ABSTRACT

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Previous studies have demonstrated the importance of anaerobic capacity to performance during high intensity exercise. Competitive cyclists/skaters (N = 9) performed three randomly ordered control (C), sodium bicarbonate (SB), glycogen depletion (GD) 1500m cycling time trials. Power output was recorded using a strain gauge interfaced with a windload simulator attached to a racing bicycle. VO_2 was measured by open circuit spirometry. Power output and VO_2 were linked based on steady state exercise completed before the trials. Repeated measures ANOVA analyzed the outcomes. The GD trial took significantly longer than the C or SB trials (C133.96, SB133.49, & GD137.73s) $p < .05$. Mean total and anaerobic power output during the last 200m segment was significantly greater in SB compared to GD (C42879, SB44956 & GD40642 J), and (C20189, SB22194 & GD18741 J), but not for aerobic work (C22683, SB22762 & GD21901 J). The results support the hypothesis of augmenting anaerobic capacity during the sodium bicarbonate trial and reducing anaerobic capacity with glycogen depletion.

EFFECT OF SODIUM BICARBONATE AND GLYCOGEN DEPLETION ON 1500M TIME TRIALS

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THESIS FINAL ORAL DEFENSE FORM

Candidate Laura C.Behr

We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree:

Master of Science in Adult Fitness/Cardiac Rehabilitation

The candidate has successfully completed the thesis final oral defense.

Signature of Thesis Chairperson	Department	Date
Signature of Committee Member	Department	Date
Signature of Committee Member	Department	Date

This thesis is approved by the College of Health, Physical Education, Recreation, and Teacher Education.

Associate Dean, College of HPERTE	Date
Director of University Graduate Studies	Date

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INTRODUCTION

Performance during high intensity exercise depends on a combination of anaerobic and aerobic sources of energy. At the beginning of exercise, the active muscles in the body experience a lag in the delivery of oxygen necessary to maintain a high intensity work capacity [2,9,15]. The lack of availability of oxygen is known as oxygen deficit which has been suggested as a measure of anaerobic capacity [13, 19]. Once oxygen uptake has reached a steady state, there is ordinarily enough oxygen available for the aerobic energy system to provide for energetic resources.

During very high intensity exercise, typical of many sports events, the energy production at the maximal oxygen uptake is not adequate to meet the muscle's energetic requirement. The two main sources of anaerobic energy expenditure are phosphocreatine and anaerobic glycolysis. In short-term high intensity exercise (e.g., a 50m dash) the anaerobic metabolic system is responsible for ATP production largely through the phosphagen (ATP-PC) energy system. Since muscle stores only limited amounts of high energy phosphates, their breakdown only provides enough energy for short-term high intensity exercise bouts.

Anaerobic energy release can be directly quantified from measured changes in muscle lactate and phosphocreatine concentrations in working muscle [16,21]. However, the direct approach to measuring lactate concentrations requires muscle biopsy, which is not widely applicable. However, indirect means of measuring anaerobic energy expenditure are potentially capable of providing the same information. For example,

measurement of the accumulated oxygen deficit during cycling has been suggested as a strategy for evaluating the anaerobic capacity [3,26]. Data on accumulated oxygen deficit suggests that about two minutes of exercise to exhaustion is required to deplete anaerobic stores of energy [19].

An immediate source of energy comes from the ATP-PC system for short intensity exercise bouts. The second metabolic pathway capable of producing ATP without oxygen is glycolysis. Glycolysis involves the breakdown of glycogen to form glucose, which in turn forms two ATP and two pyruvic acids. Lactic acid is formed from pyruvate when oxygen is not readily available in the mitochondria to accept hydrogen ions from $NADH_2$. Since high intensity exercise requires the large amounts of oxygen, which cannot ordinarily be provided for, lactic acid accumulates in the muscles during high intensity exercise [16,21].

The importance of the anaerobic system to performance needs further documentation. One strategy for evaluating this issue is to manipulate anaerobic capacity by 1) taking away lactate production or 2) buffering pH changes. One strategy for manipulating the anaerobic capacity would be glycogen depletion. If no glycogen is available then no lactic acid can be produced, which in turn means a limitation in anaerobic energy production. Although still under research, sodium bicarbonate has shown to be effective in buffering the pH change resulting from lactate accumulation. Extracellular bicarbonate concentrations indirectly affect intracellular pH by maintaining a pH gradient between the two compartments. This enhances the movement of lactate and H+ out of the muscle. The administration of bicarbonate increases plasma pH and lactate

concentration following short-term high intensity exercise. The bicarbonate present in the blood enhances the pH gradient between intra- and extracellular compartments in favor of lactate efflux (movement of lactate from muscle to blood)[8,17,18]. Consequently, bicarbonate may be considered an ergogenic aid that delays fatigue in maximal exercise lasting from one to seven minutes. Accordingly, the purpose of this study is to determine oxygen deficit during 1500m cycle time trials. Our goal is to determine how effective accumulated oxygen deficit is as a marker of anaerobic capacity. The hypothesis of this study is that bicarbonate ingestion will enhance 1500m cycling performance and that with glycogen depletion performance will decline.

METHODS

Nine regionally competitive cyclists and speed skaters (2 female and 7 males) volunteered as subjects for this study. The cyclists were road specialists and the speed skaters were all metric specialists. Descriptive data for the subjects are presented in Table 1. The subjects provided informed consent prior to participation. The protocol had been previously approved by the University of Wisconsin-LaCrosse Institutional Review Board for the Protection of Human Subjects. Prior to the data collection, each subject completed practice trials of the 1500m cycle time trial in order to ensure that they were well habituated both to the task and data collection protocol. Additionally, each subject performed incremental exercise to fatigue on an electrically braked cycle ergometer with measurement of respiratory exchange by open circuit spirometry (Cosmed, K4, Italy) to allow definition of VO_2peak (peak 30s measurement) and ventilatory threshold using the v-slope technique [24].

The protocol started with a warm-up ride (see protocol Figure 1). Time trials were performed on a racing bicycle attached to a windload simulator with a heavy flywheel (Findlay Road Machine, Toronto, Canada). This device provides for velocity-VO_2 requirements and for inertia very much like cycling with a conventional bicycle [26]. Power output and distance were measured using a dynamometer (SRM, Koingskamp, Germany) based on a strain gauge built into the chain ring. Power output variables were recorded every second. Metabolic data were measured breath by breath using open-circuit spirometry. Heart rate (HR) was measured using radio telemetry (Polar Vantage XL, Polar Instruments, Port Washington, NY).

