Applied Physiology : Respiratory, Environmental and Exercise Physiology,** 49 (1) : 59 - 65.

Sheldon WH, Lewis NDC and Tucker WB (1940). **The varieties of Human Physique.** New York : Harper Brothers.

Shephard RJ (1984). Sleep, biorhythms and human performance. **Sports Medicine,** 1 (1) : 11 - 37.

Simonson E (1971). **Physiology of Work Capacity and Fatigue.** Illinois : C.C. Thomas.

Szygula Z (1990). Erythrocytic system under the influence of physical exercise and training. **Sports Medicine,** 10 (3) : 181 - 197.

Tanaka K and Matsuura Y (1982). A multivariate analysis of the role of certain anthropometric and physiological attributes in distance running. **Annals of Human Biology,** 9 (5) : 473 - 482.

Tanner JM (1964). **The Physique of the Olympic Athlete.** London: George Allen and Unwin Ltd.

Tharion WJ, Strowman SR and Rauch TM (1988). Profile and changes in moods of ultramarathoners. **Journal of Sport and Exercise Psychology,** 10 (2) : 229 - 235.

Thomas TR, Zebas CJ, Bahrke MS, Aroujo J and Etheridge GL (1983). Physiological and psychological correlates of success in track and field athletes. **British Journal of Sports Medicine,** 17 (2) :102 - 109.

* Troup JDG, Reilly T, Eklund JAE and Leatt P (1985). Changes in stature with spinal loading and their relation to the perception of exertion or discomfort. **Stress Medicine,** 1 : 303 -307 (see Helminen et al.,1987).

Underwood B, Froming WJ and Moore BS (1980). Mood and personality : a search for the causal relationship. **Journal of Personality**, 48 (1) : 15 - 23.

van Beaumont W, Strand JC, Petrofsky JS, Hipskind SG and Greenleaf JE (1973). Changes in total plasma content of electrolytes and proteins with maximal exercise. **Journal of Applied Physiology**, 34 (1) : 102 - 106.

Viru A (1983). Exercise metabolism and endocrine function. In HG Knuttgen, JA Vogel and J Poortmans (Eds). **Biochemistry of Exercise**. Champaign : Human Kinetics Publishers Inc.

von Glutz G, Luethi U and Howald H (1978). Plasma growth hormone, aldosterone, cortisol and insulin changes in a 100-kilometer run. In F Landry and WAR Orban (Eds). **International Symposium on Biochemistry of Exercise**. Miami: Symposia Specialists International Inc.

Weitzman ED (1976). Circadian rhythms and episodic hormone secretion in man. **Annual Reviews of Medicine**, 27 : 225 - 243.

Wilks B (1991). Stress management for athletes. **Sports Medicine**, 11 (5) ; 289 - 299.

Wilson VE, Morley NC and Bird EI (1980). Mood profiles of marathon runners, joggers and non-exercisers. **Perceptual and Motor Skills**, 50 (1) : 117 - 118.

Winter DA (1978). Calculation and interpretation of mechanical energy of movement. **Exercise and Sports Sciences Review**, 6 : 183 - 201.

Withers RT, Craig NP and Norton KI (1986). Somatotypes of Australian male athletes. **Human Biology**, 58 (3) : 337 - 356.

Wyndham CH, Van der Walt WH, Van Rensburg AJ, Rogers GG and Strydom NB (1971). The influence of body weight on energy expenditure during walking on a road and on a treadmill. **Internationale Zeitschrift fuer Angewandte Physiologie**, 29 : 285 - 292.

* Yerkes RM and Dodson JD (1908). The relationship of strength of stimulus to rapidity of habit-formation. **Journal of Comparative Neurology and Psychology**, 18 : 459 - 482 (see Kerr,1985).

Zilva JF and Pannall PR (1978). **Clinical Chemistry in Diagnosis and Treatment**, second edition. London : Lloyd-Luke Ltd.

APPENDICES

Subject consent forms

Pre- and post-race data sheets

Heath-Carter Somatotype rating form

Eysenck's Personality Inventory questionnaire

Profile of Mood States questionnaire

RPE instructions sheet

Borg scale

RPE data sheet

APPENDIX 1

RHODES UNIVERSITY

DEPARTMENT OF HUMAN MOVEMENT STUDIES

SUBJECT CONSENT FORM

I, _____, having been fully informed of the nature of the research entitled : THE EFFECTS OF RUNNING 160 KM ON CERTAIN PHYSIOLOGICAL AND PSYCHOPHYSICAL PARAMETERS, do hereby give my consent to act as a subject in the above named research.

PROCEDURE, RISKS AND BENEFITS

There will be 2 testing sessions : one before the start of the Washie 160 km race, and the other within 15 min of completing the race.

During the first session you will be required to complete two psychological questionnaires: the shortened Eysenck Personality Inventory (EPI) and the Profile of Mood State (POMS) questionnaire. Thereafter the following body measurements will be made: height, weight, 5 skinfolds, femoral and humeral bi-epicondylar diameters and arm and calf circumferences. Then a 10 ml blood sample will be taken from a vein in your arm.

