METABOLIC EFFECTS OF PROLONGED EXERCISE
IN SPINAL CORD INJURED WHEELCHAIR ATHLETES

BY

HELGA McKay

A Thesis
Submitted to the Faculty of Graduate Studies
in Partial Fulfilment of the Requirements
for the Degree of

MASTER OF SCIENCE

Department of Foods and Nutrition
University of Manitoba
Winnipeg, Manitoba

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ABSTRACT

Diminished sympathetic nervous system (SNS) innervation in spinal cord injured (SCI) quadriplegics (Q) may reduce the use of fat as an energy source during exercise, thereby accelerating carbohydrate depletion and onset of fatigue. As the level of spinal cord injury is lower in paraplegics (P), fuel use should be unaffected. The purpose of this study was to compare the metabolic responses of 4 SCI P and 4 SCI Q athletes during 2 hours of wheeling at a speed corresponding to 75% of their VO₂ max; as determined during a progressive maximal test. Subjects were nationally ranked track and road racers. Blood samples were taken pre-exercise, and at 30, 60 and 120 min for the determination of glucose, insulin, lactate, free fatty acids (FFA), and catecholamines. Results significant at p<0.05 are reported below. Respiratory exchange ratio (RER) decreased between 30 and 120 min in P and between 30 and 90 min in the Q. Norepinephrine was higher in P than Q at each blood draw. Norepinephrine and epinephrine increased between 60 and 120 min in P. Blood glucose and insulin decreased in the first 30 min of exercise in Q. Insulin was significantly higher in Q then in P prior to exercise. FFA profiles were similar in Q and P. In both groups, FFA increased in the last 60 minutes of exercise. These results suggest that initially there may be a greater dependence on carbohydrates during exercise in Q athletes. Therefore, in order to produce the greatest amount of improvement in performance in Q athletes, distinct diet and training methods need to be developed.
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CHAPTER I
INTRODUCTION

Wheelchair sports has grown dramatically over the past few years. Not only has the level of participation increased, wheelchair athletes have been integrated into able bodied sports. Training methods, equipment, and technique have all helped to improve the image of wheelchair sport and competition between wheelchair athletes. Despite these changes, there still exists a substantial gap between paraplegic (P) and quadriplegic (Q) athletic performance. This difference is due in part to the smaller amount of functional muscle in Q than P. Quadriplegics also have diminished sympathetic nervous system innervation, which decreases their maximum heart rate and maximum oxygen consumption, and may also alter their metabolic response to exercise.

Normally, during prolonged exercise, the sympathetic nervous system is stimulated resulting in an increased utilization of fat for fuel. If the sympathetic nervous system innervation is diminished, fat mobilization may not occur as efficiently, resulting in an increased dependence on carbohydrates for fuel. A spinal cord injured Q would have a lesion in the cervical area, therefore the entire sympathetic nervous system would be affected.

The purpose of this study was to examine the fuel use during prolonged submaximal exercise in spinal cord injured Q and compare this to spinal cord injured P.
CHAPTER II
LITERATURE REVIEW

INTRODUCTION

It is estimated that there are 750 to 1000 new spinal cord injuries (SCI) each year (personal communication, Canadian Paraplegic Association). Out of every 100,000 Canadians, 5 injure their spinal cord each year (1).

The spinal cord contains hundreds of nerves which travel from the brain through the cord and branch out at various levels of the cord to muscles. These nerves connect the brain to the muscles facilitating communication and coordination. An injury to the spinal cord breaks this connection and there is a loss of sensation and movement below the injury level. A paraplegic (P) has a SCI below the cervical area. This damage causes complete or partial paralysis of the lower limbs, because innervation of the muscles of the legs are in the lumbar area, below the level of lesion. Therefore, the higher the injury of the cord the greater the loss of sensation and movement because more nerves are affected. A quadriplegic (Q) has damaged the spinal cord in the cervical area, resulting in a loss of sensation of all or part of the four limbs. SCI affects both the central and autonomic nervous system. Since the sympathetic nervous system (SNS) branching occurs in the thoracic-lumbar area, the entire SNS would be affected in Q because it is below the level of lesion. This decreased innervation will affect such body functions as heart function.
(2-10), respiration (2-10) and, possibly, metabolism. The purpose of this literature review is to describe the effects of SCI on metabolism, particularly during exercise.

The importance of exercise for the able bodied (AB) has been well documented (11). Sports for SCI has also been shown to aid in health maintenance. Stotts (12) found that SCI nonathletes were hospitalized 3 times more frequently than SCI athletes. Aerobic training increases cardiovascular function (2). Exercise also increases the level of high density lipoprotein cholesterol (HDL-C) (13) which is associated with a decreased risk of heart disease (14), an important consideration since coronary heart disease is one of the major causes of death in SCI (15). For these reasons, exercise should be encouraged in all SCI in order to decrease the risk of certain diseases and maintain health.

Wheelchair sport has grown dramatically since the introduction of sport for SCI by Sir Ludwig Guttmann in 1948 (16). Up until 1975 the longest race in Track and Field for P and Q was the 200m and 60m respectively (16). Acceptance of wheelchair sport by the Sports Medicine community has been slow. This was due in part to the fact that doctors did not know how much physical stress could be tolerated by SCI athletes. Up until 1948, when antibodies were introduced, most SCI died within a year after their injury due to infection (17). Since few SCI lived for any length of time, little was known about their ability to participate in sports.
Today the 1500m race for men and the 800m race for women are demonstration Olympic events and may be incorporated into the Olympic program in the near future.

The major reason for the difference in athletic performance between P and Q wheelchair athletes, is the smaller amount of functional muscle mass in Q. The diminished SNS innervation in Q also acts to impair performance by decreasing maximum heart rate (2-10), VO₂max (2-10) stroke volume (10), cardiac output (10) and alters regulation of body temperature (18). These factors affect performance. Table 1 summarizes some of the physiological responses to maximal exercise of P, Q and upper body trained AB athletes.

Glycogen use may be a factor affecting performance in SCI Q athletes. Normally, during prolonged exercise a combination of fats and carbohydrates are used as fuel by exercising muscle. When fat is preferentially metabolized, glycogen depletion is delayed. The SNS is stimulated during exercise to mobilize fat from adipose tissue thereby decreasing the metabolism of carbohydrates (25). Quadriplegics may experience decreased lipolysis during exercise because of the decreased SNS innervation which may impair performance.

It has been established that fatigue in prolonged high intensity exercise is associated with glycogen depletion in AB (26-30) and paraplegic wheelchair athletes (31). Metabolism during exercise has not been investigated in Q and may be altered, thereby affecting their performance during competition and training.
TABLE 1. PHYSIOLOGICAL RESPONSES TO MAXIMAL EXERCISE IN ABLE BODIED, PARAPLEGICS AND QUADRIPLEGICS *