The subjects performed five cycle time trials; two were practice trials to habituate the subjects to the protocol. The subject had measures taken on three different days twenty-fours hours apart completing three randomly ordered cycle time trials of 1500m. The subjects completed a practice time trial of cycling under normal dietary conditions. On different day (>24h later), the subjects rode a preliminary 1500m trial (control) and then performed two hours of intense training on their own. Following this training ride, carbohydrate intake was restricted that night to cause a glycogen depletion for the 1500m time trial the next day [5]. The third trial consisted of 1500m of cycling preceded by sodium bicarbonate consumption at a dosage of 150 $mg*kg^{-1}$. Each subject warmed up prior to each ride according to a standard protocol (Figure 1). As a portion of the warm-up (Figure 1 Protocol), a five-minute submaximal ride was completed at a power output just below the ventilatory threshold (~200W (men) and ~150 W (women)) for the purpose of defining the relationship between power output and VO_2.

During the time trial, the only instruction to the subject was to complete each trial as quickly as possible. Feedback, including their performance (e.g., mean velocity) during the habituation rides, momentary velocity, and cumulative distances completed was provided to the subject, just as during competition. Split times were recorded at successive 100m intervals. From the submaximal trial, the relationship between the mechanical and metabolic work accomplished was calculated according to Garby et al. [10]. Subsequently, the average power output and the average VO_2 during each segment of each trial were calculated. We assumed that respiratory exchange ratios in excess of 1.00 were attributable to buffering. Accordingly, during the time trials VCO_2/VO_2 ratios in excess of 1.00 were treated as if they equaled 1.00 relative to calculating metabolic work. The mechanical work attributable to anaerobic energetic sources was calculated by subtracting the work attributable to aerobic metabolism from the total work toVO_2 relationship for each segment of the ride as well as for the total ride [10].

Statistical analysis was accomplished using repeated measures ANOVA comparing aerobic and anaerobic energy expenditure both within each trial and among trials to test the hypothesis that glycogen depletion would worsen performance by reducing anaerobic power output and that sodium bicarbonate would enhance performance by increasing anaerobic output. Statistical analyses were performed on data collected during the last 200m segment of each ride (except for total time) for aerobic, anaerobic, and total power output.

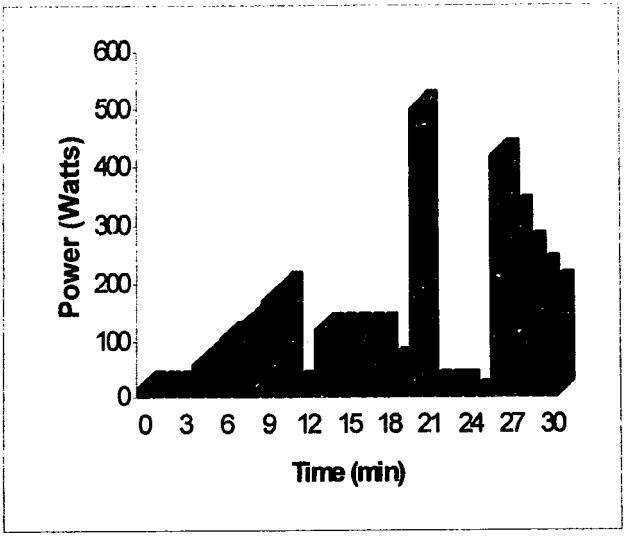


Figure 1. Protocol Submaximal Ride

RESULTS

Table 1 shows the descriptive characteristics of the subjects. The males were older and taller than the females. VO_2 peak ($L*min^{-1}$) was 3.82 ± 0.32 for males and 3.00 ± 0.42 for females. VO_2@ VT ($L*min^{-1}$) was 2.63 ± 0.44 for males and 1.90 ± 0.21 for females.

Table 1. Characteristics of the Subjects (±SD)

	Male (n=7)	Female (n=2)
Age (years)	32.6 ± 9.9	26.3 ± 7.5
Height (cm)	174.0 ± 12.6	174.3 ± 5.8
Weight (kg)	72.1 ± 3.8	68.0 ± 9.9
VO_2peak ($L*min^{-1}$)	3.8 ± 0.3	3. 0 ± 0.4
VO_2 @ VT ($L*min^{-1}$)	2.6 ± 0.4	1.9 ± 0.2

Figure 2 shows velocity over the three 1500m time trials. There was a decrease in velocity through each segment of each trial. Repeated measures ANOVA revealed that the bicarbonate trial produced a higher velocity for a longer period of time. Glycogen depletion was the slowest and the control was intermediate (Figure 2). The heart rate and power output during the submaximal trials were not significantly different but was used to determine efficiency (Table 2). Table 2 points out that velocity was not significantly different among the three submaximal trials.

Table 2. Submaximal Results (±SD) for Velocity, Power @ 5min, and HR @ 5min for Each Trial

	Control	Bicarb	GD
Velocity($m*s^{-1}$)	11.0 ± 1.2	11.0 ± 1.3	11.0 ± 1.2
Power @ 5min(watts)	141.0 ± 24.0	140.0 ± 23.2	134.0 ± 28.1
HR @ 5min	156.0 ± 23.6	152.0 ± 20.0	151.0 ± 27.4

