

CHAPTER 4

RESULTS AND DISCUSSION

4.1 GENERAL RESULTS:

It has to be stated that the results obtained from the tests cannot be used to make conclusions about all marathon athletes because the sample is not big enough or random enough. Therefore it is a **case study** of five Comrades Marathon athletes who were training for the 1998 Comrades Marathon.

4.1.1 Physical characteristics of the athletes

The change in the anthropometric characteristics over a nine-month period of the 5 Comrades Marathon athletes is summarized in Table 1. The results indicate an increase in muscle percentage (2.45%), and a decrease in lean body mass (-3.37%), BMI (-2.49%), body mass (-2.53%) and fat percentage (-4.09%).

Below (Table 1) is a summary of the percentage changes that took place from October 1997 to May 1998. Individual values for each athlete are shown.

TABLE 1:Percentage change in the anthropometric parameters over nine-months

Athlete #	% change in				
	in mass	in %	in % fat	in BMI	lean body
		muscle			mass (LBM)
#1	0.07 %	2.99 %	7.10 %	0.00 %	-2.02 %
#2	-3.15 %	2.45 %	-13.84 %	-3.15 %	-1.12 %
#3	-4.07 %	2.60 %	-4.19 %	-4.22 %	-2.16 %
#4	0.57 %	1.07 %	8.24 %	0.19 %	-9.63 %
#5	-6.06 %	3.12 %	-17.78 %	-5.99 %	-1.93 %
Average	-2.53 %	2.45%	-4.09 %	-2.49 %	-3.37 %



4.1.2 Changes in anthropometric parameters over the 5 test sessions

Tests were done 5 times before the Comrades Marathon in October, January, March, April and May. Changes in total body mass (kg), fat percentage (%) and lean body mass (kg) were measured during the training months.

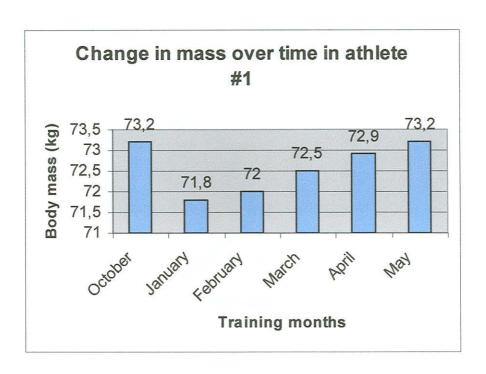
4.1.2.1 Total body mass

Several studies reported a high relationship between VO₂ max, body composition and endurance performance (Noakes, 1990; Brandon et al., 1995; Brisswalter et al., 1996). Berg et al. (1998) found a strong linear relationship between VO₂ max (L/min) and gross body mass for ectomorphs and mesomorphs, while these two variables are unrelated to endomorphs. VO₂ max (ml/kg/min) is dependent on gross body mass and increases with increasing body mass for ectomorphs and mesomorphs. The opposite is true of endomorphs in which group a strong linear decrease in VO₂ max expressed as ml/kg/min is observed with an increase in body mass.

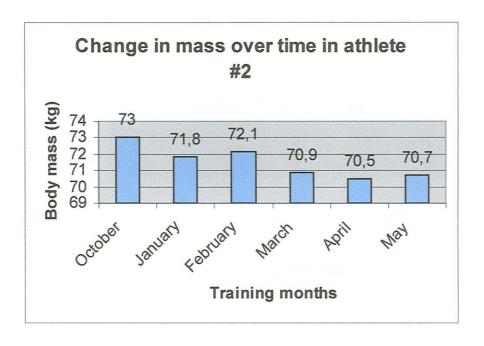
The VO₂ max (mlO₂/kg/min) is therefore considered to be a weight adjusted expression of VO₂ max where the effects of differences in body mass have been factored out. VO₂ max is usually reported as an absolute volume per minute (L/min) for sports such as rowing, in which total work output is important, and as a volume per minute relative to body weight (ml/kg/min) in activities such as running, in which the body weight is supported during the performance (McDougal et al., 1991).

It is estimated that the energy cost of running at 4 min/km for a 50 kg person is 54kJ/min, and for a 60 kg person 65 kJ/min (Burke, 1988). Costill & Fox (1969) estimated that one of the subjects in their study, who completed the Boston Marathon in a time of 2 h 22 min and weighed 63 kg, expended an average of 74 kJ/min, which would equate to an energy expenditure of about 10 579 kJ for the entire race. According to the regression equation formulated by Costill & Fox (1969), the caloric energy expenditure per kilogram of body mass is about 1.26 kJ/kg body mass. The above calculation gives a useful estimate of the energy consumption of marathon running and thus on weight loss during training.





Figures 14: Change in mass over time in athlete 1



Figures 15: Change in mass over time in athlete 2



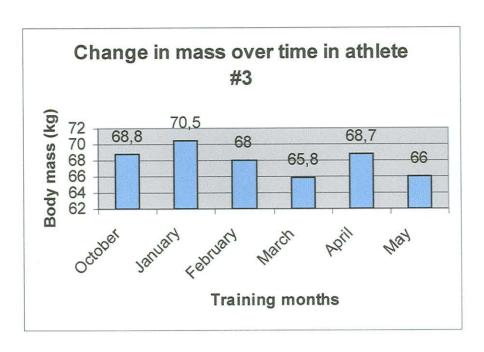


Figure 16: Change in mass over time in athlete 3

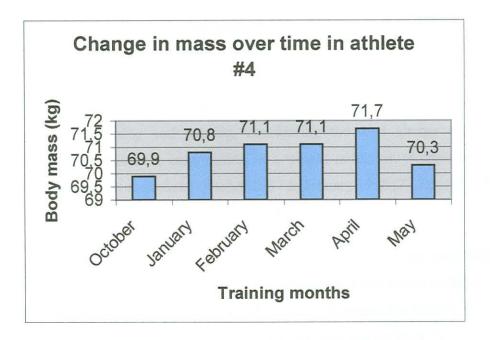


Figure 17: Change in mass over time in athlete 4



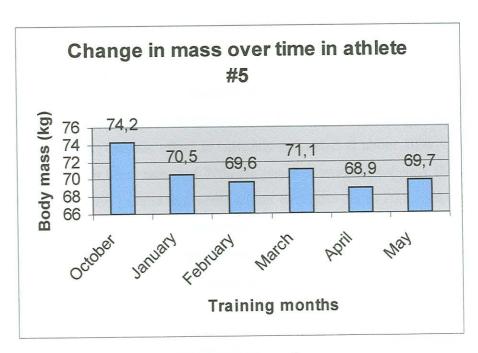


Figure 18: Change in mass over time in athlete 5

The change in mass in all the athletes are shown in Figure 14-18. The mean body mass (kg) of athletes 1 and 4 did not change much during the nine-month training period (0.07% and 0.57% respectively), while a significant decrease was shown in athletes 2, 3 and 5 (-3.15%, -4.07% and -6.06% respectively). Athletes 2 and 3 were also the athletes with the highest mileage in the nine-month period. Chad & Wenger. (1985) report that the duration of activity rather than the intensity has a greater effect on post-exercise oxygen consumption. The metabolic aftereffects of marathon running and training, considering the total energy expenditure of prolonged exercise, can make a contribution in a weight reduction programme. Athlete 1 showed a decrease in body mass from October 1997 to January 1998 (73.2kg to 71.8kg) as he was training intensively for the Iron Man in February 1998.

Athlete 3's genetic endowment also plays a role in his body composition, since athlete 3 is a black runner. When trained white males were compared to their black counterparts, Coetzer et al. (1993) found that the main anthropometric differences between the runners in their study were that the black runners were significantly shorter and lighter than the white middle-distance track athletes and had a considerably smaller muscle mass and lean thigh volume. Athlete 3 was slightly lighter than all the other athletes and had the lowest fat percentage. They also had smaller front thigh and medial calf skinfold thickness. The inertia of the limbs



would be less and thus, theoretically, less energy would be expended when moving the limbs (Bosch et al., 1990).