<table>
<thead>
<tr>
<th>SUBJECTS</th>
<th>TEST</th>
<th>AGE (yrs)</th>
<th>WEIGHT (kg)</th>
<th>mHR (bpm)</th>
<th>m(\dot{V}O_2) (L/min)</th>
<th>(\dot{V}E) (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABLE BODIED</td>
<td>AC</td>
<td>26</td>
<td>73.4</td>
<td>177</td>
<td>2.5</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td>(10,19,20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ABLE BODIED</td>
<td>WERG</td>
<td>—</td>
<td>—</td>
<td>164</td>
<td>3.7</td>
<td>152</td>
</tr>
<tr>
<td>ATHLETES</td>
<td>(6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PARAPLEGIC ATHLETES</td>
<td>AC</td>
<td>30</td>
<td>65.3</td>
<td>171</td>
<td>1.8</td>
<td>84</td>
</tr>
<tr>
<td></td>
<td>(10,19,20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WERG - Reg.</td>
<td>30</td>
<td>69.0</td>
<td>183</td>
<td>2.2</td>
<td>83</td>
</tr>
<tr>
<td></td>
<td>(3,6,21-23)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WERG - Race</td>
<td>26</td>
<td>76.6</td>
<td>189</td>
<td>2.7</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td>(5,9,24)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>QUADRIPLEGIC ATHLETES</td>
<td>AC</td>
<td>28</td>
<td>67.0</td>
<td>116</td>
<td>0.9</td>
<td>36</td>
</tr>
<tr>
<td></td>
<td>(9,10)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WERG - Reg.</td>
<td>30</td>
<td>68.7</td>
<td>113</td>
<td>1.0</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td>(2,3,5,6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>WERG - Race</td>
<td>33</td>
<td>—</td>
<td>119</td>
<td>1.2</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>(6)</td>
<td></td>
<td></td>
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* Only data from SCI were used where possible
AC - arm crank
WERG - Reg. wheelchair ergometer using regular chair
WERG - Race wheelchair ergometer using racing chair
FUEL USE DURING EXERCISE

There are three phases of fuel use during prolonged exercise (Figure 1) (32). Initially, muscle glycogen is used for fuel, with blood glucose and plasma free fatty acids (FFA) becoming more important as a fuel source after 5 to 10 min. After approximately 40 min, blood glucose contributes 75 to 90% of the total carbohydrates used by exercising muscle (33). The uptake of FFA by muscles is the major source of energy after one to four hours of exercise (34).

During exercise SNS activity increases and norepinephrine (NE) and epinephrine (E) are released (35,36) from the adrenal medulla. The sympathetic nerves also stimulate NE release directly (25). Epinephrine acts to stimulate glycogenolysis in the liver and muscle (37,38,). Norepinephrine and E also act to stimulate hormone sensitive lipase in adipose tissue which facilitates the catabolism of triglycerides (TG) resulting in increased plasma FFA (25) (Figure 2). During exercise, plasma insulin decreases (34,39) which also aids in increasing lipolysis of adipose tissue (40). Uptake of FFA by exercising muscles is regulated by the concentration of FFA in circulation. The increase in adipose tissue lipolysis, increases plasma FFA which results in increased muscle utilization (34,41). The greater the availability of plasma FFA, the less utilization of carbohydrates and consequently there is a sparing of glycogen (41). The SNS may be involved in stimulating glucagon secretion (42) but this does not
Figure 1. Fuel use during exercise (Adapted from Calles-Escandon, J. and Felig, P. Clin. Chest Med. 5:3-11. 1984.)
Figure 2: Sympathetic Nervous System Control of Energy Production During Exercise
appear to be a major determinant of lipolysis during exercise in humans (43). These factors help to maintain blood glucose levels by increasing the availability of FFA for fuel during exercise. Once glycogen stores are depleted fatigue occurs (26-31).

Training results in a number of metabolic adaptations which enhance performance. An endurance trained athlete has a lower rate of muscle glycogen utilization and a greater use of fat at the same relative workload, than an untrained person (44). This is due to an increase in:

a) the number of mitochondria in muscle cells (45)
b) the activity of oxidation enzymes in the mitochondria (46)
c) lipoprotein lipase activity (47)
d) muscle TG use (44,48)

Training also results in changes in hormone release and activity. Insulin (49), glucagon (39), E (39) and NE (39,49) levels are lower during exercise in trained than in untrained subjects. Tissue sensitivity to NE (49) and insulin (50) also increases with training (49).

AUTONOMIC NERVOUS SYSTEM RECEPTORS

The effector organs of the Autonomic Nervous System have either α or β receptors for E and NE (25). The β receptors can be further subdivided into β₁ or β₂ receptors. Adipose tissue contains mainly β₁ receptors (51). These
catecholamines bind with the $\beta$ receptors of adipose tissue where they activate adenylate cyclase to form cAMP. The secondary messenger, cAMP activates a series of reactions stimulating glucose and FFA mobilization (Figure 2). The FFA and glucose are then taken up by exercising muscle and used for fuel during exercise. When the $\beta_1$ receptors are blocked, FFA release is suppressed (52).

Norepinephrine and E are equally potent activators of $\beta_1$ receptors in adipose tissue (53). Since 80% of the adrenal medulla catecholamine secretion is E, it has a greater metabolic effect than NE in humans (25,54). The adrenal medulla has a major effect on metabolism. The effects of the hormones released from this gland lasts about 10 times longer than those released directly by the SNS because they are removed more slowly from the blood (25).

BETA BLOCKERS AND EXERCISE

Patients with angina, hypertension and certain cardiac arrhythmias are often treated with $\beta$-adrenergic blocking drugs. This medication inhibits the SNS by blocking the $\beta$ receptors. Thus, stress on the heart is decreased by decreasing the heart rate, cardiac output and blood pressure (55). Two types of $\beta$-blockers are available. Non-selective $\beta$-blockers, such as propranolol, nadolol and pindolol, which block both the $\beta_1$ and $\beta_2$ receptors. Selective $\beta$-blockers, such as metaprolol and atenolol, block only the $\beta_1$ receptors and
spare the $\beta_2$ receptors (55). It has been observed that exercise performance decreases in people taking the $\beta$-blocking drugs, particularly the non-selective $\beta$-blockers (52,56-58). This is caused by decreases in mobilization of FFA from adipose tissue (52,56-61), a decreased utilization of muscle TG (56) and an accelerated depletion of glycogen, leading to a decrease in blood glucose (57,59). The muscles must therefore rely on circulating glucose which is quickly depleted and leads to early exhaustion and/or hypoglycemia (52,56-61).

When healthy subjects are given 80 - 160 mg of a non-selective $\beta$-blocker for a short period of time, plasma FFA tend to increase more slowly during moderate exercise (52,56-58). In a recent study by Cleroux (56), the time to exhaustion was decreased by 33% after 1 week of 80 mg of nadolol in eight healthy subjects and endurance time on the bike decreased 14% while on 100 mg of atenolol. Lipolysis was inhibited while on both $\beta$-blocking treatments. Muscle TG utilization was significantly blocked while on the selective $\beta$-blocker and completely blocked while on the non-selective $\beta$-blocker nadolol. Plasma E was not significantly different between placebo and atenolol but the E increase was much greater with nadolol. This suggests that the adipose tissue lipolysis is partially controlled by the adrenergic system mainly through $\beta_1$ receptors, whereas muscle TG lipolysis is
controlled exclusively by the adrenergic system through $\beta_2$ receptors.

Hall (52) observed that a dose of 160 mg of propranolol was needed to produce a significant lower level of plasma FFA during the exercise. The exercise duration, however, was only 20 min which may have not been sufficient time to test the effect of propranolol on lipolysis because plasma FFAs normally become an important fuel source after an hour of exercise (62). When a similar drug trial was performed in hypertensive patients, $\beta$-blockade was shown to impair metabolic response in exercise by limiting FFA mobilization, thereby increasing the reliance of exercising muscle on glucose for fuel (59).

Lundborg (57) found exercise time to exhaustion was decreased significantly while receiving propranolol. After 2 days of propranolol treatment, a non-selective $\beta$-blocker, the running time to exhaustion was only 58% of that on placebo. After metaprolol, a selective $\beta$-blocker, treatment running time to exhaustion was 78% of that on placebo.

The effects of $\beta$-blockers on exercise performance has also been examined in animals. Juhlin-Dannfelt (60) treated rats with propranolol one hour before a prolonged exercise test of moderate to high intensity. Although plasma FFAs increased during the exercise in all groups, the plasma FFA concentration was lower in the group pretreated with propranolol. Glycogen depletion was 50% greater in the
propranolol group, showing a greater reliance on glycogen/glucose for fuel.