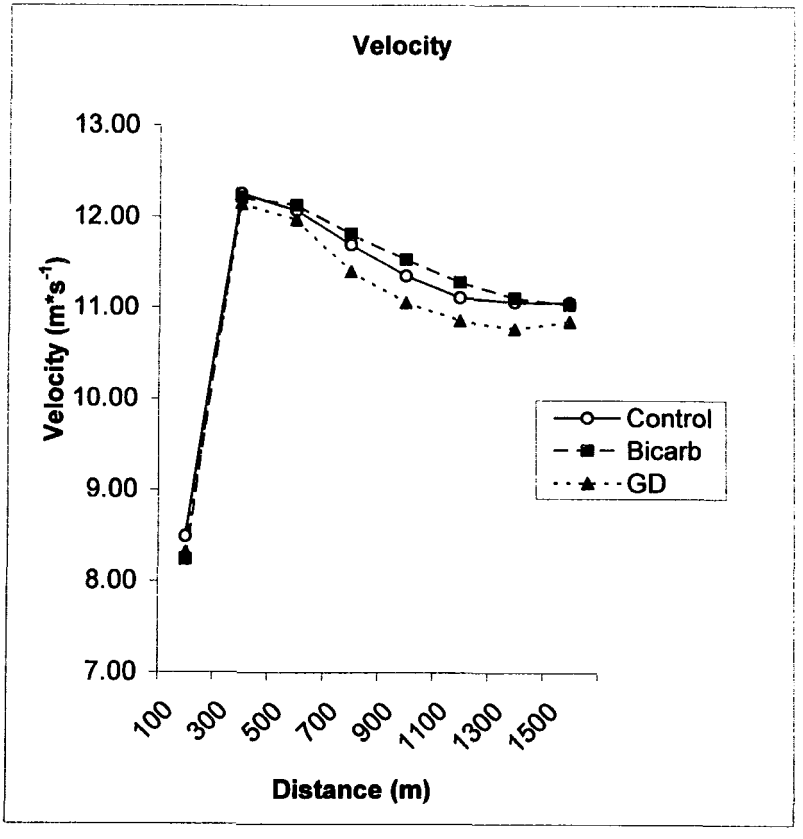


Figure 2. Mean Velocity during the 1500m Time Trial Measured every 200m

On average, the time trials were finished in 134.0 ± 5.8s in the control, 133.5 ± 6.1s in the bicarbonate, and 137.8 ± 6.7s in the glycogen depletion trials (Figure 3). There was a significant difference in time between the glycogen depletion trial ($p < 0.05$) versus both the bicarbonate and control trials. There was no significant difference between the bicarbonate and control trials ($p > 0.05$).

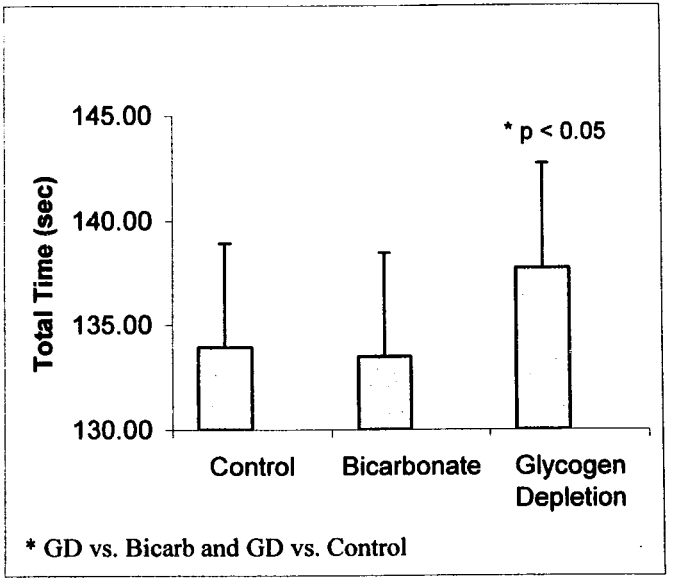


Figure 3. Mean Total Time for the Control, Sodium Bicarbonate, and Glycogen Depletion Conditions ($p < 0.05$)

Table 3 presents the data for total power, aerobic power, and anaerobic power over each 200m segment (with exception of the first 100m) for control, sodium bicarbonate, and glycogen depletion conditions. Figures 4, 5, and 6 present the same data graphically. Figure 7 and Table 4 present the VO_2 measured at various distances. There

was a significant difference in total power output over the last 200m between the glycogen depletion (268.0 ± 49.2W) and bicarbonate (292.0 ± 35.7W) conditions. The total power output during the control trial was 283.0 ± 66.3W, which was not significantly different from either glycogen depletion or bicarbonate conditions.

Table 3. Power Output (±SD) for the Control, Sodium Bicarbonate, and Glycogen Depletion Time Trials

	Total Power			Aerobic Power			Anaerobic Power		
	Control	Bicarb	GD	Control	Bicarb	GD	Control	Bicarb	GD
100	451 ± 92	431± 98	402 ± 114	76 ± 17	68 ± 16	74 ± 13	375 ± 93	363 ± 101	327 ±110
300	390 ± 93	409 ± 85	368 ± 91	137 ± 16	137 ± 28	121 ± 33	253 ± 88	272 ± 94	247 ± 80
500	347 ± 59	366 ± 50	333 ± 61	179 ± 24	178 ± 46	166 ± 61	168 ± 55	188 ± 64	167 ± 39
700	314 ± 40	341 ± 39	291 ± 46	184 ± 31	184 ± 49	169 ± 59	130 ± 39	156 ± 54	122 ± 34
900	292 ± 41	318 ± 31	269± 39	189 ± 36	194 ± 41	178 ± 33	91 ± 38	124 ± 42	103 ± 31
1100	278 ± 48	302 ± 30	258 ± 38	186 ± 36	192 ± 51	183 ± 47	92 ± 30	110 ± 46	75 ± 36
1300	280 ± 53	290 ± 31	255 ± 45	184 ± 36	188 ± 52	184 ± 47	96 ± 38	102 ± 39	71 ± 31
1500	283 ± 66	292 ± 35	268 ± 49	189 ± 39	187 ± 55	181 ± 51	94 ±49	105 ± 47	87 ± 27