Questions concerning the relative contribution of natural endowment (genotype) to physiologic function and exercise performance (phenotype) have frequently been raised (McArdle et al., 1996). Genetic effect is currently estimated at about 10% – 30% for VO₂ max, 50% for maximum heart rate, and 70% for physical working capacity (McArdle et al., 1996). Coetzer et al. (1993) found that white milers were also about 20 kg heavier (72 vs. 52 kg) than the black distance runners. Furthermore, in a test of muscle function, Coetzer et al. (1993) show that, when undergoing repeated cycles of contraction and relaxation, the muscles of the black distance runners were able to complete more contraction / relaxation cycles before they developed marked fatigue.

Although a vigorous programme of physical training will enhance a person's level of fitness regardless of genetic background, it is clear that the limits for developing fitness capacity are linked to natural endowment (McArdle et al., 1996). Genetic makeup plays such a predominant role in determining the training response that it is almost impossible to predict a particular individual's response to a given training stimulus (McArdle et al., 1996). It is clear that genetic endowment plays an important role in the potential of an athlete. There is evidence that the muscles of white middle-distance runners do not have the same resistance to fatigue as do those of the black distance runners, despite their having the same proportion of T fibres. Black runners are therefore characterised by their extreme fatigue-resistance (Noakes, 1992).



4.1.2.2 Percentage body fat



Figure 19: Change in percentage fat over time in athlete 1

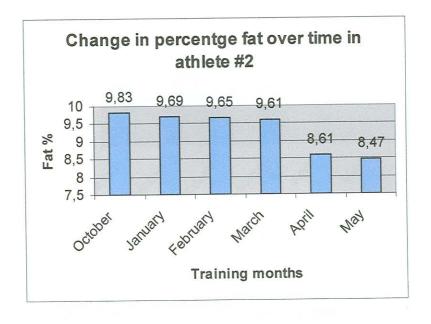


Figure 20: Change in percentage fat over time in athlete 2



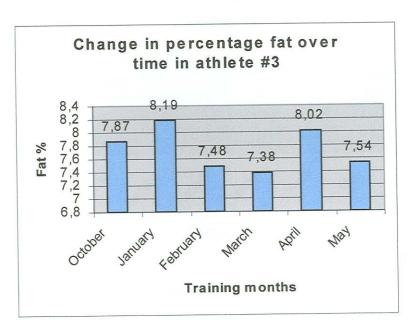


Figure 21: Change in percentage fat over time in athlete 3

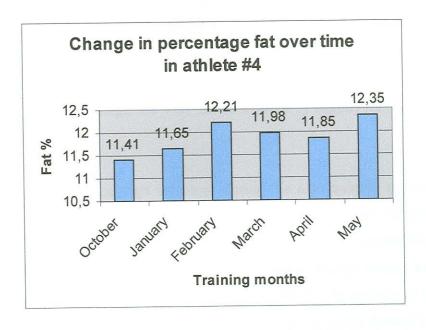


Figure 22: Change in percentage fat over time in athlete 4





Figure 23: Change in percentage fat over time in athlete 5

Individual changes in fat precentage are shown in Figures 19-23. Lower fat percentges were shown in athletes 2, 3, and 5 (-13.48%, -4.19%, and-17.78% respectively). The fat percentage of athletes 1 and 4 increased with 7.10% and 8.24% respectively. This finding correlates with the increase in both these athletes' total body mass. Since the energy cost of running is a function of body mass, one way of maximising performance is to reduce excess body fat. Previous studies indicate that lower fat-free body weight is one of the variables primarily characterising the faster endurance runners (Costill, 1967; Housh et al., 1988). With every one percent increase in fat percentage, the VO₂ max is reduced by slightly more than one percent (Londeree, 1986).

Although athlete 1's fat percentage increased during the nine training months it was still within the normal range for marathon athletes. He started with a very low fat percentage as he was training for the Iron Man. The results of Mutton et al. (1993) support the use of cross training as an alternative to increasing performance while adding variety to the training programme and possibly reducing the potential for injuries due to overuse or high intensity activity. In the study of Mutton et al. (1993), the cross training group showed improvements in running speed, % VO₂ utilised and a decrease in blood lactate values. The increase in athlete 4's fat percentage can be related to a decrease in training distance as a result of a hamstring injury.



4.1.2.3 Lean Body Mass (LBM)

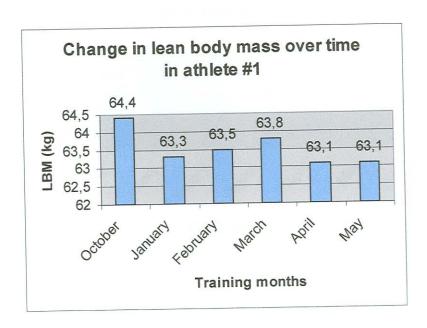


Figure 24: Change in lean body mass over time in athlete 1



Figure 25: Change in lean body mass over time in athlete 2



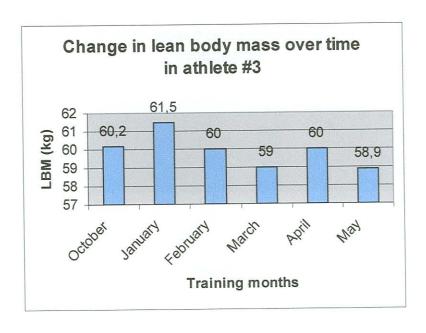


Figure 26: Change in lean body mass over time in athlete 3

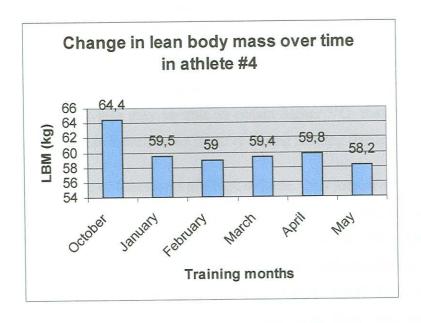


Figure 27: Change in lean body mass over time in athlete 4



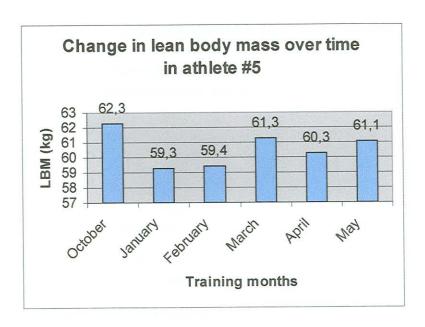
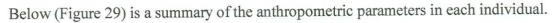


Figure 28: Change in lean body mass over time in athlete 5

All the athletes showed a decrease in lean body mass (Figures 24-28). Results indicate a decrease of 2.02%, 1.12%, 2.16%, 9.63% and 1.93% in athletes 1-5. Morgan et al. (1989) also observed a modest inverse relationship between body mass or weight and economy in èlite runners. However, greater body mass in the trunk area appears to be advantageous in terms of running economy. Conversely, those individuals who possess greater percentages of their body mass in the arms and legs may be able to obtain higher VO₂ max values because a greater proportion of their lean muscle mass is active during running (Bailey et al., 1991).

Athlete 2 trained for a period of four weeks at a high altitude. Only long-term exposure to high altitude produces a significant loss in lean body mass and body fat. The higher basal metabolic rate also plays a role in weight loss. Severe high altitude is thus catabolic. The decrease in muscle fibre size may be directly responsible for increasing capillary density and reducing effective diffusion distance to muscle mitochondria (Green et al., 1989). Thus the loss in lean body mass cannot be the reason for the athlete's lower lean body mass because of his relatively short stay at high altitude. It seems that more interval training and gymnasium work are necessary to build sufficient stamina endurance, which is an important parameter for Comrades athletes.





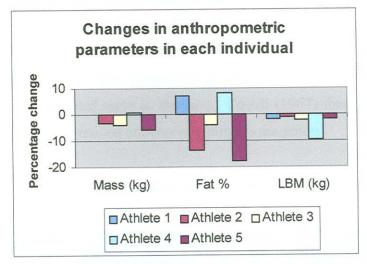


Figure 29: Summary of the changes in anthropometric parameters in each individual

4.2 RUNNING PERFORMANCE RESULTS

4.2.1 Physiological characteristics of the athletes

The results indicated statistically that some of the VO_2 max parameters changed during the time period of eight months. It has been found, however, that some of the maximum parameters did not change to a great extent ($VE/VO_2 = 0.01\%$, $VO_2/HR = 1.3\%$, VO_2 max = 3.54%, VO_2 absolute = 0.98%, RQ = 0.58%, VT = 4.31%, VE = 0.73%; speed and heartrate showed a decrease at the maximal exercise intensity; speed = -4.94%, heartrate = -4.37%). There was a greater improvement in parameters measured at threshold level ($VE/VO_2 = 1.45\%$, $VO_2/HR = 5.43\%$, VO_2 max = 5.73%, VO_2 absolute = 6.62%, RQ = 1.70%, VT = 4.19%, VE = 7.78%, speed = 9.10% and heartrate = 6.34%).