Nazar (61) examined sympathetic control of metabolism during exercise in dogs. Dogs were given propranolol 10 min before exercise which consisted of a moderate intensity run until exhaustion. Total work capacity was 48% less after propranolol treatment than in the control animals. After propranolol injections, plasma FFA levels were lower, hypoglycemia occurred sooner, and the rate of glycogen depletion was greater during exercise in the propranolol treated animals compared to the controls. The respiratory exchange ratio (RER) also increased during the exercise, indicating an increase in carbohydrate metabolism during exercise. In the control test, RER decreased during the exercise, which was the normal metabolic response to prolonged exercise. This study supports the theory that SNS stimulation is needed to increase fat metabolism and spare glycogen during exercise in order to delay fatigue.

RELATION TO SPINAL CORD INJURED QUADRIPLEGICS

There is a difference between the responses to SCI and the AB receiving β-blocking drugs. The response in SCI is effected at the SNS stimulation level, rather than at the receptor. When AB are given β-blocking drugs, the SNS will still be stimulated during exercise and E and NE are released (56). However, the catecholamines are not able to bind with
the β receptors because the receptors are blocked, therefore, the normal response to catecholamines is negated. In the SCI Q, the SNS is intact, however, because of spinal cord damage, innervation to the adrenal medulla is diminished. The SNS may not be stimulated during exercise in complete SCI Q, and release of E and NE may be inhibited.

Regardless of whether the block is at the point of release or the point of binding, TG lipase activity is not stimulated and carbohydrate metabolism is increased.

**EVIDENCE FOR METABOLIC CHANGES IN SPINAL CORD INJURED PEOPLE**

Several studies have demonstrated alterations in metabolism following a SCI. These include changes in blood glucose tolerance (13,63-65), urinary and plasma catecholamine concentrations (13,65-70) and low HDL-C concentrations (72,73).

Blood glucose levels in the SCI have been reported to be greater than that observed in the normal population. From retrospective chart analyses, fasting blood glucose levels were found to be above 130 mg/dl in 23% of a group of 52 SCI patients (63). These values were similar to those observed in diabetics (40). In normal AB subjects, fasting blood glucose levels are 90 mg/dl (40). In an earlier study by Duckworth (64), 41 SCI volunteers were given oral glucose tolerance tests. After receiving 100 g of glucose, 23 had blood glucose levels exceeding 200 mg/dl 2 hours after the glucose load and
remained higher than the control group after 4 hours. Eighteen volunteers had significantly lower values than the 23 glucose intolerant SCI, but significantly higher values than the control AB. The 23 glucose intolerant SCI were further subdivided into insulin sensitive and insulin resistant group based on their peripheral insulin activity.

In contrast, Claus-Walker (65) found that fasting blood glucose levels in chronic Q subjects were 79 mg/dl, slightly lower than normal. Following an oral glucose tolerance test blood levels were essentially normal in these subjects, but there was a significantly lower glycemia at 30 min compared to the P and AB groups. A recent study by Dearwater (73) also found fasting blood glucose levels to be in the low normal range for sedentary SCI subjects (82.8 mg/dl) and SCI athletes (79.6 mg/dl). Fasting insulin concentrations were not found to be significantly different between the SCI and the AB groups. Differences between the groups were thought to be due to neuronal control, because both the sedentary and athletic SCI subjects appear to be a homogeneous population. It was also suggested that SCI may have increased insulin sensitivity because fasting blood glucose levels were significantly lower than the AB controls. Dearwater used both P and Q SCI subjects which may explain why these results differ from those in the previous studies reported by Duckworth.

Brown (66) also found fasting blood glucose levels to be slightly low (86 mg/dl) in SCI Q. When these subjects were
given 1 g/kg glucose their glucose tolerance curve reached a higher value and was maintained at a higher level longer in the SCI P than AB. The intravenous glucose tolerance curve and response to injected insulin, were similar for both types of SCI subjects.

Palmer (67) found the SCI Q required 0.20 U/kg of insulin to induce a similar response to controls receiving 0.15 U/kg. Even with the larger dose of insulin the glucose values fell more slowly in Q subjects and reached the nadir later than the controls.

The results are conflicting, however, there appears to be a general agreement that there is an increased incidence of low normal fasting blood glucose levels and insulin resistance in SCI Q subjects. Although a limited number of studies have been done in this area, when P and Q are separated, the evidence supporting a lower fasting blood glucose and insulin resistance is much more convincing. The main point, however, is that there is a difference in metabolism following a SCI, particularly in Q persons. These changes could result in negative effects during exercise when the control of available fuel sources is important. Another difference in metabolism between SCI and AB subjects is related to the amount of HDL-C. Several studies have shown that SCI persons have HDL-C concentrations below the normal sedentary population level (13,71,72). Most researchers have attributed this to inactivity.
In general, physical activity was assessed using a large scale integrated (LSI) activity monitor (73). The LSI activity monitor is an instrument which measures body movement. The instruments were worn on the subjects dominant wrist and ankle for 2 weekdays. The SCI sedentary patients were 30 times less active than college freshman and 8 times less active than sedentary middle aged men. Based on training programs alone, the SCI athletes were found to be significantly more active than the SCI sedentary group. The athletes also had higher HDL-C concentrations (13). Although the LSI activity monitor is able to distinguish between small activity differences, the comparisons on the amount of activity must be viewed with caution since the SCI subjects are limited to upper body activity and therefore may appear to be less active.

Although activity was higher in the SCI athletes, and HDL-C concentrations were higher than in the sedentary SCI, HDL-C concentrations were still slightly but not significantly lower than in the sedentary AB population. The higher total HDL-C concentrations in the sedentary AB population was mainly due to the amount of the HDL$_3$ subfraction, which is reported to have less association to coronary heart disease than the HDL$_2$ subfraction (14). The difference between the two SCI groups is due to the amount of the HDL$_2$ subfraction, indicating a positive effect of exercise in SCI athletes (74). Again, there are metabolic similarities between AB subjects
receiving β-blocking drugs and SCI subjects. Hypertensive patients receiving 100 mg of atenolol had significantly lower HDL-C concentrations and higher TG concentrations than before the treatment, again showing similarities between SCI and those receiving β-blocking drugs. This change was attributed to inhibition of the lipoprotein lipase enzyme (74).

Insulin action is thought to be a factor in HDL-C metabolism. Insulin is the determinant of lipoprotein lipase enzyme activity (75). Therefore, the less efficient insulin is in controlling blood glucose, the less efficient it is in stimulating lipoprotein lipase (LPL), with a consequent decreased LPL activity, higher plasma TG and lower HDL-C concentrations (75). Again, these studies support the hypothesis of a change in metabolism in SCI which may affect athletic performance.

Plasma and urinary catecholamines are also altered after SCI. Normally, resting plasma and urinary E and NE are low in SCI Q subjects compared to AB controls (65,67-69). When a normal healthy male is given an intravenous insulin tolerance test, plasma NE increases in the response to the insulin induced hypoglycemia. In the SCI Q plasma NE does not change (66).

In Q subjects, during bladder percussion or muscle excitation, by electrical stimulation, plasma NE increases to normal levels and plasma E increases slightly (68). This leads to an increase in blood pressure and a decrease in heart
rate. The decrease in heart rate is due to the activity of the cranial portion of the parasympathetic nervous system (18) which is still intact in Q. The greater increase in NE would suggest that bladder and muscle stimulation causes an increase in SNS activity rather than adrenal secretion. These reactions are due to autonomic dysreflexia where the responses are the result of spinal sympathetic reflexes. The effects during autonomic dysreflexia are considered to be reflex in nature because of the short time lapse between stimulus and response (18).