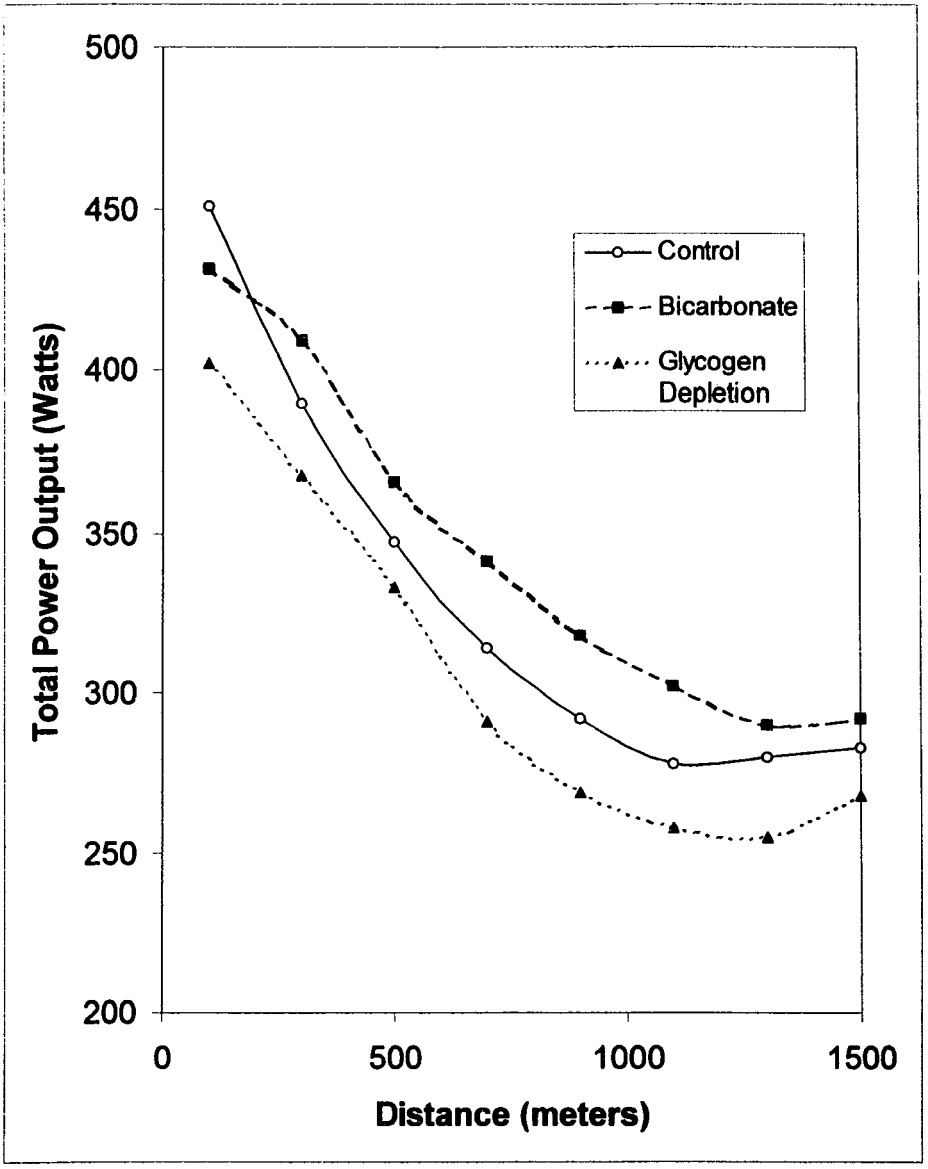


Figure 4. Mean Total Power Output during the 1500m Time Trial Measured every 200m

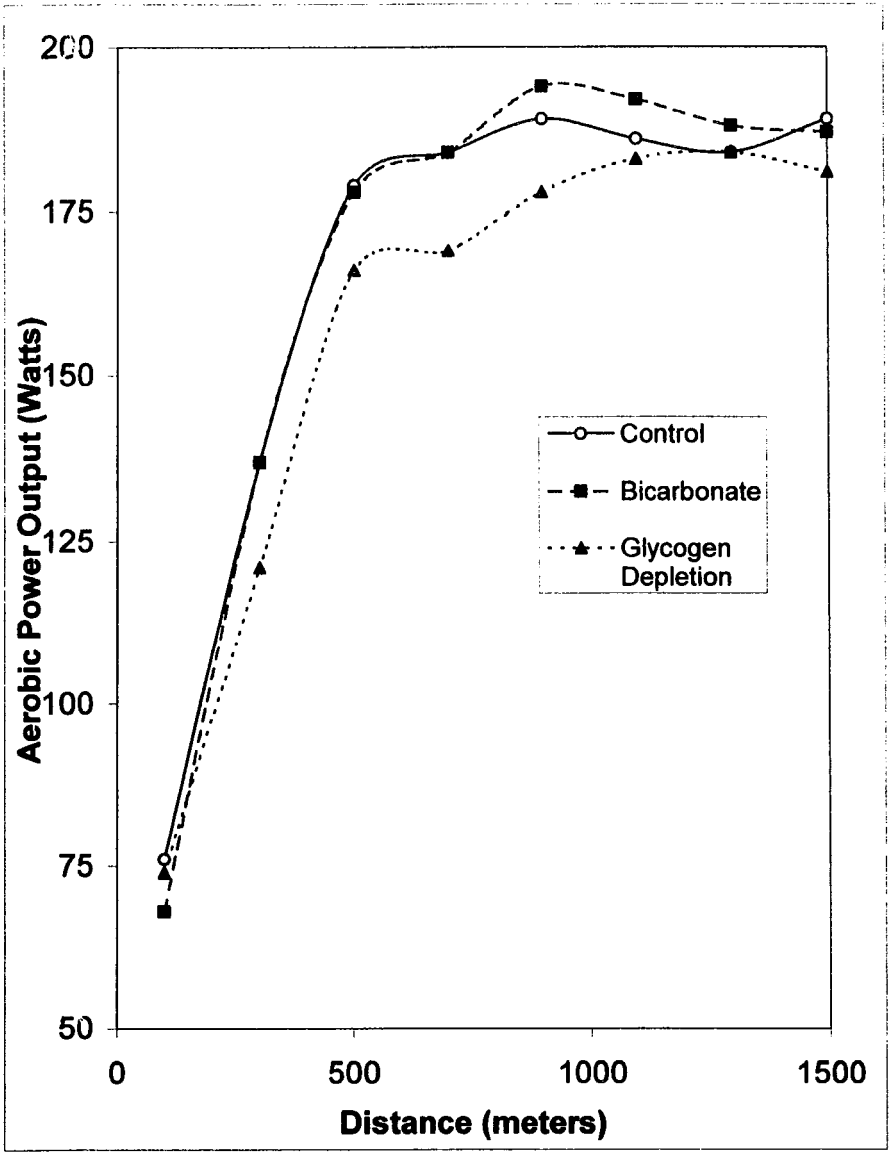


Figure 5. Mean Aerobic Power Output during the 1500m Time Trials Measured every 200m