Table 2-11 represents the improvements in speed, heart rate, VE/VO₂, VO2/HR, VO₂, VO₂ absolute, RQ, VT, VE and RR at maximum and at lactate threshold intensity from October 1997 to May 1998.

4.2.1.1 Speed

Two important physiological variables are important in evaluating distance-running abilities. One is the velocity at anaerobic threshold – the pace at which blood lactate starts to rise substantially. Marathon pace is slightly slower than this. When an athlete starts a race too fast,



lactate will rise to very high levels and muscle glycogen stores will be depleted early in the race. The other important physiological variable is the velocity at VO₂ max, which is typically close to a 3000m race pace (Martin & Coe, 1997). The accumulation of an excessive amount of lactic acid in muscles under stress is a contributing factor to fatigue (Gupta et al., 1996). When an athlete starts a race too fast, lactate will accumulate to very high levels and muscle glycogen stores will be depleted early in the race. Costill (1967) found only a 2–3 times increase in lactate values after trained athletes completed a marathon.

TABLE 2: Percentage change in maximum speed versus change in speed at lactate threshold intensity

SPEED (km/hr)									
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change			
#1	20	18	-10%	18.4	17.5	-4.89%			
#2	20	17	-15%	18	17	-5.56%			
#3	18	19	5.56%	14.4	18	25%			
#4	19	18	-5.26%	14	16	14.29%			
#5	16	16	0%	12	14	16.67%			
Average	18.6	17.6	-4.94%	15.36	16.5	9.10%			

Athlete 1 presents a decrease in speed at both lactate threshold (-4.89%) and maximal intensity (-10%). However, his speed (17.5km/hr) at the lactate threshold in May before the Comrades Marathon was still very good for a marathon athlete. This correlates with the good Comrades Marathon time (6h12) which he achieved. Peaking for the Iron Man in February also played a role in his preparation for the Comrades Marathon. Top-level marathon runners who remain injuryfree with excellent general fitness can function well with a cycle that repeats every four to five months. There are 10 to 12 weeks of intense preparation and a few weeks of tapering; then the race, and a month of mental and physical recovery (Lenzi, 1987). This is why the world's healthiest and most consistent marathoners typically compete no more than two to three times a year.



Athlete 2 also shows a decrease in speed at both lactate threshold (-5.56%) and maximal intensity (-15%). He also did the highest training distances per week. Intense distance running has been shown to decrease muscular power (Houmard et al., 1994), however, muscular power is positively associated with distance running performance. Therefore, every effort to maximise power should be made.

Athlete's 3, 4 and 5 show a very positive improvement in speed at lactate threshold intensity (25%, 14.29% and 16.67% respectively). Thus, once VO2 max has been elevated as high as possible without inordinate additional training volumes, anaerobic development will make the additional difference between being optimally fit and marginally fit. Noakes et al. (1990) show that the physiological variables determining success at distances from 10-90km are the same; at least in marathon and ultramarathon specialists. This suggests that with appropriate training for longer distance events, the fastest 10km runners will also be the fastest marathon and ultramarathon runners. Billat et al. (1994) report that the subjects capable of sustaining the maximal aerobic speed for a longer period of time were also those who displayed a marked increase in lactate concentration at a later stage of a progressive exercise test and who run a 21.1 km race faster. Athletes 1, 2 and 3 showed a very good speed at the lactate threshold (17.5-, 17.0- and 18.0 km/hr) in May before the Comrades Marathon. This correlates with the Comrades Marathon times that they achieved: 6h12 for athlete 1, 6h06 for athlete 2 and 6h08 for athlete 3. Athletes 4 and 5 indicate lower threshold speeds (16- and 14 km/hr). Their times achieved (8h41 and 9h18 respectively) were also much lower than those of athletes 1, 2 and 3.

Noakes et al. (1990) noted a relationship between peak treadmill running velocity and running economy; those athletes who reached the highest treadmill running velocities were also the most economical. The positive correlation found between running economy and running times by Housh et al. (1988) further substantiates the fact that the faster runners were also more metabolically economical. Morgan & Craib (1992) state that athletes who specialise in shorter distance events have been shown to exhibit a better economy at faster speeds, whereas long-distance specialists tend to be more economical at slower running speeds.



According to Kindermann et al. (1979), exercise characterised by a lactate concentration of 4 mmol/L may be carried out for 45-60 min and occasionally longer. Studies done by Mognoni et al. (1990) and Stegmann et al. (1982) are not in agreement with this statement; their results are, however, probably the consequence of an overestimation of the threshold intensity. The exercise duration should nevertheless be kept in mind, since lactate increases with prolonged exercise. Thus the duration of the test is also a variable when determining lactate threshold. The athletes with improved speed ability also present a better fatigue resistance. For exercises lasting more than two hours (e.g. a marathon), it has been shown that the running economy decreased at the end of a long-distance run (Hausswirth et al., 1996). Thus fatigue affects economy in a negative way, increasing aerobic demand through the use of increasingly tired primary movers (i.e. arms and legs) as well as other physical efforts to help maintain pace.

Bosch et al. (1990) report on the difference between black and white runners. Black athletes run at 89% of VO₂ max; white runners at 81% of VO₂ max, when both groups were running at the same percentage of their best marathon race speed. Coetzer et al. (1993) report that the black runners also trained at a higher average exercise intensity than the white runners. Lower blood lactate concentrations were found in the black runners. The lower blood lactate concentration at any given running speed might have contributed to the superior fatigue resistance of the black athletes. These findings are reinforced by this study, since athlete 3, who is a black athlete, performed in accordance with these trends.

Anaerobic metabolism may also occur among the Comrades athletes at the start, or while running up hills, but the lactic acid concentration in the blood immediately after a standard marathon (Costill, 1970) and the Comrades Marathon (Jooste et al., 1981) is fairly low. These facts confirm the conviction that the Comrades Marathon is run mainly on aerobic energy. The pace of each athlete is thus limited largely by his lactic acid turning point, since running above this point causes muscular fatigue (Jooste et al., 1981).

The optimum training intensity for improving endurance performance remains established; on theoretical grounds it has been suggested to be the maximum intensity that can be maintained in a steady state. Jacobs (1987) suggests that a blood lactate concentration of 4 mmol/L represents the optimum intensity. Aerobic interval training is necessary to improve tolerance



to racing at the lactate threshold. This method of training is very effective for improving VO₂ at lactate threshold and/or economy of movement (Foss & Keteyian, 1998). Heck et al. (1985) suggest that èlite runners who were stagnated in their performance improved only after decreasing the intensity of their training to that associated with the aerobic anaerobic threshold. Weltman et al. (1992a) have demonstrated that at least some training above the lactate threshold is required for improvement. Yoshida et al. (1990) report that in distance runners and competitive walkers, blood lactate variables such as lactate threshold (VO₂ at threshold, and velocity at threshold) and OBLA (VO₂ at OBLA, velocity at OBLA) as well as running velocity were significantly improved after extra endurance training, but VO₂ max was not significantly improved.

4.2.1.2 Heart rate

TABLE 3: Percentage change in maximum heart rate versus change in heart rate at lactate threshold intensity

HEART RATE										
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change				
#1	176	176	0%	165	172	4.24%				
#2	185	177	-4.32%	170	177	4.12%				
#3	172	166	-3.49%	165	166	0.61%				
#4	195	180	-7.69%	161	169	4.97%				
#5	174	163	-6.32%	135	159	17.78%				
Average	180.4	172.4	-4.37%	159.2	168.6	6.34%				

Results indicated a decrease in the heart rate of all the athletes at maximal intensity and an increase in heart rate at lactate threshold intensity, except for athlete 1 who showed no difference at maximal intensity (see table 3). Thus, training prescriptions based on heart rate at designated metabolic markers with subsequent heart rate monitoring will enable coaches and athletes to monitor training intensity accurately. The ability, then, to sustain high heart rates during prolonged exercise could be hypothesised to be necessary for fast paces and fast



finishing times. According to this statement, athletes 1-5 improved threshold heart rate; an improvement which is very important for Comrades Marathon athletes.