Mathias (70) had patients with complete cervical SCI transferred to an electrical tilting bed. The bed was tilted smoothly at a rate of 3°/s from horizontal to 45° head up, remained there for 30 min and was then returned to the horizontal. Plasma catecholamines and blood pressure were measured before tilting, at 10, 20 and 30 min intervals during tilting and again at the end of the procedure. NE increased 14% in SCI subjects as compared to 115% in the AB controls. In our own lab findings, this increase in catecholamines is at the lowest end for detection. Blood pressure dropped during tilting, an expected response when NE increase is very small.

Claus-Walker (65) found no increases in urinary NE, E or methylhydroxymandelic acid (a catecholamine metabolite) during tilting in SCI Q, suggesting that the body is not responding normally to a stimuli. The result of this is often a drop in blood pressure (65,70) and an increase in heart rate (65) as
was seen in the previous study. In AB subjects under similar conditions there is an increase in NE and E which aids in the maintenance of blood pressure (70).

These examples show that the metabolism of glucose, lipoproteins and catecholamines during rest is altered in the SCI Q. The metabolism of these substances is affected by SNS innervation. During exercise, when the SNS is normally stimulated, these and other metabolic pathways that are stimulated by SNS innervation may be inhibited, thereby causing an earlier onset of fatigue and a decrease in performance.

CONCLUSION

Past research has found that there are alterations in metabolism during rest in sedentary SCI (13,63-70,72,73). Few studies have investigated the metabolic response during exercise in SCI P. To our knowledge, No one has studied the metabolic responses during exercise in SCI Q. Much of the work that has been done in the past on SCI and exercise has used a relatively small sample size and inappropriate subjects, mixing males and females (3,5,10), P with Q (2,21,31), incomplete with complete SCI (23,31,86) and traumatic SCI with poliomyelitis or other diseases (3-5,21-23,31,86). At the present time there is a significant difference in performance between P and Q due to the differences in amount of functional muscle mass and lack of
SNS innervation in Q. Quadriplegics must deal with more physiological factors. Our research will investigate the metabolic responses during exercise in Q athletes and compare these to P athletes. From this information we will be able to develop more appropriate training programs for Q athletes which will consequently result in better performances.
Chapter III
EXPERIMENT

INTRODUCTION

During prolonged exercise in able bodied (AB) athletes, the sympathetic nervous system (SNS) is stimulated and norepinephrine (NE) and epinephrine (E) are released (35,36). NE stimulates triglyceride (TG) mobilization from adipose tissue and increases plasma free fatty acids (FFA) (25). The increase in plasma FFA stimulates fatty acid use by exercising muscle (76). Muscle tissue can thus rely less on carbohydrates for fuel and conserve existing muscle glycogen.

The spinal cord contains hundreds of nerves which travel from the brain through the cord and branch out at various levels of the cord to muscles. These nerves connect the brain to the muscles facilitating communication and coordination. An injury to the spinal cord breaks this connection and there is a loss of sensation and movement below the injury level. SCI affects both the central and autonomic nervous system. A paraplegic (P) has a SCI below the cervical area. This damage causes complete or partial paralysis of the lower limbs, because the nerves innervating the muscles of the legs are in the lumbar area, below the level of lesion. Therefore, a higher injury of the cord results in a greater loss of sensation and movement because more nerves are affected. A quadriplegic (Q) has damaged the spinal cord in the cervical area, resulting in a loss of sensation of all or part of the
four limbs. Since the SNS branching occurs in the thoracic-lumbar area, the entire SNS would be affected in a Q because it is below the level of lesion. Decreased SNS innervation may alter the metabolic response to exercise. During exercise a Q may rely more on carbohydrates for fuel because TG are not mobilized and FFA are not as readily available to working muscle as in a P or an AB person. This would result in a reduced ability to perform and earlier fatigue due to the depletion of muscle glycogen (26-30). The degree of diminished SNS innervation, and possibly extent of altered metabolism, in a P would depend on the level of lesion.

Diminished SNS activity has been shown to impair performance in AB subjects (52,56-61). The use of non-selective β-blocker medication inhibits the SNS activity and impairs performance in the AB by decreasing adipose tissue TG hydrolysis, by reducing FFA available to working muscle as a fuel source. This results in accelerated glycogen depletion, decreased blood glucose, fatigue and impaired performance (57,59). Since Q subjects have diminished SNS capacity, an inhibition of TG mobilization, similar to that observed in β-adrenoreceptor blockade, may reduce prolonged submaximal activity of these subjects due to lack of substrate available to working muscle.

Few studies have looked at fuel use during exercise in P subjects (24,31). Metabolism during exercise in Q wheelchair athletes has not been investigated to our knowledge. The
purpose of the present study was to compare the metabolic responses of trained SCI P and Q wheelchair athletes during prolonged submaximal exercise.

METHODS

SUBJECTS: Eight SCI male subjects, four P and four Q athletes 25 to 36 years of age, volunteered for this study. Subjects were recruited from elite track/road racing athletes representing highly trained wheelchair athletes. After an initial familiarization session, including an explanation of the risks and benefits of the proposed study, a written informed consent was obtained from each subject. The use of human subjects conformed to the guidelines of the Human Ethics Review Board at the University of Manitoba Faculty of Human Ecology.

The subjects were tested at the University of Manitoba Sport and Exercise Sciences Research Institute Laboratory on two consecutive days. On day one, anthropometric measurements were taken and a progressive maximum VO₂ test was performed to establish the appropriate conditions for the submaximal test. On day two, athletes completed a two hour submaximal test at a speed corresponding to 75% of their VO₂max. All subjects used their own custom made racing chair for the tests. Each wheelchair had a cyclocomputer (Cateye) attached to the rear wheel for monitoring the speed. The accuracy of the cyclocomputer was ± 1 kph. The chairs were attached to a pair of frictionless rollers (Top End).
ANTHROPOMETRIC MEASUREMENTS: Length of total body, weight, girths for the upper arm, forearm, wrist, chest and upper thigh were measured. Skinfolds were measured on pectorals, biceps, triceps, subscapular, and abdominals using a calliper (77). Anthropometric measurements were taken to determine if physical differences exist between Q and P athletes.

MAXIMAL TEST: The protocol used was similar to that described by Eriksson (6). Subjects began a slow four min warm up at 7 to 12 kph and 18 kph for Q and P respectively. Athletes then increased their speed by two kilometres per hour every two min until voluntary exhaustion. Total time to exhaustion was 6 to 15 min for the Q subjects and 10 to 17 min for the P subjects. During the progressive exercise test heart rate (HR) was monitored continuously from a CM5 lead placement and recorded during the last 10 seconds of each 2 min stage. Subjects breathed through a two-way valve (Hans Rudolf) and the expired gases were analyzed using an automated system (MMC Sensormedics). Thirty second calculations were made of oxygen uptake (\(V_O_2\) in L/min and ml/kg/min), minute ventilation corrected to BTPS (\(V_E\) in L/min), tidal volume (VT in L/breathe), and breath frequency (f in Br/min). The metabolic system was calibrated between each test using known gases.

SUBMAXIMAL TEST: Prior to the submaximal test, subjects were instructed to eat a small carbohydrate meal at least two
hours before their test and refrain from consuming caffeine beverages. Examples of a small carbohydrate meal was given. Subjects worked at the speed corresponding to 75% of their maximal VO₂ (Figure 3) for 2 hours. Water was given ad libitum during the 2 hours. Expired gases were collected for five min prior to the commencement of exercise and at 25, 55, 85, and 115 min after the commencement of exercise. VO₂, VE, VT, f and Respiratory Exchange Ratio (RER) were determined in accordance with previously described procedures.