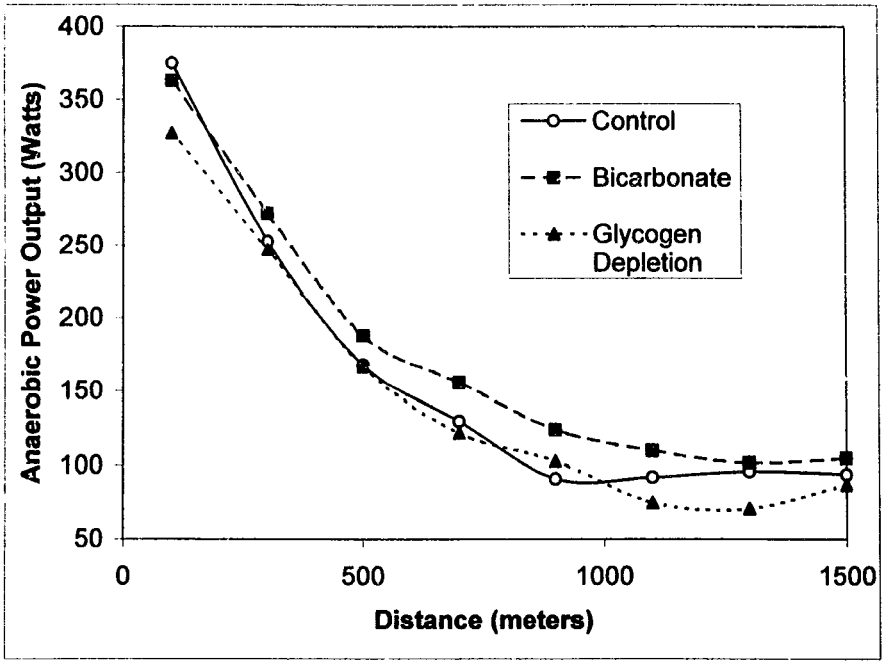


Figure 6. Mean Anaerobic Power Output during the 1500m Time Trial Measured every 200m

For aerobic power output there was no significant difference between the three trials ($p > 0.05$) over the last 200m. In the control trial, aerobic power output averaged 189.0 ± 39.6W, for bicarbonate 187.0 ± 55.6W, and 181.0 ± 51.4W for glycogen depletion (Figure 5). For anaerobic power output there were significant differences among conditions over the last 200m. Bicarbonate had a higher mean anaerobic power output (105.0 ± 47.8W), compared to the control (94.0 ± 49.2W) and to the glycogen depletion trials (87.0 ± 27.6W).

There were no significant differences in VO_2 among the different trials ($p > 0.05$) over the last 200m segment of the time trials. The mean VO_2 for the control trial was 3.34 ± 0.44 ($L*min^{-1}$), for bicarbonate 3.38 ± 0.60 ($L*min^{-1}$), and for glycogen depletion 3.23 ± 0.49 ($L*min^{-1}$) (Figure 7).

The total, aerobic, anaerobic work for each trial is presented in Figure 8 for the last 200m segment of each trial. There were no significant differences in total or aerobic work among conditions. However, anaerobic work was significantly greater in the bicarbonate trial compared to the glycogen depletion trial. This result is particularly evident when the relative proportion of energetic contribution is evaluated (Figure 9).

Table 4. VO_2 (l/min) (+/- SD) for the Control, Sodium Bicarbonate, and Glycogen Depletion Time Trials

	Control	Bicarb	GD
100	1.31 ± 0.15	1.24 ± 0.10	1.37 ± 0.16
300	2.42 ± 0.27	2.53 ± 0.35	2.27 ± 0.66
500	3.20 ± 0.37	3.17 ± 0.40	2.88 ± 0.90
700	3.31 ± 0.42	3.33 ± 0.49	3.05 ± 0.58
900	3.36 ± 0.46	3.38 ± 0.51	3.19 ± 0.62
1100	3.34 ± 0.46	3.41 ± 0.55	3.26 ± 0.46
1300	3.29 ± 0.44	3.35 ± 0.58	3.28 ± 0.50
1500	3.34 ± 0.44	3.38 ± 0.60	3.23 ± 0.49