Trained individuals have a much higher maximal cardiac output than untrained individuals -40 versus 25 L/min. Maximal cardiac output is limited by the maximal rate of depolarisation of the sino-artrial (SA) node and the structural limits of the ventricle. It is estimated that 70 -85% of the limitation in VO2 max is linked to maximal cardiac output (David et al., 2000). Athletes 3 and 5 indicate a very low maximum heart rate (166 and 163 beats per minute); whereas the Comrades Marathon star Nick Bester said after the 1997 Comrades that in the last few kilometers he was unable to keep his heart rate up at the remarkable 170 beats per minute he had sustained the whole race, "because my legs were too tired, I just couldn't move any faster" (Comrades Marathon update, 1997). Heart rates generally rise quickly to a steady state heart rate which is maintained early in exercise (i.e. up to approximately 30 minutes), followed by gradually increasing heart rates if exercise is continued for prolonged periods (Toole et al., 1998). Although specific reasons for slowing pace during prolonged exercise may vary among individual athletes, potential contributing reasons include substrate depletion, altered muscle efficiency, fluid and electrolyte imbalances, thermoregulatory problems, cardiac fatigue and psychological factors (Toole et al., 1998). The ability, then, to sustain high heart rates during prolonged exercise could be hypothesised to be necessary for fast paces and fast finishing times.

Athlete 2, who exercise for a month period at high altitude, showed a decrease in his maximal heart rate (185-177 beats per minute). Although some adaptations during acclimatisation to altitude should enhance aerobic capacity and endurance performance upon return to sea level, research results do not support this effect (Banister et al., 1978). This is probably the result of the altitude-related decrease in both maximum heartrate and stroke volume. For the highly trained athlete, the training intensity required for the maintenance of peak performances cannot be achieved at altitude.

Heck et al. (1985) suggest that èlite runners who stagnated in their performance improved only after decreasing the intensity of their training to that associated with the aerobic anaerobic threshold. Weltman et al. (1992) have demonstrated that at least some training above the



lactate threshold is required for improvement. However, Lehmann et al. (1992) suggest that athletes respond better to increases in training intensity than in training volume. If training at the maximal steady state was the "best" method to exercise, one would have hypothesised that the increased volume study would have been tolerated better than increases in the homeostasis disturbing training intensity (Snyder et al., 1994). However, if training intensity associated with maximal lactate steady state is not the best form to exercise in, it may be that this intensity simply represents the most time effective way of integrating training volume and intensity (Snyder et al., 1994).

4.2.1.3 Ventilatory equivalent (VE/VO₂)

TABLE 4: Percentage change in maximum VE/VO₂ versus change in VE/VO₂ at lactate threshold intensity

VE/VO_2									
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change			
#1	29.9	27.5	-8.03%	26.4	23.3	-11.74%			
#2	25.6	26	1.56%	26.3	25.6	-2.66%			
#3	27.4	27.2	-0.73%	23.9	27.2	13.81%			
#4	33.3	30.5	-8.41%	25.7	28.3	10.12%			
#5	28.3	32.7	15.55%	26.4	25.8	-2.27%			
Average	28.9	28.78	-0.01%	25.74	26.04	1.45%			

Only athlete 1 indicated a decrease in VE/VO₂ at both lactate threshold (-11.74%) and maximal intensity (-8.03%). Athletes 3 and 4 showed an increase in VE/VO₂ at lactate threshold intensity (13.81% and 10.12%). Athlete 5 showed an improvement only at the lactate threshold intensity (-2.27%). In healthy young adults, this ratio is usually maintained at approximately 25 L during submaximal exercise up to approximately 55% of the oxygen uptake. Thus, a decrease in VE/VO₂ at ventilatory threshold indicates a better oxygen extraction potential, and is therefore more advantageous for marathon athletes. Thus, although



athletes 3 and 4 indicate a increase in VE/VO_2 , al the athletes' (1-5) ventilatory equivalent are still below 30L, which is ideal for marathon runners (see table 4).

Dempsey (1986) states that the ability of the skeletal muscle to adapt to training is far greater than what is observed in the lung. Thus, the main significance of the training-induced increase in capillary density is not to accommodate blood flow but rather to maintain mean transit. This enhances oxygen delivery by maintaining oxygen extraction even at high rates of muscle blood flow.

The study by Ramsbottom et al. (1989) demonstrates a decrease in the ventilatory equivalent for oxygen with endurance training. This decrease represents a more efficient utilisation of oxygen and may reflect an increased mechanical efficiency of the running action, and hence on increased aerobic capacity of human skeletal muscle. Wasserman et al. (1986) suggest that lactate production during exercise depends mainly on the availability of oxygen in the active tissue. Therefore there is a decrease in blood lactate concentrations with endurance training.

Endurance trained athletes demand a lower V_E than do untrained athletes (Bailey et al., 1991). Lower ventilation, particularly over a prolonged effort (e.g. the Marathon), would mean, on a ratio basis, less oxygen to the respiratory muscles and more to the working skeletal muscles (Fox et al., 1993). Thus manipulation of the amount of ventilatory work necessary at a given running velocity could alter overall running economy (Bailey et al., 1991).



4.2.1.4 Oxygen pulse (VO₂/HR)

TABLE 5: Percentage change in maximum VO₂/HR versus change in VO₂/HR at lactate threshold intensity

VO ₂ /HR									
Athlete #	October max	May max	Maximum percentage change	October lactate threshold	May lactate threshold	Lactate threshold percentage change			
#1	25.5	27	5.88%	23.2	27.09	16.77%			
#2	23.5	24.8	5.53%	20.5	21.5	4.88%			
#3	27.7	28.4	2.53%	24.5	28.4	15.92%			
#4	22.5	24.3	8%	22.1	23.3	5.43%			
#5	25.9	21.9	-15.44%	24	20.2	-15.83%			
Average	25.02	25.28	1.3%	22.86	24.09	5.43%			

The VO₂ max is limited primarily by the rate of oxygen delivery, not by the ability of the muscles to take up oxygen from the blood. Therefore, the following factors could play a role in the limiting of VO₂ max: the pulmonary diffusing capacity, maximal cardiac output, the oxygen-carrying capacity of the blood, and skeletal muscle characteristics (David et al., 2000). Therefore, oxygen pulse plays a prominent role in the determination of aerobic endurance.

The predicted maximum oxygen pulse is the quotient of predicted maximum VO_2 and predicted maximum HR. In any given individual there is a close relationship between VO_2 and HR during exercise. The quotient of the VO_2 and HR is the oxygen pulse. The normal relationship of VO_2 to HR is linear over a wide range with a positive intercept on the HR axis. All the athletes showed an increase in the oxygen pulse at lactate threshold and maximal intensity, except for athlete 5. Athlete 5 indicates a lower maximum heartrate (174 – 163 beats per minute) and his training was not very high in terms of distance. The average improvement in lactate threshold is 5.43%, compared to 1.3% at maximal intensity (table 5).



The predicted O_2 pulse at any given VO_2 , including maximum VO_2 , is strongly dependent on the normal individual's body size, sex, age, degree of fitness, and hemoglobin concentration. The O_2 pulse can be considerably higher than predicted in the cardiovascularly fit person. All the athletes' (1-5) oxygen pulse is within the ideal range of 20-25 mlO₂/HR.

4.2.1.5 Oxygen consumption (VO₂)

TABLE 6: Percentage change in maximum VO₂ versus change in VO₂ at lactate threshold intensity

$ m VO_2$									
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change			
#1	61.5	65.3	6.18%	52.5	62.8	19.62%			
#2	58.5	62.2	6.32%	56.9	50	-12.13%			
#3	61.3	62	1.14%	58.8	62	5.44%			
#4	62.4	62.7	0.48%	51.6	56.4	9.30%			
#5	50.1	51.9	3.59%	43.8	46.6	6.39%			
Average	58.76	60.82	3.54%	52.72	55.56	5.73%			

A very positive improvement in the VO₂ is shown at lactate threshold (5.73%) and maximal intensity (3.54%) (table 6). Although some researchers claim that VO₂ max among èlite-level runners changes little over the course of a year, Martin & Coe (1997) found substantial differences as either training load or training emphasis shifts. Powers et al. (1983) demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO₂ max or running economy (Louanne et al., 1989; Schneider et al, 1991).