Blood samples were drawn from the antecubital vein by a registered laboratory technologist at pre-exercise and after 30, 60, and 120 min of exercise. To facilitate sampling, exercise was stopped at 30 and 60 min for two to three min while blood samples were taken.

GLUCOSE ANALYSIS: Blood was collected in a vacutainer tube and stored on ice. The sample was allowed to clot for one hour prior to centrifugation at 1500 g. Approximately 0.5 ml of serum was transferred to 1.5 ml polypropylene tube and stored at 4 °C. Samples were analyzed within 2 days after collection using a Sigma Glucose Kit, procedure number 115 (Sigma Chemical Company, St. Louis, MO, U.S.A.).

INSULIN ANALYSIS: Blood was collected in a vacutainer tube and allowed to clot for one hour at room temperature. Samples were centrifuged at 1500 g for 10 min, and the serum was
Figure 3. CALCULATION FOR DETERMINATION OF SPEED DURING PROLONGED TEST FOR PARAPLEGIC AND QUADRIPLAGIC WHEELCHAIR ATHLETES
transferred to polypropylene tubes. Samples were stored at 
-20 °C and analyzed using a Pharmacia Insulin RIA Diagnostic 
Kit (Pharmacia Fine Chemicals, Uppsala, Sweden).

LACTATE ANALYSIS: Blood was collected in heparinized 
vacutainer tubes, and 0.5 ml of whole blood was immediately 
pipetted into 1 ml of ice cold 10% TCA solution. The sample 
was vortexed for 30s, left in an ice bath for 5 min and then 
spun at 1500 g for 10 min. The supernatant was transferred in 
a glass vial and stored at 4 °C. Samples were analyzed within 
4 days after collection using a Sigma Lactate Kit 826 UV 
(Sigma Chemical Company, St. Louis, MO, U.S.A.).

FREE FATTY ACID ANALYSIS: Blood was collected in a vacutainer 
tube and stored on ice. The sample was allowed to clot for 
one hour before centrifugation at 1500 g. Two aliquots of 
serum were transferred to 1.5 ml polypropylene tubes and 
stored at -80 °C until analyzed. Samples were analyzed for 
FFA according to the method of W.G. Duncombe (78). All 
samples were analyzed within 4 weeks after collection.

CATECHOLAMINES ANALYSIS: Blood was collected in ice cold 
heparinized vacutainer tubes, kept on ice for 5 min. and then 
spun at 1500 g for 10 min. Two aliquots of plasma were 
transferred to glass vials and stored at -80 °C until 
analyzed.
For analysis, 1.0 ml of plasma was added to 20 mg of alumina in a polypropylene tube and mixed on a vortex. One ml cold tris buffer (1M containing 0.05M EDTA, pH 8.6) was added and the tube was mixed for an additional 20 s. Tubes were capped tightly and placed horizontally in a shaker water bath for 20 min.

After shaking, the tubes were inverted several times to dislodge any alumina particles stuck to the tube walls and the left upright for several min to allow the alumina to settle. The supernatant was discarded and the alumina washed 3 times with 1 ml of cold HPLC water, mixing the tubes for 15 s after the addition of water and allowing the tubes to settle for 1 min before discarding the supernatant. One hundred µl of cold 0.1M perchloric acid was added to the tubes and the tubes were mixed for 60 s on a vortex and then centrifuged at 2000 g for 5 min. The supernatant was transferred to a 1.5 ml conical polypropylene tube and stored at 5 °C until analyzed.

Catecholamines were analyzed by HPLC using a Beckman System Gold with 116 pump (Beckman Instruments Limited, Toronto, Ont.) and an ESA Coulochem 5100A detector equipped with a model 5011 analytical cell and a model 5021 conditioning cell. The column was supplied by Chromatography Science Company (CSC) Inc. and was an OD52, 5 µm column, 25 x 0.46 cm. The precolumn contained Alltech pellicular C18 packing. The mobile phase contained 75 mM sodium phosphate, 50 µM EDTA, 2 mM octane sulfonic acid and 10% acetonitrile,
and was adjusted to a pH of 3.25 with phosphoric acid. Because of co-eluting peaks found in many of the plasma samples from the SCI subjects, no internal standard was used in this procedure. Recovery of both NE and E standards added to plasma samples was 67.1% ± 2.5 (SE) for 10 samples. Data was corrected to 100% recovery. All samples were analyzed within 5 weeks after collection.

STATISTICAL ANALYSIS:

The data were analyzed using the Statview Statistical Computer Program by an analysis of variance with repeated measures (ANOVA). The significance level chosen for ANOVA was p<0.05. If significance was obtained, a post hoc test (Fisher PLSD) was used to determine which means were significantly different.

RESULTS:

PHYSICAL CHARACTERISTICS: Physical characteristics and anthropometric measurements are presented in Tables 2, 3 and 4. No statistical differences were found between P and Q subjects for age, length, weight, girths or skinfolds.

MAXIMAL TEST: Maximum heart rate was significantly greater for P (194 ± 13 BPM) than Q (127 ± 34) (p<0.05). VE and VT were also significantly greater in the P (122.2 ± 18.9 and 1.9 ± 0.6) than the Q (63.9 ± 16.5 and 1.2 ± 0.3) (p<0.05).
TABLE 2. PHYSICAL CHARACTERISTICS OF PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>CLASS</th>
<th>AGE (yrs)</th>
<th>LENGTH (cm)</th>
<th>WEIGHT (kg)</th>
<th>AVE MARATHON SPEED (kph)</th>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>P1</td>
<td>T4</td>
<td>24.9</td>
<td>164.1</td>
<td>69.7</td>
<td>22.5</td>
</tr>
<tr>
<td>P2</td>
<td>T4</td>
<td>24.8</td>
<td>175.1</td>
<td>65.5</td>
<td>22.1</td>
</tr>
<tr>
<td>P3</td>
<td>T3</td>
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<td>175.5</td>
<td>74.0</td>
<td>24.7</td>
</tr>
<tr>
<td>P4</td>
<td>T3</td>
<td>32.8</td>
<td>164.7</td>
<td>64.3</td>
<td>22.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>X</td>
<td></td>
<td>27.4</td>
<td>169.8</td>
<td>68.3</td>
<td>23.1 *</td>
</tr>
<tr>
<td>S.D.</td>
<td></td>
<td>3.75</td>
<td>6.30</td>
<td>4.41</td>
<td>1.15</td>
</tr>
</tbody>
</table>

| Quadriplegics | | | | | |
| Q1 | T1 | 26.7 | 159.6 | 51.8 | — |
| Q2 | T2 | 35.9 | 176.6 | 80.1 | 17.0 |
| Q3 | T2 | 31.1 | 172.8 | 62.1 | 20.7 |
| Q4 | T2 | 25.6 | 167.9 | 62.4 | 20.3 |
| | | | | | |
| X | | 29.8 | 169.2 | 64.1 | 19.3 * |
| S.D. | | 4.70 | 7.36 | 11.77 | 2.0 |

* Significantly different at p<0.05
### TABLE 3. SKINFOLD MEASUREMENTS OF PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES *

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>PECTORAL</th>
<th>BICEPS</th>
<th>TRICEPS</th>
<th>SUBSCAPULAR</th>
<th>SUM OF SKINFOLEDS</th>
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<td></td>
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</tr>
<tr>
<td>P1</td>
<td>5.9</td>
<td>3.4</td>
<td>5.7</td>
<td>9.6</td>
<td>24.5</td>
</tr>
<tr>
<td>P2</td>
<td>3.7</td>
<td>2.9</td>
<td>4.6</td>
<td>7.2</td>
<td>18.4</td>
</tr>
<tr>
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<td>11.0</td>
<td>15.1</td>
<td>41.3</td>
</tr>
<tr>
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<td>6.8</td>
<td>12.1</td>
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</tr>
<tr>
<td>$\bar{X}$</td>
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<td>3.6</td>
<td>7.0</td>
<td>11.0</td>
<td>28.1</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1</td>
<td>7.3</td>
<td>3.2</td>
<td>5.8</td>
<td>7.3</td>
<td>23.6</td>
</tr>
<tr>
<td>Q2</td>
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<td>17.2</td>
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<td>67.0</td>
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<td>Q4</td>
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<td>8.1</td>
<td>26.3</td>
</tr>
<tr>
<td>$\bar{X}$</td>
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<td>5.2</td>
<td>9.2</td>
<td>11.8</td>
<td>34.4</td>
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<td>5.77</td>
<td>8.78</td>
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</table>

* All measurements reported in mm.
TABLE 4. Girth Measurements of Paraplegic and Quadriplegic Wheelchair Athletes

<table>
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<tr>
<th>SUBJECT</th>
<th>ARM</th>
<th>FOREARM</th>
<th>WRIST</th>
<th>CHEST</th>
<th>THIGH</th>
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<td></td>
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<tr>
<td>P1</td>
<td>34.5</td>
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<td>18.1</td>
<td>109.7</td>
<td>34.2</td>
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<tr>
<td>P2</td>
<td>32.0</td>
<td>28.8</td>
<td>17.2</td>
<td>98.2</td>
<td>38.0</td>
</tr>
<tr>
<td>P3</td>
<td>36.2</td>
<td>31.2</td>
<td>17.6</td>
<td>112.7</td>
<td>42.7</td>
</tr>
<tr>
<td>P4</td>
<td>31.6</td>
<td>28.3</td>
<td>16.9</td>
<td>98.3</td>
<td>36.2</td>
</tr>
<tr>
<td>( \bar{X} )</td>
<td>33.6</td>
<td>30.1</td>
<td>17.4</td>
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<td>37.8</td>
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<td>S.D.</td>
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<td>0.50</td>
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<tr>
<td>Q1</td>
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<tr>
<td>Q3</td>
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<td>16.3</td>
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<td>Q4</td>
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<td>27.2</td>
<td>16.3</td>
<td>92.7</td>
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</tr>
<tr>
<td>( \bar{X} )</td>
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<td>26.5</td>
<td>16.6</td>
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<td>S.D.</td>
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<td>2.43</td>
<td>0.61</td>
<td>8.15</td>
<td>3.64</td>
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</tbody>
</table>

* All measurements are reported in cm.
Maximum VO₂ for P (2.5 ± 0.3 L/min) was greater, but not significantly than Q (1.5 ± 0.6 L/min) (Table 5).

SUBMAXIMAL TEST: The average VO₂ during the 2 hour submaximal exercise period (Table 6) was 1.5 ± 0.4 and 0.9 ± 0.2 L/min, which corresponded to 60% and 68% of the max VO₂ for the P and Q subjects, respectively as determined in the maximal test. This difference was not significant, however the large amount of variability between subjects may have obscured this difference. Heart rate during the submaximal test was 141 ± 29 and 123 ± 13 bpm for the P and Q subjects, respectively. Although there was no significant difference in heart rates between the P and Q, the percent of maximal heart rate was significantly greater in the Q (p<0.05).

VE steadily decreased in the Q during the prolonged test from 40.0 to 33.3 L/min. In P, VE remained constant. R E R decreased progressively during the last 90 min of exercise from .93 to .80 and from .99 to .85 in the P and Q subjects, respectively (Figure 4). There was a significant decrease from 30 min to 120 min for P and from 30 min to 90 min in Q (p<0.05). The slightly lower RER in the P was not significant.

Norepinephrine was significantly higher in the P during the prolonged exercise than in the Q (p<0.05) (Figure 5). In the P, NE increased during exercise and was significantly higher at the end of the exercise than prior to exercise.
TABLE 5. PHYSIOLOGICAL DATA FROM MAXIMUM VO₂ TEST FOR PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>m(\dot{V}O_2) (ml·kg(^{-1})·min(^{-1}))</th>
<th>m(\dot{V}O_2) (L·min(^{-1}))</th>
<th>mHR (bpm)</th>
<th>(\dot{VE}) (L·min(^{-1}))</th>
<th>VT (L·br(^{-1}))</th>
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<tr>
<td>P1</td>
<td>41.9</td>
<td>2.9</td>
<td>207</td>
<td>138.0</td>
<td>2.7</td>
</tr>
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<td>P2</td>
<td>34.1</td>
<td>2.2</td>
<td>200</td>
<td>95.2</td>
<td>1.9</td>
</tr>
<tr>
<td>P3</td>
<td>34.8</td>
<td>2.6</td>
<td>177</td>
<td>124.4</td>
<td>1.5</td>
</tr>
<tr>
<td>P4</td>
<td>35.4</td>
<td>2.3</td>
<td>191</td>
<td>131.3</td>
<td>1.4</td>
</tr>
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<td>(\bar{X})</td>
<td>36.8</td>
<td>2.5</td>
<td>194*</td>
<td>122.2*</td>
<td>1.9*</td>
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<td>0.3</td>
<td>13</td>
<td>18.9</td>
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</tr>
<tr>
<td>Q1</td>
<td>14.5</td>
<td>.8</td>
<td>97</td>
<td>41.1</td>
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</tr>
<tr>
<td>Q2</td>
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<td>1.9</td>
<td>122</td>
<td>68.8</td>
<td>1.6</td>
</tr>
<tr>
<td>Q3</td>
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<td>1.4</td>
<td>115</td>
<td>65.2</td>
<td>1.3</td>
</tr>
<tr>
<td>Q4</td>
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<td>2.0</td>
<td>175</td>
<td>80.4</td>
<td>0.9</td>
</tr>
<tr>
<td>(\bar{X})</td>
<td>23.0</td>
<td>1.5</td>
<td>127*</td>
<td>63.9*</td>
<td>1.2*</td>
</tr>
<tr>
<td>S.D.</td>
<td>7.2</td>
<td>0.6</td>
<td>34</td>
<td>16.5</td>
<td>0.3</td>
</tr>
</tbody>
</table>

* Significant difference at \(p<0.05\)
## TABLE 6. PHYSIOLOGICAL DATA FROM THE SUBMAXIMAL TEST FOR PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>( \text{VO}_2 ) (L min(^{-1}))</th>
<th>VE (L min(^{-1}))</th>
<th>VT (L br(^{-1}))</th>
<th>( f ) (br min(^{-1}))</th>
<th>HR (bpm)</th>
<th>%m( \text{VO}_2 )</th>
<th>%mHR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paraplegics</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>P1</td>
<td>1.820</td>
<td>72.0</td>
<td>1.5</td>
<td>51.4</td>
<td>161</td>
<td>63</td>
<td>78</td>
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<td>33.2</td>
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<td>55</td>
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<td>73</td>
<td>96</td>
</tr>
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<td>0.9</td>
<td>56.8</td>
<td>124</td>
<td>55</td>
<td>65</td>
</tr>
<tr>
<td>( \bar{X} )</td>
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<td>55.5</td>
<td>1.1*</td>
<td>49.2</td>
<td>141</td>
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<td>0.3</td>
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<td>0.8</td>
<td>23.6</td>
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<tr>
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<td>0.9</td>
<td>48.9</td>
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<tr>
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<td>0.7</td>
<td>59.8</td>
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<td>85</td>
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<tr>
<td>( \bar{X} )</td>
<td>0.906</td>
<td>38.5</td>
<td>0.8*</td>
<td>47.5</td>
<td>123</td>
<td>68</td>
<td>99*</td>
</tr>
<tr>
<td>S.D.</td>
<td>0.237</td>
<td>13.9</td>
<td>0.1</td>
<td>16.6</td>
<td>20</td>
<td>17</td>
<td>10</td>
</tr>
</tbody>
</table>

* Significant difference at p<0.05
Figure 4. RER RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES
Figure 5. NOREPEINEPHRINE RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES
Norepinephrine remained unchanged for the first 60 min then increased, but not significantly, during the last 60 min in the Q. Epinephrine increased significantly in the last 60 min of exercise in the P and was significantly higher than in the Q after exercise. Epinephrine was virtually unchanged in the Q (Figure 6).