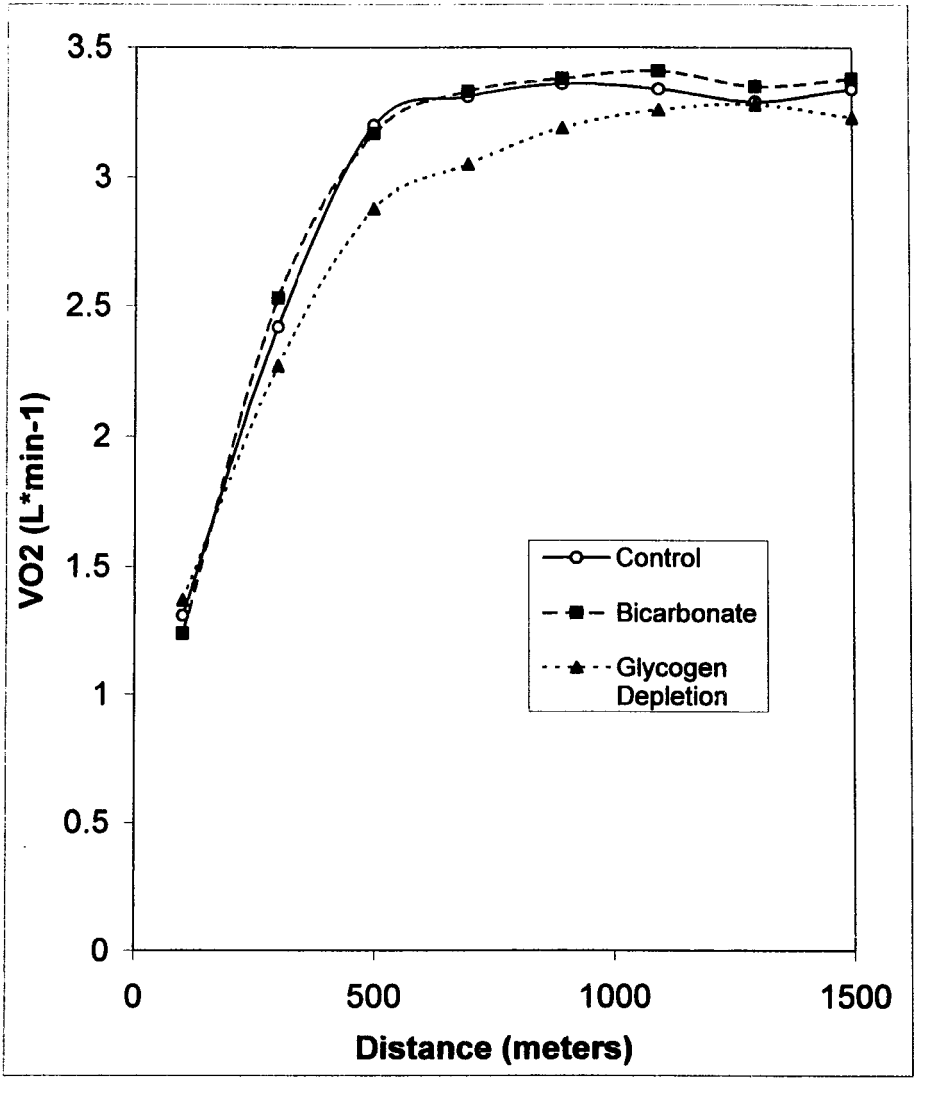


Figure 7. Mean VO_2 during 1500m Time Trial Measured every 200m

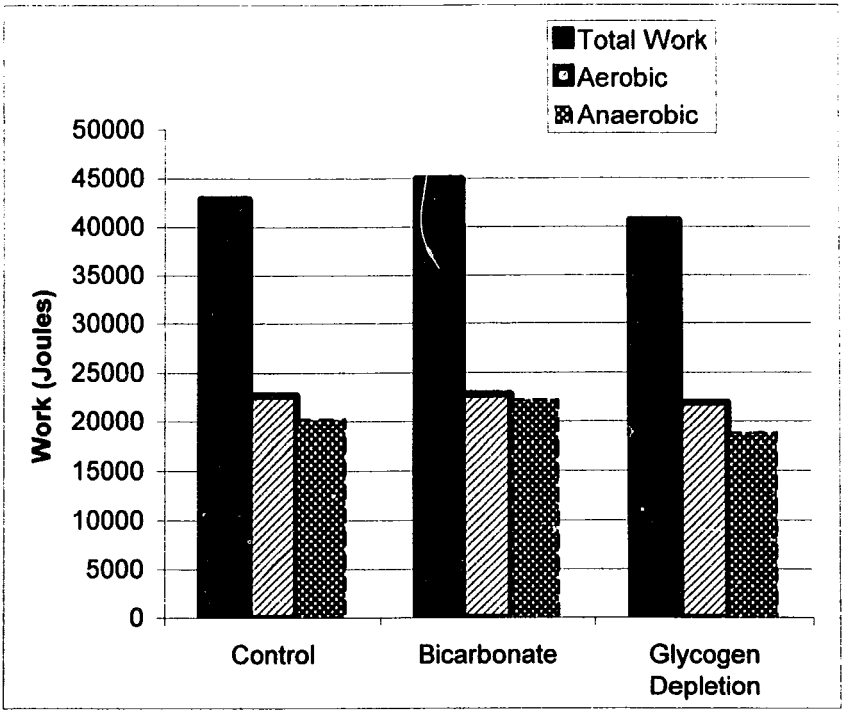
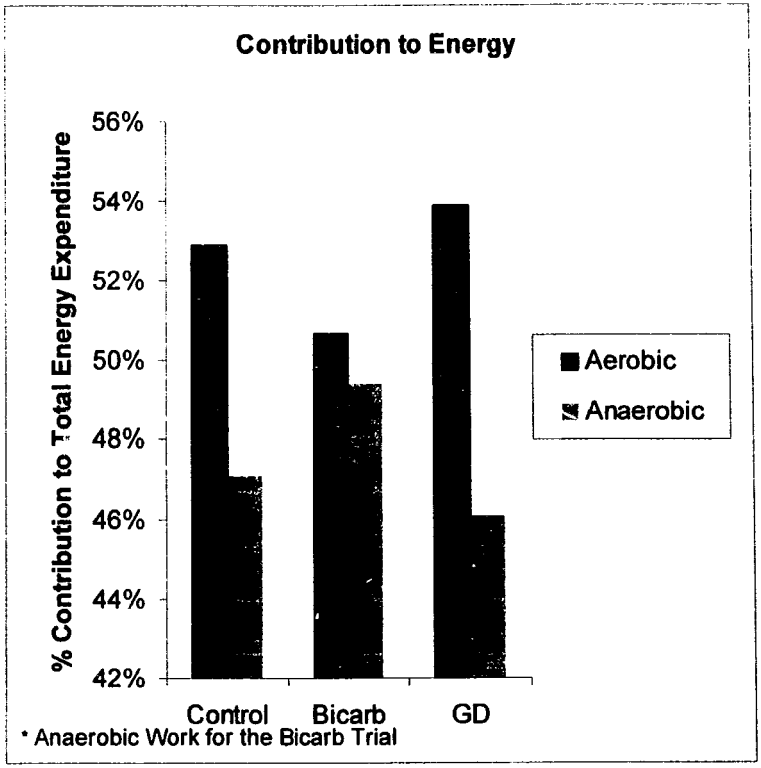


Figure 8. Mean Work for Each Trial: Control, Sodium Bicarbonate, and Glycogen Depletion



* $p < 0.05$

Figure 9: Percentage of Contribution to Energy for each Trial: Control, Sodium Bicarbonate, and Glycogen Depletion

DISCUSSION

In general, the present findings support our hypothesis that energy expenditure in the glycogen depletion (GD) and sodium bicarbonate (bicarb) trials would vary in relation to the changes in anaerobic energy expenditure. The results demonstrated a significant reduction in anaerobic energetic contribution in the glycogen depleted state, which is consistent with expectations if the glycogenolytic energy system if restricted by lack of substrate. The tendency (although not statistically significant) for a greater anaerobic energetic contribution in the presence of a buffering agent is, likewise, consistent with expectations if proton accumulation secondary to muscle lactate accumulation is somehow related to muscle fatigue. Particularly within the context of a progressive increase in anaerobic work accomplished from glycogen depletion to control to bicarbonate, the overall pattern of the results supports our hypothesis. This particular approach toward evaluating anaerobic energy expenditure has not previously been published. Preliminary data by Zajakowski et al. [26], using the conceptually similar accumulated oxygen deficit technique, revealed similar results. A variety of other studies using bicarbonate have demonstrated not only improved performance but also, increased peak exercise lactate concentrations [17]. Although post-exercise blood lactate accumulation is not in of itself proof of a greater anaerobic energetic contribution, it is consistent with the expectations if anaerobic energetic contribution is increased.