However, athlete 2 indicates a decrease (-12.13%) in VO_2 at lactate threshold intensity. Various reasons can be given for the decrease in VO_2 max. According to McArdle et al. (1996), maximal oxygen uptake will vary between 5% – 20%, depending on whether a person is "in shape" or "out of shape" at the time of measurement. Douglas et al. (1981) report that



distance runner Jim Rhyn's maximal aerobic capacity varied from 65-81 ml O₂/kg/min depending on his state of conditioning. Bouchard et al. (1992) state that there are high and low responders to training, and this factor is hereditary. This again demonstrates that VO₂ max is in fact a poor indicator of fitness, since one's ability to run both longer and faster will increase by more than 15% with training. For example, former mile world record holder, Jim Ryan, increased his VO₂ max from 65 ml O₂/kg/min in the partially trained state to 82 ml O₂/kg/min in the trained state, a huge 26% increase (Noakes et al., 1992). This once again emphasises the ability of the èlite athlete to show a greater adaptation to a training response. Most of the increase in VO₂ max is due to an increase in muscle contractility, which increases the capacity of the muscles to produce power.

Day to day variation could also play a role in the decrease or increase of VO₂ max; however, a number of studies have examined the problem of day-to-day variability in oxygen consumption during submaximal running. Variability across subjects ranges from 0.30 to 4.40% in Morgan et al. (1991) and 1.20 to 5.80% in Williams et al. (1991). Pereira et al. (1991) demonstrated that intra-individual variation in VO₂ during steady-state graded treadmill running is small.

Weltman (1990) reports that it is possible to increase velocity and VO₂ at OBLA without changing VO₂ max. Housh et al. (1988) reports that trained males exhibit lower submaximal steady-state VO₂ responses than untrained males. Therefore, while short-term endurance training may not be effective in modifying running economy, it is possible that prolonged training could result in improved biomechanical efficiency and therefore lower submaximal steady-state VO₂ values.

Endurance training has been shown to delay the onset of both the lactate and the ventilatory thresholds (Schneider et al., 1991; Hoffmann et al., 1993). Top marathon runners (i.e. sub 2h20 min) can sustain 86% of VO₂ max for the duration of a race (Hawley, 1995), whereas slower runners (i.e. 2 h 45 min up to 3 h) can sustain only 75% of their VO₂ max for the same distance (Farrel et al., 1979; Hawley, 1995). The optimum training intensity for improving endurance performance remains established; on theoretical grounds it has been suggested to be the maximum intensity that can be maintained in a steady state.



Martin & Coe (1997) question the benefit of more than 115 to 120 km a week at low intensity aerobic conditioning paces for distance runners seeking to improve their VO₂ max. The studies of Scrimgeour et al. (1986), which report that athletes training less than 60 km a week have as much as 19% less running economy than athletes training more than 100 km a week, might support this suggestion. This can explain athlete 5's lower lactate threshold as athlete 5's average training distances were lower than 60 km per week. Athletes 2 and 3 trained more than 140 km per week from January to May. However, both these athletes' VO₂ max increase slightly, but athlete 2's lactate threshold VO₂ max did not improved. Athlete 1 trained on average 100 km per week and peaked on 160 km the last two months before the Comrades. He indicated the greatest improvement (19.2%) at lactate threshold intensity (52.5 to 62.8 mlO₂/kg/min). Martin & Coe (1997) report that as athletes become better trained, not only does the VO₂ max rise, but so does the lactate/ventilatory threshold, both in absolute terms and as a percentage of VO₂ max. These long runs are not only important for endurance development but also for mental preparation for the Comrades.

Thus, endurance athletes have higher anaerobic thresholds than non-athletes, where the anaerobic threshold is expressed as a fraction of VO₂ max (Haffor et al., 1990). Ramsbottom et al. (1989) states that training status could be better explained in terms of the highest proportion of the VO₂ max at which a steady-state could be achieved rather than in terms of VO₂ max alone. Most of the athletes indicate a greater improvement in the fraction of VO₂ max at threshold intensity (5.73%) than in the change in VO₂ max (3.54%). Weltman et al. (1990) report that it is possible to increase velocity and VO₂ at OBLA without changing VO₂ max.

Yoshida et al. (1990) report that in distance runners and competitive walkers, blood lactate variables such as lactate threshold (VO₂ at threshold, and velocity at threshold) and OBLA (VO₂ at OBLA, and velocity at OBLA), as well as running velocity, were significantly improved after extra endurance training, but VO₂ max was not significantly improved.

Yoshida et al. (1992) name three factors that contribute to the improvement of VO₂ parameters after training, namely:

an improved capacity for mitochondrial respiration in muscle;



- an increased availability of blood and/or muscle O₂ stores; and
- an elevation of cardiac output and/or an increase in muscle blood flow.

In this context, Yoshida et al. (1992) document that endurance training induces an improvement of mitochondrial respiratory function, resulting in a reduced production of lactate during heavy exercise. According to Yoshida et al. (1992) the positive improvement in the VO₂ max (5.73%) and speed (16.67%) at lactate threshold intensity can be related to athletes' endurance training.

2.4.1.6 Respiratory quotient (RQ)

TABLE 7: Percentage change in maximum RQ versus change in RQ at lactate threshold intensity

	RQ								
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change			
#1	1.1	1.03	-6.36%	1.08	0.98	-9.26%			
#2	0.96	0.98	2.08%	0.95	0.97	2.11%			
#3	1.1	0.99	-10.00%	1.07	0.99	-7.48%			
#4	1.13	1.1	-2.65%	0.97	0.99	2.06%			
#5	1.07	1.22	14.02%	0.98	1.02	4.08%			
Average	1.072	1.064	-0.58%	1.01	0.99	-1.70%			

The application of the RQ is based on the assumption that the exchange of oxygen and carbon dioxide measured at the lungs reflects the actual gas exchange from nutrient catabolism in the cell (McArdle et al., 1996). This assumption is only valid during steady state or resting conditions (lactate threshold intensity). When other factors such as high intensity exercise or hyperventilation affect the RQ so that it no longer reflects only the substrate mixture in energy metabolism, it is described as respiratory exchange ratio (R).



A decrease in the RQ values (at lactate threshold intensity) in marathon athletes is more advantageous; thus the decrease in both lactate threshold (-1.7%) and maximal intensity (-0.58%) is an improvement. Athletes 1, 3 and 4 indicate a decrease in RQ at maximum intensity. As a result of high volumes of endurance training and less speed work it is possible to have a decrease in the lactate tolerance and therefore a decrease in RQ values. Their speed at lactate threshold had, however, improved; this is therefore an indication of an improvement in their aerobic system.

Only athletes 2 and 5 showed an increase in their RQ values at maximum intensity. During exhaustive exercise, R can rise significantly above 1,00. The lactic acid generated during anaerobic metabolism is buffered by sodium bicarbonate in the blood to maintain the acid-base balance. Because of the buffering effect, the CO₂ values rise to very high levels, above the quantity normally released during energy metabolism. Carbon dioxide elimination increases during hyperventilation, and as a result of that, the normal level of carbon dioxide in the blood is reduced. This elimination is not accompanied by a rise in the oxygen uptake; thus, the rise in the RQ does not represent the oxidation of food.

However, McArthur et al. (1983) found that higher muscle glycogen levels occurred after and higher RQ values during marathon races in the better runners. Hirokoba et al. (1992) found that endurancetrained men generate more CO₂ excess at the same blood lactate concentration when compared with non-endurancetrained and untrained men. There are two possible explanations for this:

- the increase in CO₂ excess per unit of body mass per lactate accumulation may be due to the decrease of buffering in the non-bicarbonate system; or
- the increase of buffering in the bicarbonate system.

These reasons can possibly explain the RQ increase in athletes 2 and 5.

4.3 BREATHING DYNAMICS

The primary task of the pulmonary system in accommodating the needs of an endurance athlete for either hard training or competition is to provide adequate gas exchange between alveoli and arterial blood with minimal work required by the lungs and chest. The ventilatory



system's efficiency for providing airflow is self-optimising (Martin & Coe, 1997). Expired ventilation (V_E) is the product of breathing rate (RR) and tidal volume (V_T) .