Blood lactate increased from 1 to 3 mmol/L (p>0.05) in the first 30 min in both the P and Q. (Figure 7). Blood lactate decreased to pre-exercise levels in the Q but remained elevated in the P for the duration of the exercise period.

Blood glucose concentrations were relatively constant throughout the two hour exercise period in the P athletes (Figure 8). Blood glucose decreased significantly in the Q athletes during the first 30 min of exercise (p<0.05) and then increased from 60 min to 120 min to a point where the difference between time 0 and time 120 was insignificant.

Insulin also decreased significantly in the first 30 min of exercise in the Q and then remained constant. In the P, insulin remained unchanged throughout the two hour exercise period (Figure 9). The insulin concentrations in the Q (217 ± 227 pmol/L) was significantly higher prior to exercise than the P (88 ± 75 pmol/L). After 30 min there was no significant difference in insulin levels between P and Q.

Plasma FFA profiles were similar for Q and P during the 2 hour exercise period. Free fatty acid levels increased significantly (p<0.05) from .4 ± .2 to 1.0 ± .8 mmol/L and
Figure 6  EPINEPHRINE RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPEGIC WHEELCHAIR ATHLETES
Figure 7. LACTATE RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES
Figure 8. GLUCOSE RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES
Figure 9. INSULIN RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES.
from 0.2 ± 0.1 to 1.4 ± 0.3 mmol/L in P and Q, respectively in the last 60 min of exercise in both groups (p<0.05) (Figure 10). There was no significant difference between the two groups.

DISCUSSION:
Anthropometric Measurements: The girths and skinfolds of those areas that were not paralysed were similar to those of AB upper body trained athletes (79).
Maximal Test: The VO_{2} max values achieved by the P subjects in our study are comparable to those in the literature of trained P (3,6,8,10,). It is, however, difficult to make direct comparisons between our results and those found in previous studies because of different testing protocols used. Also, previous studies used subjects with a variety of disabilities and both male and female subjects. We specifically chose only male traumatic SCI, well trained, track and road racing athletes and separated into P and Q to make the groups as homogeneous as possible.

The VO_{2} max for one subject (P3) may have been underestimated because work time during his progressive test was too long. The most accurate maximum VO_{2} test is one where subjects reach their maximum within 3 to 10 min (80). Subject P3 reached his maximum in 20 min, thereby making the test more of aerobic capacity rather than aerobic power. A solution to this problem in the future may be to ask the subject to reach
Figure 10. FFA RESPONSE DURING PROLONGED EXERCISE IN PARAPLEGIC AND QUADRIPLEGIC WHEELCHAIR ATHLETES
his top speed before the test to determine his highest speed. The subject would then warm up at 10 kph below his highest speed and continue increasing his speed by 2 kph every two min until exhaustion.

Although significant differences in VO$_{2}$max were not found between P and Q, this may be due to subject Q4, who is an incomplete Q. Because of the nature of Q4 injury, some nerves are spared below his lesion, thereby allowing him to use more muscle during exercise. When Q4 is taken out of the analysis, significant differences are seen between P and Q.

The significant differences in VT are most likely due to the differences in amount of functional expiratory muscles in P and Q. The paralysis of the abdominal muscles involved in breathing cause the Q to rely on the diaphragm muscles only (18).

Submaximal Test: The workloads during the submaximal test were under predicted for most of the subjects. This is expected when a progressive maximum test is used for predicting the submaximal workload (81). The intensities also varied between the subjects but on average all were working at a moderate intensity.

It was interesting to note that the difference between percent VO$_{2}$_max and percent max heart rate during the submaximal test was greater in the Q than that for the P. The P subjects average oxygen uptake corresponded to 60% of their VO$_{2}$_max and their average heart rate was 74% of their max heart
rate which is similar to an AB (80). The Q subjects average oxygen uptake during the prolonged exercise was, however, 68% of their VO₂max but their average heart rate was 99% of their max heart rate. Q athletes appear to be able to work at a higher percent of their max heart rate than P or AB athletes for a longer period of time, possibly because their max heart rate is much lower. Therefore, heart rate may be an unreliable intensity measure for Q athletes.

The average heart rate during exercise was 74% and 99% of max for P and Q subjects, respectively. The estimated max heart rate for an individual is generally calculated by subtracting age from 220 bpm (80). A Q max heart rate would be approximately 120 bpm due to lack of SNS innervation. It is speculated that P3, Q1 and Q3 did not reach their true max heart rate during the maximal test. P3 had max heart rate that was well below his estimated heart rate. Q1 and Q3 were working at a higher average heart rate during the submaximal exercise than the maximum heart rate measured during the maximal test. This would cause the average percent of max heart rate during the submaximal test to appear higher than it really is. Therefore, according to their maximal test, their max heart rates during the submaximal test were 96, 103, and 108% but in reality may have been less than 80%.

Q4 had a physiological profile that was between the P and Q subjects. His VO₂max was 2.0 L/min, 0.5 L/min less than the mean for the P but 0.7 L/min more than the Q average. His
heart rate was 175 bpm, indicating there is some SNS innervation. Insulin response was similar to that of the P. Norepinephrine and RER responses were between those observed for P and Q, while glucose and FFA followed the general trend of the Q. Because of these differences between incomplete and complete Q, the physiological potential may be greater for the incomplete Q and should be taken into consideration when reviewing the present classification system for wheelchair sports. At the present time wheelchair athletes are simply classified according to functional skeletal muscle (16). This is done to permit fair competition. An incomplete Q athlete would have more functional muscle than the complete Q athlete yet less than the P athlete. His SNS would be less effected and therefore would have an advantage over the other Q athletes. Perhaps a separate class is needed for incomplete Q in order to maintain fair competition for all.

During the submaximal, test the RER for P was slightly lower than that of the Q. RER in the Q was higher during the first 60 min than those of competitive cyclists and untrained AB working at 62% of their VO2max for 60 min (44). The RER of the P was similar to those of the untrained AB yet higher than the trained cyclists (44).

The lactate values were slightly lower in the Q. This difference is most likely due to the smaller amount of functional muscle producing the lactate. In turn, the liver
would be able to metabolize the lactate more quickly because of the smaller absolute amount produced.

FFA increased during the last 60 min of exercise in both groups. The average FFA value for the P is skewed upwards at 120 min because of P3. If P3 is taken out, a greater difference is seen between the P and Q, with the Q being higher at 120 min. Although the FFA concentration would be greater in the Q, their RER was also slightly higher than the P. This could be due to an increased mobilization of the FFA but an inability for muscle to take up the FFA for metabolism. It could also possibly be due to normal mobilization, but because a smaller amount of muscle is used, less fat would be utilized for fuel.

There may also be a greater use of muscle triglycerides in the P athletes, as seen in endurance trained AB athletes (44, 48). With endurance training, fat use increases at the same relative intensity as prior to training (44,48). Trained athletes also use more muscle triglycerides for fuel than untrained (44,48). This is evidenced by a lower RER and FFA concentration in endurance trained AB athletes (44,48).