During the race your seconds will monitor and record perceived exertion (RPE) every two hours.

The second session again involves having a 10 ml blood sample taken, completing the Profile of Mood State (POMS) questionnaire and having stature and weight measured.

There are minimal risks involved in these procedures. We will have a qualified nursing sister taking the blood samples.

Feedback will be supplied to you as a subject as soon as it becomes available. You will be providing a valuable service to the advancement of our knowledge in ultra-endurance running.

I am fully aware of the procedures involved, as well as the potential risks and benefits attendant to my participation as explained to me verbally and in writing. In agreeing to participate in this research I waive any legal recourse against the researchers or Rhodes University. This waiver shall be binding upon my heirs and personal representatives. I realise that it is necessary for me to promptly report to the researcher any signs or symptoms indicating any abnormality or distress.

I am aware that I may withdraw my consent and withdraw from participation in the research at any time. I am aware that my anonymity will be protected at all times, and agree that the information collected may be used and published for statistical or scientific purposes.

I have read the foregoing and I understand it. Any questions which may have occurred to me have been answered to my satisfaction.

PERSON INVOLVED	PRINT NAME	SIGNATURE	DATE
SUBJECT			
ADMINISTRATOR OF INFORMED CONSENT			
WITNESS			
PROJECT SUPERVISOR			

Your participation as a subject is greatly appreciated.

Thank you for your time.

CARMEN OLTMANN

APPENDIX 2

PRE-RACE DATA SHEET

NAME: _____

TIME OF DAY: _____

CONSENT FORM SIGNED: _____

PSYCHOLOGICAL QUESTIONNAIRES COMPLETED:

EPI:

POMS:

ANTHROPOMETRY:

STATURE (cm) : _____

MASS (kg) : _____

SKINFOLDS (mm)

biceps : _____

triceps : _____

subscapular : _____

supra-iliac : _____

calf : _____

Bi-epicondylar diameters (cm)

humerus : _____

femur : _____

Circumferences (cm)

upper arm : _____

calf : _____

BLOOD SAMPLES

2 x 5 ml tubes : _____

2 x HCT tubes : _____ Hct :.....%

RPE forms handed out and explained

POST-RACE DATA SHEET

NAME: _____

TIME OF DAY:

BLOOD SAMPLES:

2 x 5 ml tubes :

2 x HCT tubes: Hct :.....%

POMS questionnaire

ANTHROPOMETRY:

Stature (cm) :

Mass (kg) :

RPE record sheet handed in :

Comments:

_____

_____

_____

_____

_____

_____

FINISHING TIME :.....

APPENDIX 3

HEATH-CARTER SOMATOTYPE RATING FORM

NAME .... AGE .... SEX: M F NO: ....

OCCUPATION .... ETHNIC GROUP .... DATE ....

PROJECT: .... MEASURED BY: ....

Skinfolds mm

Triceps =

Subcapular =

Supraliac =

TOTAL SKINFOLDS = [ ]

Calf =

TOTAL SKINFOLDS (mm)

Upper Limit	10.9	14.9	18.9	22.9	26.9	31.2	35.8	40.7	46.2	52.2	58.7	65.7	73.2	81.2	89.7	98.9	108.9	119.7	131.2	143.7	157.2	171.9	187.9	204.0
Mid-point	9.0	13.0	17 0	21.0	25.0	29.0	33.5	38.0	43.5	49.0	55.5	62.0	69.5	77.0	85.5	94.0	104.0	114.0	125.5	137.0	150.5	164.0	180.0	196.0
Lower Limit	7.0	11.0	15.0	19.0	23.0	27.0	31.3	35.9	40.8	46.3	52.3	58.8	65.8	73.3	81.3	89.8	99.0	109.0	119.8	131.3	143.8	157.3	172.0	188.0
FIRST COMPONENT	½	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	8½	9	9½	10	10½	11	11½	12

Height cm [ ]	1397	1435	1473	1511	154.9	158 8	1626	1664	1702	1740	1778	1816	1854	1892	193.0	195.9	2007	2045	2083	2121	2159	2197	2235	2273
Humerus width cm [ ]	5.19	5.34	5.49	5.64	5.78	5.93	6.07	6.22	6.37	5.51	6.65	6.80	6.95	7.09	7.24	7.38	7.53	7.67	7.82	7.97	8.11	8.25	8.40	8.55
Femur width cm [ ]	7.41	7.62	7.83	8.04	8.24	8.45	8.66	8.87	9.08	9.28	9.49	9.70	9.91	10.12	10.33	10.53	10.74	10.95	11.16	11.36	11.57	11.78	11.99	12.21
Biceps girth [ ] -T* [ ]	23.7	24.4	25.0	25.7	26.3	27.0	27.7	28.3	29.0	29.7	30.3	31.0	31.6	32.2	33.0	33.6	34.3	35.0	35.6	36.3	37.0	37.6	38.3	39 0
Calf girth [ ] -C* [ ]	27.7	28.5	29.3	30.1	30.8	31.6	32.4	33.2	33.9	34.7	35.5	36.3	37.1	37.8	38.6	39.4	40.2	41.0	41.7	42.5	43.3	44.1	44 9	45 6