4.3.1 Tidal volume (VT)

TABLE 8: Percentage change in maximum VT versus change in VT at lactate threshold intensity

Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change
#1	2883	2903	0.69%	3204	3240	1.12%
#2	2138	2354	10.10%	1831	1980	8.14%
#3	1933	2165	12.00%	2073	2165	4.44%
#4	2885	2884	-0.03%	2844	2783	-2.14%
#5	2617	2585	-1.22%	2520	2757	9.405%
Average	2491.2	2578	4.31%	2494.4	2585	4.19%

During exercise that elicits VO_2 max, as much as 8% - 11% of the total oxygen uptake is required for respiratory muscle work. The respiratory muscles use approximately 40% - 60% of their maximum capacity to generate pressure at this exercise level (Aaron, 1992). Breathing can be optimized when the tidal volume is never more than 60% to 65% of the vital capacity, defined as the maximum amount of air that can be exhaled after a maximal inspiration (Martin & Coe, 1997). Breathing rate values recorded in highly trained athletes were no greater than 55 per minute (Martin & Coe, 1997).

Lower ventilation, particularly over a prolonged effort (e.g. the Comrades Marathon), would mean, on a ratio basis, less oxygen to the respiratory muscles and more to the working skeletal muscles (Fox et al., 1993). Thus, manipulation of the amount of ventilatory work necessary at a given running velocity could alter overall running economy (Bailey et al., 1991).

Tidal volume increased in both maximal (4.31%) and lactate threshold intensity (4.19%). The increase indicates a better percentage lungfilling with exercise at maximum and at lactate



threshold intensity. All the athletes' lungfilling were below 65% of vital capacity, thus the improvement in V_T does not affect the running economy negatively.

During long duration exercise at relatively low work intensities, such as between 50% to 60% of VO₂ max for about 2 h, a gradual but measurable rise in breathing rate (15% to 40%) does occur. This is accompanied by a reduction in tidal volume of about 10%-15%. The decrease in tidal volume does not exactly compensate for the increased frequency, because V_E increases as well. This drift is not observed during the short-duration runs (Martin & Coe, 1997) and is therefore not noticeable in the test results. Thus the drift will be observed during the Comrades Marathon.

4.3.2 Minute ventilation (VE)

TABLE 9: Percentage change in maximum VE versus change in VE at lactate threshold intensity

VE										
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change				
#1	134.5	131.6	-2.16%	101.4	106.9	5.42%				
#2	109.7	113.4	3.37%	91	89.8	-1.32%				
#3	114.4	111.4	-2.62%	96	111.4	16.04%				
#4	143.8	134.2	-6.68%	91.7	111.8	21.92%				
#5	105	117.3	11.71%	85.7	83	-3.15%				
Average	121.48	121.5	0.73%	93.16	100.58	7.78%				

Minute ventilation showed a small increase in maximal intensity (0.73%) and an increase of 7.78% at lactate threshold intensity. During strenuous exercise, the breathing rate increases between 35–45 breaths per minute, although rates as high as 60–70 have been measured in èlite athletes. In male endurance athletes, minute ventilation can increase to 160 L/min. Ventilation volumes of 200 L have been reported in research studies (McArdle et al., 1996). Even with such large V_E, tidal volumes for both trained and untrained individuals rarely exceed 60% of vital capacity. Endurance trained athletes demand a lower V_E than do



untrained athletes (Bailey et al., 1991). Athletes 1 and 4 have exercise induced asthma and they indicate the highest minute ventilation (134.5 and 143.8 L respectively). Their values did, however, improve during their training for Comrades. The other athletes indicate a relatively low V_E (below 120 L)

Wasserman et al. (1986) report V_E values of 15 liter per minute or 20 to 40% of the maximal voluntary ventilation in very fit individuals. A low breathing reserve is characteristic of patients with lung disease who are ventilatorily limited. CO_2 is a powerful ventilatory stimulant, and a small rise in the P_a CO_2 probably increases the V_E by 10%-30%. The level of V_E , with its removal of CO_2 , thereby serves as the major determinant of arterial H^+ ion concentration during this submaximal long-term work (i.e. at workloads ranging from a long training run to marathon or ultradistance racing).

The average rise in V_E (7.78%) at lactate threshold intensity found in this case study can be a result of the higher speed (9.10%) achieved by the athletes during the last test before OBLA was reached. These changes in volume and rate dynamics are controlled automatically to optimise mechanical efficiency while maintaining normal blood O_2 and CO_2 concentrations. Thus, it is unwise for coaches or athletes to attempt voluntary regulation of breathing patterns (Martin & Coe, 1997).



4.3.3 Respiration rate (RR)

TABLE 10: Percentage change in maximum RR versus change in RR at lactate threshold intensity

RR									
Athlete #	October max	May max	Maximum % change	October lactate threshold	May lactate threshold	Lactate threshold % change			
#1	47	45	-4.26%	32	33	3.13%			
#2	51	48	-5.88%	50	45	-10.00%			
#3	59	51	-13.56%	46	51	10.87%			
#4	50	47	-6.00%	32	40	25.00%			
#5	40	45	-12.50%	34	30	-11.76%			
Average	49.4	47	-3.445%	38	39.8	3.45%			

Excessively deep breaths, few in number, would be too energy costly. A large number of breaths, each small in volume, would not provide effective alveolar gas exchange. Breathing can be optimised with tidal volume never more than 60% to 65% of the vital capacity. Breathing rate values recorded in highly trained athletes were no greater than 55 per minute (Martin & Coe, 1997). None of the athletes reached a breathing rate of over 55 per minute except for athlete 3 during the first test. It is possible that he was feeling uncomfortable with the mask the first time. His respiration rate did, however, improve from 59 to 51 per minute. All the other athletes also indicated a decrease (-3.445%) in respiration rate at maximum intensity.

Only athletes 2 and 5 showed a decreased at lactate threshold intensity, while athletes 1, 3 and 4 indicated an increase. Carbon dioxide elimination increases during hyperventilation; for example, athlete 5 indicates a lower maximum heartrate (174 - 163 beats per minute), lower oxygen pulse and a higher RQ. The exercise induced asthma of athletes 2 and 5 also plays a role in their higher respiration rate.



Quite often, runners synchronise their breathing rate to their stride frequency. One practical implication of this breathing pattern is the usefulness of shortening stride and quickening cadence when climbing hills. The resulting increased breathing rate with increased stride frequency helps increase O₂ intake.

4.4 PERCENTAGE IMPROVEMENT IN THE VO₂ PARAMETERS AT LACTATE AND MAXIMAL INTENSITY

Figures 30 to 34 show the graph for each VO₂ parameter for each of the 5 athletes and it shows how the parameter has increased/decreased over time. On each graph the maximum as well as the lactate threshold is shown.