Skrinar (31) reported an increase in FFA in a group of P basketball players after 60 min of exercise at 60% to 70% \( V_{O_2\text{max}} \). The FFAs were similar to our Q athletes, and lower than the P athletes at 60 min. The possible greater dependence on carbohydrates in Skrinar's subjects than our P athletes may be an indicator of aerobic fitness since
basketball is less of an endurance sport than road racing. We observed an increase in FFA and a decrease in RER in the P athletes. This observation is consistent with the research on AB subjects. Gass and Camp (24) observed an increase in FFA but no change in RER with P athletes working at the same relative intensity. The difference between the athletes in this study and those of Gass and Camp may be due to the higher fitness level of our subjects. The criteria for participating in the study by Gass and Camp was a marathon time of less than 2 hours 30 min. The best average marathon time for our P athletes was 1 hour and 49 min. Our subjects may have also had a lower FFA concentration because of a greater dependence on muscle triglycerides for fuel (44,48).

Compared to endurance trained AB runners, increased plasma FFA induced by exercise was less in P and Q athletes (29,44). This may be an indication of a greater dependence on carbohydrates in both the SCI P and Q athletes.

Blood glucose levels normally stay constant (39) or increase slightly (29,44) during prolonged exercise in trained AB. This is comparable to our P athletes. The blood glucose response in Q athletes, however is more like an untrained AB (39,44) where glucose drops during the exercise. A drop in glucose was also seen in the P wheelchair basketball players (31), further supporting the theory of increased fat with endurance training in P with little change in endurance trained Q athletes.
Insulin response followed that of glucose in both the P and Q athletes. It is not known why resting insulin and glucose values would be elevated in the Q subjects. Some subjects may not have adhered to the diet protocol and had consumed a food containing sugar before the prolonged exercise. Insulin has not been measured during exercise in wheelchair athletes to our knowledge. Some studies (13,62-64) have suggested insulin and glucose responses in SCI persons to be different from healthy AB persons during rest. Although the results are conflicting, the majority of studies which have separated the P and Q have found fasting blood glucose levels to be in the low normal range (65,66,73). More insulin was also required to elicit the same response as that in AB (67), suggesting possible insulin resistance. Insulin response during exercise in AB is similar to the P in our study (80).

Epinephrine and NE increased during exercise in the P athletes suggesting a greater SNS stimulation. In AB athletes, NE and E increases during exercise (35,36). This increase stimulates fat mobilization from adipose tissue (25), muscle triglyceride use (82) and glucose release from the liver (37,38). Paraplegic NE levels prior to exercise are similar to AB (39) but are higher at the completion of exercise. Epinephrine, however, is lower in the P than AB (39). This may indicate that there is still some difference
in metabolic response between P and AB since part of the SNS is effected in P depending on the level of injury.

It is important to note that the two Q with the greatest NE response were also dysreflexic during the last stage of the prolonged exercise. Autonomic dysreflexia is a response to a negative stimulus below the level of lesion (i.e. distended bladder, broken bone, urinary tract infection, etc.)(18). Symptoms include increased blood pressure, goose bumps, red blotchy spots, and sweating. During dysreflexia, NE and E increase (68). In the last 60 min of exercise when NE increased in the Q, FFA and glucose increased and RER decreased. Many Q athletes stimulate dysreflexia during competitions by drinking large quantities of water and filling the bladder. Some will also wear a catheter and clamp the catheter closed to prevent the bladder from draining. A feeling of increased strength and stamina are usually experienced. The changes in blood parameters may explain the improved performance while dysreflexic during exercise. Because of the possible negative ramifications of autonomic dysreflexia, the practice of stimulating this response is not encouraged for improving performance.

CONCLUSION

Because of the small subject number it is not possible to make definite conclusions, however there does appear to be differences in metabolism between P and Q athletes during
exercise. This is evidenced by the slightly higher RER and greater initial drop in glucose and insulin values in the Q. The lower NE and E levels may be related to this difference. These results suggest that initially there may be a greater dependence on carbohydrates in Q but after 60 min of submaximal exercise, these metabolic differences are minimal. This suggests the need for a longer warm up for Q athletes in order to work towards a stage where their metabolism is similar to the P. A high carbohydrate diet may also be necessary in order to perform in initial stage of work where carbohydrate use is greater. Further studies with more subjects and greater control of work intensity during the prolonged test is needed. These findings may help to further understand the responses during training and improve the development of training methods for P and Q athletes.
CHAPTER IV

IMPLICATIONS OF POSSIBLE DECREASED LIPOLYSIS

These changes in metabolism can have several implications for both the SCI athlete and nonathlete. A possible decrease in lipolysis during exercise in SCI Q would indicate the need for attention in certain aspects of training and diet.

Implications for athlete:

1. For the athlete this would indicate the importance of a high carbohydrate diet during training. Prolonged exercise at above 60% max VO2 taxes glycogen stores in AB (26-30) and wheelchair athletes (31). A high carbohydrate diet would ensure adequate glycogen stores for work outs and also replenish stores after a work out or competition that has partially depleted them. A normal diet of 40-50% carbohydrates requires more time for glycogen repletion (83) than a diet providing 60 - 70% of total energy as carbohydrates (27,84).

2. Carbohydrate loading prior to an event would be particularly important for the Q. If there is increased carbohydrate metabolism during the first stage of exercise, full muscle glycogen stores will allow the Q athlete to work through the first stage until metabolism is more similar to that of P athletes and AB.

3. Carbohydrate drinks, such as glucose polymer drinks, may need to be consumed during work outs or races to spare glycogen. Studies have shown that consuming a glucose
polymer drink during exercise can delay fatigue in AB athletes (85). Consuming a small amount of carbohydrate (45 g) in the form of a glucose polymer drink or confectionery bar 5 min prior to a 1 hour exercise bout has also increased total work output (86).

4. Alterations in training programs may be needed, such as changes in rest and intensity. Hooker and Wells (87) found an intensity of at least 70% $V_O_2_{max}$ was required to elicit a training effect in SCI. This intensity is higher than that needed for AB (80). Perhaps a higher intensity is needed in Q because maximum heart rate is lower and a particular percent of max $V_O_2$ in Q athletes corresponds to a higher percent of maximum heart rate.

Implications for nonathlete:

1. A high carbohydrate diet would also be recommended for a non athlete to ensure adequate glycogen stores for activities of daily living which may require more effort for a Q than P.

2. Prevention of obesity. Weight loss in the form of fat may be more difficult in the SCI because fat is not mobilized effectively. Many SCI gain weight after rehabilitation because energy requirements are lower for the SCI than the AB (81), but preinjury intake is often maintained after injury. This may also be important for the control of blood glucose (64).
3. Exercise should be encouraged to prevent obesity. As mentioned previously, alterations are seen in AB subjects with training (44-47). Some of these adaptations may involve the SNS. For example, the L-hormone sensitive lipase which hydrolyses muscle TG is stimulated by E (88) and the sensitivity to E and NE increases with training in AB subjects (49). Sensitivity to insulin increases with training (50,89). Insulin inhibits the action of hormone sensitive lipase (40), which is involved in adipose tissue TG hydrolysis. When insulin levels are high, TG breakdown and release of FFA would decrease. Exercise also helps to decrease insulin resistance in insulin resistant diabetics by increasing insulin binding to monocytes (90). These factors could affect fat metabolism. The difference in VO2max between trained and untrained is not as great in Q as seen in P (6,91). There is an increase in performance indicated by the maximum speed achieved (6) and power output (7), however, which may be due to metabolic adaptations since changes in lactate and blood lipids are found with training (7). Exercise is also very important in the prevention of cardiovascular diseases. Exercise can increase HDL-C in SCI (7,13) and decrease TG and low density lipoproteins (12) due to increased lipoprotein lipase activity (5).
REFERENCES