Some limitations to this study were the lack of blood lactate analysis. Further, we assumed glycogen depletion without measuring muscle glycogen concentrations. In a

similar test, Zajakowski et al. [26] observed a reduced accumulated oxygen deficit under similar exercise and dietary circumstances that should have produced muscle glycogen depletion. The protocol consisted of a low dietary carbohydrate intake and heavy exercise preceding the day of the trial.

Another limitation of the study is the small amount of sodium bicarbonate ingested by the subjects. Most investigators have observed that bicarbonate is effective in doses of 300mg/kg body weight. However, to prevent the gastrointestinal discomfort typically associated with bicarbonate ingestion, the dosage was reduced to150mg/kg body weight. It seems reasonable to speculate that this dosage of bicarbonate may have been inadequate to improve performance.

Many of the subjects complained of dyspnea during the bicarbonate trial. We speculate that the increased VCO_2 during this trial (a natural consequence of augmented buffering) may have resulted in an enhanced end tidal CO_2, resulting from the presence of the respiratory apparatus. This may have caused dyspnea by creating an effective increase in F_ICO_2. In a situation where the respiratory apparatus was not used, performance might have reasonably been expected to enhance performance.

The last assumption made was the exclusive use of carbohydrates for energy production during the tests. From the submaximal ride we determined that a constant efficiency was determined at any point along the ride through the measurements of total power output (by the SRM) and VO_2. In order to quantify anaerobic capacity we subtracted aerobic work (VO_2) from mechanical work (total power output from SRM) to

get anaerobic work. Then, we estimated the anaerobic metabolic rate to a RER of 1.0. We assumed glycogen depletion on the basis that glycogen was the sole contributor to the respiratory exchange ratio (RER). It is generally accepted that work at 0.70 RER is classified as fat metabolism and 1.00 as CHO metabolism. Less than 2% of energy metabolism comes from protein in exercise bouts <1hr [15,23]. By ruling out the use of protein and fat use to provide energy, we assumed the cyclists' RER was closer to 1.00 (VCO_2/VO_2) [10]. Therefore carbohydrates were considered to be the main energy source for forming ATP during the time trials. Garby and Astrup [10] came up with a linear and unique way to show oxidation of glucose, lipids, and synthesis of lipids from glucose. Their research showed linear and unique relationships between energy equivalents of O_2 and non-protein respiratory quotients. Replicating this study with higher doses of bicarbonate might be beneficial to better evaluate its use.

In summary, the results of this study support the concept that experimental manipulation of the anaerobic glycolytic energy system either by glycogen depletion or bicarbonate administration can influence high intensity cycling performance. Overall total power output was increased in the bicarbonate trial, which supported the hypothesis that sodium bicarbonate is an effective means of enhancing performance and anaerobic energy contributions in high intensity exercise bouts.

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APPENDIX A

INFORMED CONSENT

EFFECT OF SODIUM BICARBONATE AND GLYCOGEN DEPLETION ON 1500m TIME TRIALS

Under Pacing Strategy in Cyclists: Test of a Model

1. INFORMED CONSENT FOR "Pacing Strategy in Cyclists: Test of a Model"

2. I, _____, give my informed consent to participate in this study designed to evaluate performance during cycling time trials in relation to pacing strategies identified by a mathematical model of human energy expenditure. I have been informed that the project is under the direction of Carl Foster, Ph.D., who is a Professor in the Department of Exercise and Sport Science at the University of Wisconsin-La Crosse, although other professional persons or students may act for him. I consent to the presentation, publication and other release of the summary data from the study which is not identifiable with myself.

3. I have been informed that my participation in this study will involve my completing several visits to the Human Performance Laboratory (MH 225) at UW-La Crosse. During each of these visits I will either complete an incremental exercise test on a cycle ergometer, a training session on the cycle or a cycle time trial. During the time trial, I will ride according to directions provided by the investigators during the first part of the trial, then complete the trial as rapidly as possible. During the trials my metabolic rate will be measured and I may have small samples of blood obtained from my fingertip.

4. I have been informed that the known or expected discomforts to be expected are fatigue from the exercise tests or training and sore fingers from the blood sampling. I have been informed that the risk of complications during exercise test in patients suspected of having heart disease is about 6/10,000 tests for minor complications and 1/10,000 tests for serious complications (e.g. cardiac arrest). For prospectively healthy, athletic individuals the risk is thought to be much less (approximating zero), but is less well documented simply because complications are so rare.

5. I have been informed that the primary benefit that I might expect from participating in this study is a better understanding of my performance characteristics and guidelines that may help me individualize my training.

6. I have been informed that there are no "disguised" procedures in the study. All procedures can be taken at face value.

7. I have been informed that the investigator will answer questions regarding the procedures throughout the course of the study.

8. I have been informed that I am free to withdraw from the study at any time without penalty.

9. Concerns about any aspects of the study may be referred to Dr Foster at 608 785 8687 (work) or 608 796 9959 (home). Questions regarding the protection of human subjects may be addressed to Dr Dan Duquette, chair of the UW-La Crosse Institutional Review Board at (608) 785 8161

Investigator_____ Participant_____ Date_____

Signature_____Signature _____ Date_____

APPENDIX B

DATA RECORDING SHEET

Name
Birthday
Height
Weight
Activity
Sprinter/endurance
ID
Glyc/Bicar

Name
Birthday
Height
Weight
Activity
Sprinter/endurance
ID
Glyc/Bicar

Name
Birthday
Height
Weight
Activity
Sprinter/endurance
ID
Glyc/Bicar

Name
Birthday
Height
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Activity
Sprinter/endurance
ID
Glyc/Bicar

APPENDIX C

REVIEW OF RELATED LITERATURE

REVIEW OF RELATED LITERATURE

Introduction:

Athletes depend on metabolic ATP production for accomplishing muscular tasks. Without replenishment of ATP, muscular contraction stops. The body depends on three interlocked energy systems. Factors that come into play are the general interactions of the relative contribution from each energy system. The three energy systems consist of the anaerobic phosphagen system, anaerobic lactate system and the aerobic energy system.