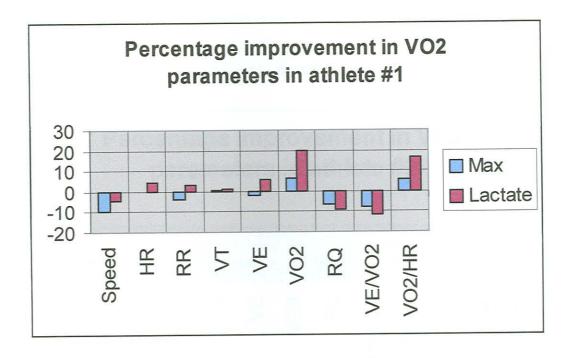


Figure 30: Percentage improvement in the VO₂ parameters in athlete 1



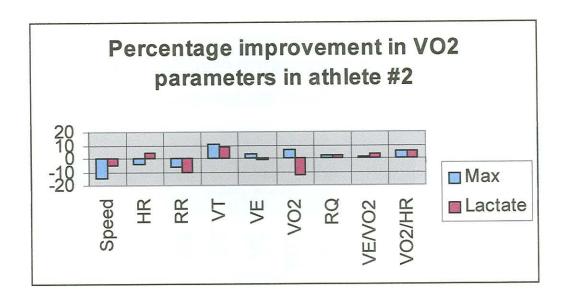


Figure 31: Percentage improvement in the VO₂ parameters in athlete 2

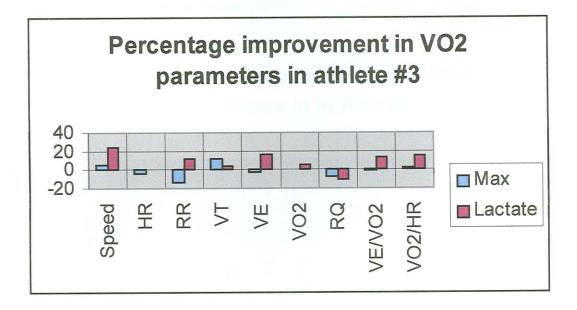


Figure 32: Percentage improvement in the VO₂ parameters in athlete 3



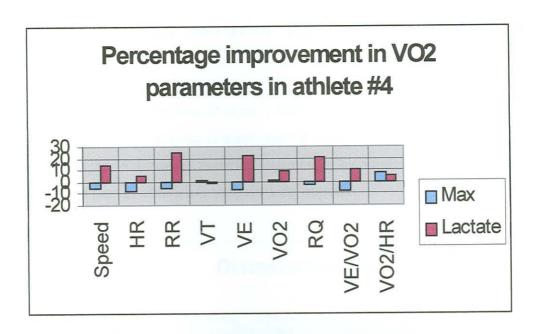


Figure 33: Percentage improvement in the VO2 parameters in athlete 4

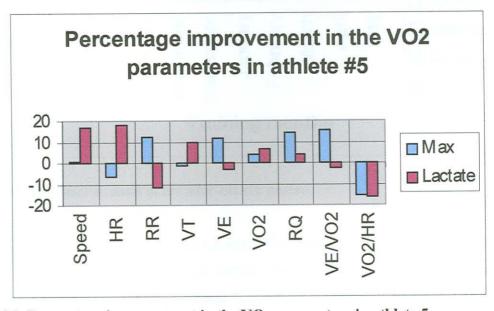


Figure 34: Percentage improvement in the VO₂ parameters in athlete 5



4.5 THE RELATIONSHIP BETWEEN THE DISTANCE TRAINED AND RUNNING TIMES

Martin & Coe (1997) question the benefit of more than 115 to 120 km a week at low intensity aerobic conditioning paces for distance runners seeking to improve their VO₂ max. Marathon runners are, however, special cases in that they require very high training volumes in order to stimulate greater fuel storage abilities in their working muscles.



Figure 35: Distance trained in the nine-month period

A positive correlation were found between the actual Comrades time (p<0.05) and the distance trained. Athlete 2 has done the highest trained distance (4463km), and completes the Comrades in the fastest time (6h06). Athletes 1, 3, 4 and 5 followed him, in that order (see figure 35)



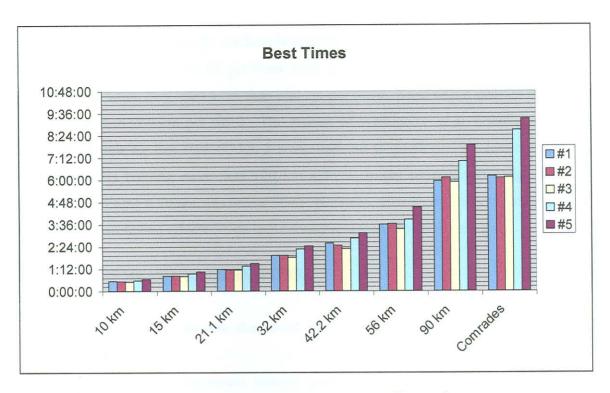


Figure 36: Best running times performed in the previous 12 months

In the case of club running for the Comrades, (i.e. someone who would run 100 to 120km per week for a period of 4 to 6 weeks in the build-up to the Comrades), the base training for this athlete should be at least 50 to 60km per week, incorporating a long run of at least 18 to 20km. Evidence from Hickson & Rosenkoetter (1981) suggests that training frequency rather than training intensity is responsible for the increase in mitochondrial enzyme concentration and endurance exercise capacity.

A marathon runner, mindful of the effect that very long runs have on the body and the recovery time required, may choose not to train beyond 35 to 38km in preparation for a hard marathon, although some of the èlite athletes do train over distances of up to 45km. Serious Comrades runners invariably train over distances of up to 75km, but usually have just one or two runs, five or six 60-70km runs, and several shorter (40-50km) runs. Even the serious Comrades runner would not train "over distance" (i.e. he she would not, on a given day, exceed the total Comrades distance). Most of the long runs are at a pace slightly slower than race pace, while the very long training runs (over 60km) would be run significantly slower than race pace (Brink, 1999).



Noakes (1992) states that èlite runners perform best in the marathon and ultramarathon races when they train between 120 to 200 km per week, with an increasing likelihood that they will perform indifferently when they train more than 200 km per week. None of the athletes in this study trained more than 200 km per week. The studies of Scrimgeour et al. (1986), which report that athletes training less than 60 km a week indicate as much as 19% less running economy than athletes training more than 100 km a week, might support this suggestion. Most of athlete 5's training distance was below 60 km per week, thus according to Scrimgeour et al. (1986), it is possible that athlete 5 was less economical than the other runners.

4.6 COMRADES HEART RATE RESPONSE

4.6.1 Relationship between lactate threshold heart rate and the actual Comrades heart rate response

By simultaneously measuring maximum aerobic power and exercise heart rate in the laboratory, the scientist can estimate the relative intensity of exercise in the field on the basis of heart rate alone and indirectly determine maximum aerobic power (MacDougall et al., 1991). The 4 mmol/L value for OBLA implies the maximum exercise intensity that a person can sustain for a prolonged period. In reality, this maximum stable lactate level is probably quite variable among individuals (Noakes 1988; Orok et al., 1989; Mognoni et al., 1990). The higher the running speed at which the lactate concentration exceeds the 4 mmol/L threshold, the higher the aerobic capacity. Spurway (1992) calls 2 mmol/L the aerobic and 4 mmol/L the anaerobic threshold.

The relationship between lactate threshold and the actual heart rate response indicates that none of the athletes could complete a 90-km race at the OBLA. It has been found that the athletes could keep their heart rate above a certain percentage of the lactate threshold only for the duration of the race (30.3% above 95% of the lactate threshold, 58.3% above 90% of the lactate threshold and 77.3% above 85% of the lactate threshold) (Figure 37-41).

Optimal times in marathon and similar events are achieved by performing at 97 to 100% of lactate threshold (Hagberg, 1984) while events of the 5000-10 000 m type require running speed nearer OBLA (Davis, 1985). Blood lactate does not accumulate to very high levels



during exercise that lasts more than an hour. A good example of this is during marathon running. At the end of a marathon, a trained athlete's blood lactic acid is only two to three times that found at rest (Costill et al., 1967). However, Mognoni et al. (1990) conclude that the effect of blood and muscle lactate on resistance to fatigue decreases rapidly with time. The anaerobic threshold is very often determined in order to obtain the corresponding heart rate value which is used to set the exercise intensity in endurance training so that there is no, or very little, lactic acid accumulation. Thus, the exercise duration should also be kept in mind as lactate increases with prolonged exercise.

Although mean lactate values representing a maximal steady-state during continuous exercise were found to be close to 4 mmol/L, individual values varied from 3 – 5.5 mmol/L (McLellan et al., 1992). Stegman et al. (1981) recognise the extent of this individual variation in maximal lactate steady-state values and introduce the concept of the individual anaerobic threshold (IAT).

There is good evidence that both the lactate VO_2 and the lactate VO_2 (absolute) have a positive correlation with the percentage of time that the heart rate was above 90% and 95% of lactate threshold (p<0.05). No significant evidence was found that the corresponding maximum VO_2 had any relation to the percentage of time that the heart rate was above a certain level of lactate threshold (p>0.05). Powers et al. (1983) demonstrated that the oxygen uptake measured at the ventilatory threshold was a better predictor of distance running success than either VO_2 max or running economy (Schneider et al., 1991; Louanne et al., 1989).

The following figures indicate the actual heart rate of the athletes over time in the race. The two horizontal lines indicate lactate threshold levels of 85% and 90%.