SECOND COMPONENT	½	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	8½	9

Weight kg =

$Ht. / \sqrt[3]{Wt.}$ = [ ]

Upper limit	39.65	40.74	41.43	42.13	42 82	43.43	44.18	44 84	45.53	46 23	46.92	47.58	48 25	48 94	49 63	50 33	50.99	51 68
Mid-point	and	40.20	41.09	41.79	42.48	43.14	43.84	44.50	45.19	45 89	46 32	47.24	47.94	48 60	49 29	49 99	50 68	51.34
Lower limit	below	39.66	40.75	41.44	42.14	42.83	43.49	44.19	44.85	45.54	46.24	46.93	47.59	48 26	48 95	49 64	50 34	51 00
THIRD COMPONENT	½	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	8½	9

	FIRST COMPONENT	SECOND COMPONENT	THIRD COMPONENT	
Anthropometric Somatotype				BY: ....
Anthropometric plus Photoscopic Somatotype				RATER: ....

APPENDIX 4

EYSENCK'S TWO DIMENSIONS OF PERSONALITY

Name :.....

Questions	Yes	No
A. Do you sometimes feel happy, sometimes depressed, without any apparent reason?		
B. Do you prefer action to planning for action?		
C. Do you have frequent ups and downs in mood, either with or without apparent cause?		
D. Are you happiest when you get involved in some project that calls for rapid action?		
E. Are you inclined to be moody?		
F. Does your mind often wander while you are trying to concentrate?		
G. Do you usually take the initiative in making new friends?		
H. Are you inclined to be quick and sure in your actions?		
I. Are you freqently 'lost in thought' even when supposed to be taking part in a conversation?		
J. Would you rate yourself as a lively individual?		
K. Are you sometimes bubbling over with energy and sometimes very sluggish?		
L. Would you be very unhappy if you were prevented from making numerous social contacts?		

APPENDIX 5

T Score	FACTOR						T Score
	Ten	Dep	Ang	Vig	Fat	Con	
80+	35·6	47+	32+			28	80+
79	34	46				27	79
78		45	31				78
77	33	44	30		28	26	77
76	32	43	29	32			76
75		42			27	25	75
74	31	41	28	31			74
73	30	40	27	30	26	24	73
72	29	38·9	26		25	23	72
71		37		29			71
70	28	36	25	28	24	22	70
69	27	35	24		23		69
68		34	23	27		21	68
67	26	33			22		67
66	25	32	22	26	21	20	66
65	24	31	21	25			65
64		30	20		20	19	64
63	23	28·9		24	19	18	63
62	22	27	19	23			62
61		26	18		18	17	61
60	21	25	17	22	17		60
59	20	24		21		16	59
58	19	23	16		16		58
57		22	15	20	15	15	57
56	18	21	14				56
55	17	20		19	14	14	55
54	16	19	13	18	13		54
53		17·8	12			13	53
52	15	16	11	17	12	12	52
51	14	15	10	16	11		51
50		14				11	50
49	13	13	9	15	10		49
48	12	12	8	14	9	10	48
47	11	11	7				47
46		10		13	8	9	46
45	10	9	6		7		45
44		7·8	5	12		8	44
43	9	6		11	6	7	43
42	8	5	4				42
41	7	4	3	10	5	6	41
40	6	3	2	9	4		40
39		2	1			5	39
38	5	1		8	3		38
37	4	0	0	7	2	4	37
36							36
35	3			6	1	3	35
34	2				0		34
33	1			5		2	33
32				4		1	32
31	0						31
30				3		0	30

APPENDIX 6
Instructions for the use of RPE

RPE stands for "Ratings of Perceived Exertion".
We want you to try and estimate how hard you feel you are running at the time i.e. we want you to rate your perception of physical effort.

You will be asked to call out a number on the scale presented, which corresponds to your rating of perceived exertion. Try and assess your ratings as honestly and objectively as possible. Try to estimate your degree of exertion as accurately as possible.
For example : a rating of 6 would correspond with feelings of exertion while standing quietly, while a rating of 20 would reflect maximum exertion.

You will be asked to give three (3) specific ratings :

First rating : is associated with the sensation of strain in the exercising muscles, specifically the legs.

Second rating : involves the feeling of effort associated with the cardio-pulmonary response i.e. heart and lungs.

Third rating : requires an overall or general evaluation of the physical exertion experienced at the time.

RPE DATA SHEET

Name :.....

Time	RPE Ratings			Comments
	1st	2nd	3rd	
21h00				
23h00				
01h00				
03h00				
05h00				
07h00				
09h00				
11h00				
13h00				
15h00				
17h00				
19h00				
21h00				

RPE SCALE

6

7 Very, very light

8

9 Very light

10

11 Fairly light

12

13 Somewhat hard

14

15 Hard

16

17 Very hard

18

19 Very, very hard

20