Anaerobic Phosphagen System "Immediate Anaerobic Energy"

The energy processes ATP for immediate use include events lasting 1-30s. The formation of ATP occurs due to the breakdown of phosphocreatine (PC). Phosphocreatine's role, is to defend ATP concentrations against sudden increases in energy demand [5]. Total depletion of ATP does not occur even in extreme exercise conditions. . According to Jacob et al. [11] on a 30s Wingate test, a thirty to forty percent decrease in ATP has been reported in high intensity exercise [1,11]. However, depletion of PC to 40% of resting concentrations is possible during exhaustive exercise of 1-3min duration.

Phosphocreatine combines with ADP, which resynthesizes in ATP and Creatine due to the catalyzing enzyme creatine kinase. The limitations of the muscles' ability to store PC inhibit the amount of ATP formation. This is called the ATP-PC system or

"phosphagen system" and provides energy for muscular contraction at the onset of exercise and during short term high intensity exercise <5s (eg. 50 yd dash). The ATP-PC system provides a simple one-enzyme reaction to produce ATP for such activities (sprinting). PC reformation requires ATP and occurs only during recovery [17].

The Phosphagen system produces the fastest ATP production but all three energy systems are activated with the onset of exercise. As far as the availability of ATP and PC during heavy or intense exercise, fatigue during intense repeated exercise does not appear to be related to the lack of energy. Bangsbbo et al [2] showed such results by demonstrating that muscle ATP rarely falls below 60% of pre-exercise levels during exhaustive voluntary exercise and even in highly fatigued fibers the ATP concentration is over 100 fold higher than the micromolar amounts required for peak force [4]. For trained athletes glycogen availability is increased, which means more ATP can be produced more rapidly than in untrained subjects [18]. Saltin [18] states that with training there is an increase in both the highest oxidative capacity fibers (red fibers) and in the lowest oxidative capacity fibers (white fibers). Enzyme activities in well trained athletes are higher as well causing an increase in oxidation SDH in the mitochondria, and PFK phosphofructose kinase (glycolytic enzyme). As athletes perform ATP-PC provides ATP as fast and as long as possible while at the same time the more involved the glycogenolytic energy system is increasing its involvement

Anaerobic Lactate System

Since ATP-PC can only sustain energy for <20s and re-synthesis does not take place until after several minutes of rest, other energetic sources are required [18]. The anaerobic lactate system depends on the availability of muscle glycogen. Typically, this system provides energy for 30s-3min of work. It works by the anaerobic degradation of carbohydrates (CHO), glucose or glycogen to either lactate or pyruvate. The lactic acid formation prevents pyruvate accumulation from acting to inhibit glycogenolysis. Pyruvate provides the link into aerobic oxidation the third energy system.

High ratios of anaerobic capacity producing ATP, results in the accumulation of lactate in the muscle and the blood, which contributes to fatigue. The accumulation of lactic acid along with the depletion of ATP-PC and glycogen bring an exercise bout to an end or the need to decrease work intensity [5]. Some of the limiting factors for anaerobic systems are lack of glycogen, lack of ATP production due to a decrease in PC available for immediate energy. Limiting ATP due to inhibition of glycolytic enzymes or lack of activation of glycolysis [10]. A gradual decrease in pH may cause these limiting factors or reduced energy demand. This may be due to inhibition in motor neuron, changes in activation of force-generating capacity and ability to load and release calcium in the sacroplasmic recticulum [4, 8]. Medbo et al [13, 14] hypothesized that anaerobic capacity was a well defined individual entity. The method for determining this factor (anaerobic capacity) during exhaustive exercise was through maximal accumulated oxygen deficit (MAOD) technique. Oxygen deficit has been used as a measure of anaerobic energy yield, as by definition the difference between the energy

supplied by the aerobic energy system and the total energy demand of work. Those who are trained in sprints and power events tend to perform significantly better on short duration high intensity events than endurance trained or sedentary individuals [6, 21].

Longitudinal studies reported improved anaerobic power and capacity performance following sprint training [3, 9]. Sprint training has been shown to increase buffer capacity [19, 20] glycolytic and oxidative enzyme concentration and lactate accumulation [12, 15].

Aerobic Energy System

The aerobic energy system is the primary energy system in events lasting longer than 2min. Oxidation occurs in the mitochondria and involves two processes; the Krebs cycle and Electron Transport Chain to produce energy. The Krebs cycle oxidation requires CHO, fats, and protein as energy sources to fuel the body of ATP. The Krebs cycle provides 4 times the amount of ATP generated by the anaerobic lactate system. During the Krebs cycle NADH and FADH are formed which are sent to the electron transport chain. It contributes to producing energy by using oxygen as a H+ acceptor to form ATP and water. Even though the aerobic system primarily for energy production in bouts >2min, 50% of energy during 60s exercise bouts is produced by the aerobic energy system [6,14] in supramaximal exercise bouts. Increasing the blood supply to the muscle is necessary to allow aerobic production of ATP. The increase in blood supply also assists lactate efflux from the muscle, which potentially results in increasing ATP production for anaerobic glycolysis before critical pH levels are reached [16].

Estimation of accumulated oxygen deficit (AOD) involves establishing a linear relationship between steady state oxygen consumption (VO_2) and submaximal power output for each subject [22].

For athletes trained for endurance exercise, tests resulted in longer periods of aerobic utilization. For sprinters the opposite occurs and anaerobic utilization is prolonged with any given test [7]. Training specificity plays an important role in attaining aerobic and anaerobic measures for a given test. However, VO_2 should be similar where VO_2peak will vary [7].

In trained athletes the amount of hemoglobin in the blood available is greater than untrained athletes. Therefore, VO_2max will be increased, VO_2peak will be higher, and the time at which variables occur will be at higher workloads and intensities than an untrained individual.

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