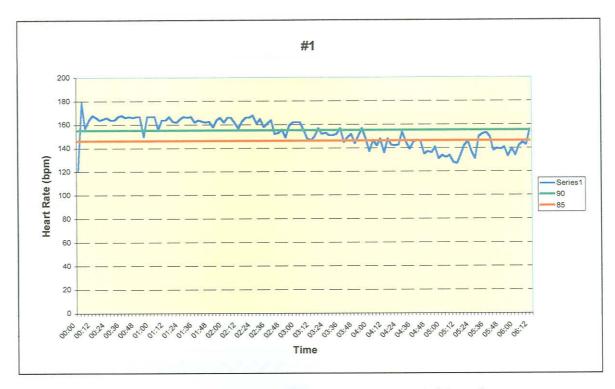


Figure 37: Heart rate response during the Comrades Marathon (athlete 1)

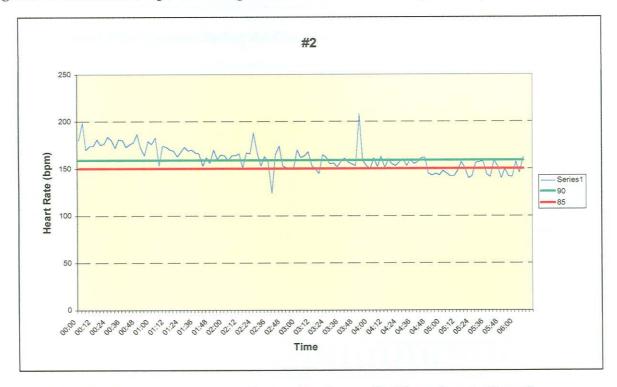


Figure 38: Heart rate response during the Comrades Marathon (athlete 2)



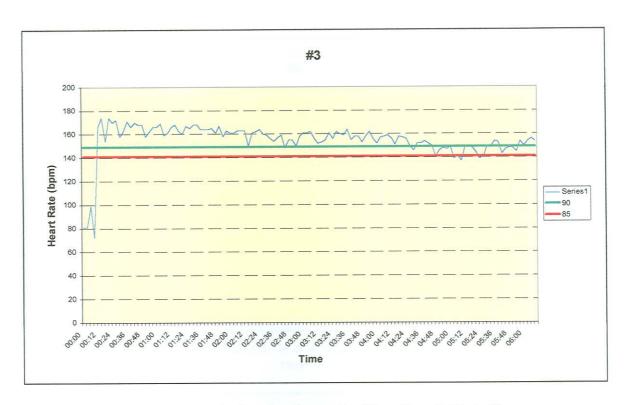


Figure 39: Heart rate response during the Comrades Marathon (athlete 3)

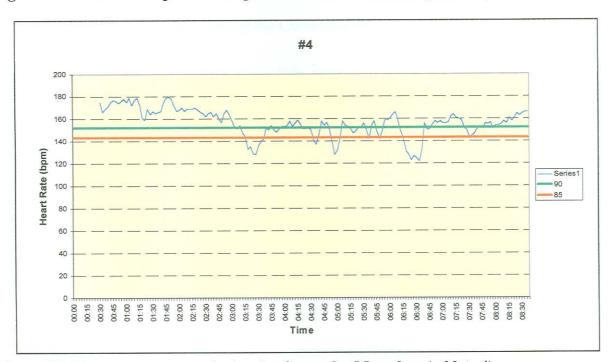


Figure 40: Heart rate response during the Comrades Marathon (athlete 4)



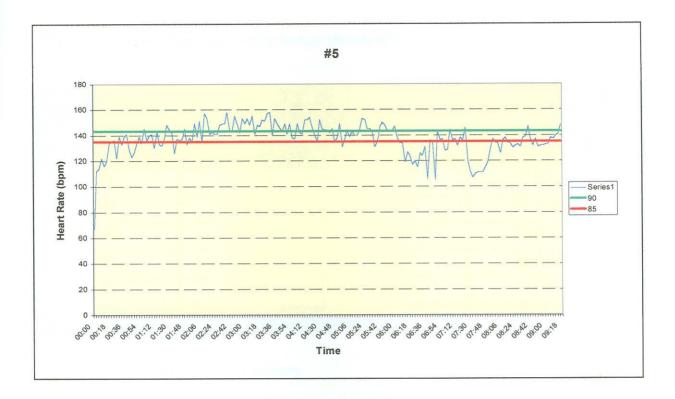


Figure 41: Heart rate response during the Comrades Marathon (athlete 5)

It has been found that the athletes could only keep their heart rate above certain lactate threshold levels for a percentage of the duration of the race. Below (Table 11 and Figure 42) is a summary of the five athletes' ability to keep their heart rate above the given level of lactate threshold.



TABLE 11: Percentage time above lactate threshold intensities of 95%, 90% and 85%

Athlete #	% time above a lactate threshold level of 95%	% time above a lactate threshold level of 90%	%time above a lactate threshold level of 85%
#1	28.8 %	68.8 %	69.6 %
#2	28.5 %	52.0 %	81.3 %
#3	49.6 %	79.2 %	92.0 %
#4	34.7 %	61.3 %	80.3 %
#5	10.2 %	29.9 %	63.1 %
Average	30.3 %	58.3 %	77.3 %

Figure 42 represents the above summary.

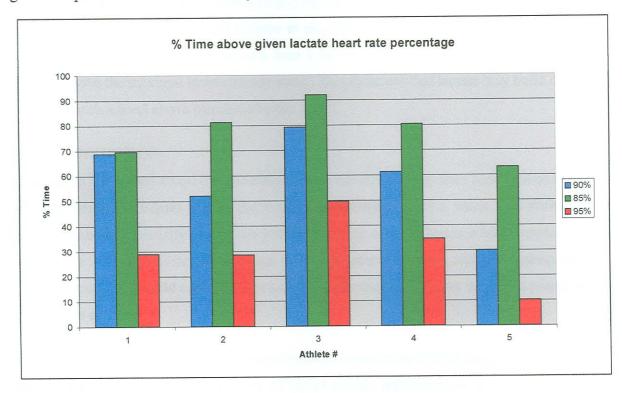


Figure 42: Percentage time above 95, 90 and 85% of lactate threshold heart rate



The relationships between the VO₂ parameters (both lactate and maximum), the actual Comrades time, distance trained and percentage time above a certain lactate threshold level (95%, 90%, 85%), have been tested using Spearman's rank correlation coefficient. It is interesting to see that there is substantial evidence that both the lactate VO₂ and the lactate VO₂ absolute parameters have a positive correlation with the percentage of time that the heart rate was above a certain level of lactate (90% and 95%), but that there was no significant evidence that the corresponding maximum parameters had any relation to the percentage of time that the heart rate was above a certain level of lactate (90% and 95%).

Anaerobic metabolism will probably occur among the Comrades athletes at the start, or while running up hills, but the lactic acid concentration in the blood immediately after a standard marathon (Costill, 1970) and the Comrades Marathon (Jooste et al., 1981) is fairly low. These facts emphasise the conviction that the Comrades Marathon is run mainly on aerobic energy. The pace of each athlete is thus limited largely by his lactic acid turning point and in such a way that this point is not exceeded (Jooste et al., 1981). Palmer et al. (1999) report that marathon runners finished their races in a time 3 to 7 min faster if they had been running at velocities above the maximal lactate steady-state. This indicates that lactate may build during the race to reach a level above threshold by the finish.

4.7 CONCLUSION

In conclusion it has been found that the athletes were not able to keep their heart rate up to just below the lactate threshold during the Comrades Marathon. As a result of deteriorated running economy, especially during the last 20km, none of the athletes could complete the race between 2-4 mmol/L lactate. It seems that more interval training and gymnasium work would be necessary to build enough of the stamina endurance which is an important parameter for Comrades athletes.

Endurance running performance has repeatedly been shown to be related more to submaximal effort measurements, such as the onset of blood lactate accumulation and the anaerobic threshold, than to VO₂ max (Maffulli et al., 1994). Thus, physiological parameters of importance are an improvement at lactate threshold intensity and not at maximum intensity because those parameters simulate the race intensity.



Although running is the world's largest participation sport, most runners have to train alone. Without the benefit of a coach, they have no one to make sure that they are using the most effective training methods; no one to show them how to achieve their maximum running potential. Sport Science can play a vital role in the success of ultra-marathons by helping the athletes to achieve their optimal fitness levels.

The results of the assessment become the basis for prescribing an optimal training programme that concentrates on identified areas of weakness. A testing programme provides feedback. Comparing the athletes' results on a given test item with those of their previous tests provides a basis for assessing the effectiveness of the intervening programme.