Human muscle metabolism during intermittent maximal exercise

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Gaitanos, G. C., C. Williams, L. H. Boobis, and S. Brooks. Human muscle metabolism during intermittent maximal exercise. J. Appl. Physiol. 75(2): 712-719, 1993.—Eight male subjects volunteered to take part in this study. The exercise protocol consisted of ten 6-s maximal sprints with 30 s of recovery between each sprint on a cycle ergometer. Needle biopsy samples were taken from the vastus lateralis muscle before and after the first sprint and 10 s before and immediately after the tenth sprint. The energy required to sustain the high mean power output (MPO) that was generated over the first 6-s sprint (870.0 ± 159.2 W) was provided by an equal contribution from phosphocreatine (PCr) degradation and anaerobic glycolysis. Indeed, within the first 6-s bout of maximal exercise PCr concentration had fallen by 57% and muscle lactate concentration had increased to 28.6 mmol/kg dry wt, confirming significant glycolytic activity. However, in the tenth sprint there was no change in muscle lactate concentration even though MPO was reduced only to 73% of that generated in the first sprint. This reduced glycogenolysis occurred despite the high plasma epinephrine concentration of 5.1 ± 1.5 nmol/l after sprint 9. In face of a considerable reduction in the contribution of anaerobic glycogenolysis to ATP production, it was suggested that during the last sprint, power output was supported by energy that was mainly derived from PCr degradation and an increased aerobic metabolism.

METHODS

The exercise test was carried out on an instrumented friction-loaded cycle ergometer (model 864, Monark) that allowed the instantaneous power output, corrected for flywheel acceleration, to be monitored and recorded precisely. The design of this equipment has been described in detail elsewhere (16). To calculate the power output generated during maximal exercise, it was assumed that the external mechanical work performed on this type of cycle ergometer was equal to the work dissipated against braking forces (i.e., inertia of the flywheel, rolling resistance of the flywheel, and freewheel mechanism) plus the kinetic energy of the flywheel. Thus the “effective load” on the cycle ergometer was determined. This is the load required to prevent acceleration of the flywheel, and it not only consisted of the frictional load that was applied to the flywheel (65 g/kg body wt) but also took into account the frictional load that would be required to stop the subject from accelerating the flywheel at any instant (16).

The instantaneous product of flywheel speed and effective load on the cycle ergometer was equal to the work dissipated against braking forces (i.e., inertia of the flywheel, rolling resistance of the flywheel, and freewheel mechanism) plus the kinetic energy of the flywheel. Thus the “effective load” on the cycle ergometer was determined. This is the load required to prevent acceleration of the flywheel, and it not only consisted of the frictional load that was applied to the flywheel (65 g/kg body wt) but also took into account the frictional load that would be required to stop the subject from accelerating the flywheel at any instant (16).

The instantaneous product of flywheel speed and effective load was used to determine corrected power output throughout each exercise period. After the final sprint, results were integrated over 1-s time intervals by computer using the trapezoid rule. The maximum 1-s integral of effective load and flywheel speed, during each sprint, is referred to as the “peak power output” (PPO).
The average 1-s integral of effective load and flywheel speed, over the total time period of each exercise period (i.e., 6 s), is referred to as "mean power output" (MPO). Each sprint was initiated from a stationary start because of the increased accuracy and ease with which the starting point could be identified by computer.

**Subjects.** Eight healthy male physical education students, whose mean age, height, and weight were 26.7 ± 8.4 (SD) yr, 175.6 ± 11.4 cm, and 71.8 ± 11.4 kg, respectively, gave their informed consent and volunteered to take part in this study, which was approved by the University Ethical Committee.

**Protocol.** All subjects were familiarized with sprint cycling until fully confident of producing an all-out effort from a stationary start. They were instructed to refrain from any form of intense physical exercise on the day before testing. On the day of the experiment, subjects reported to the laboratory after a fast of at least 4 h. They were weighed and then sat quietly on an examination couch so that resting venous blood and muscle biopsies could be obtained. The subjects then completed a period of low-intensity exercise that consisted of stretching and submaximal cycling for 5 min at 50 rpm against a resistance of 0.5 kg. This was followed by a standardized warm-up that involved two 30-s periods of submaximal cycling at 85 and 115 rpm against a resistance of 1.5 kg. These bouts of cycling were separated by 60 s of recovery and served to reacclimate the subjects to the experimental procedures. This warm-up has previously been shown to result in only minor metabolic disturbances (30). After 5 min of stretching after this standardized warm-up, subjects performed the exercise test from a stationary start. Subjects were instructed to remain seated during all sprints; their feet were secured to the pedals using toe clips. During the subsequent 30-s rest period, after each sprint, they remained quietly seated on the cycle ergometer. Each sprint was initiated from the same starting pedal position. Data logging by the computer was initiated 2 s before the subject was instructed to begin sprinting to avoid timing errors that might have arisen had the subject started too early. Subjects were able to stop pedaling almost instantaneously, although the cycle ergometer flywheel would then continue to revolve for a few moments. All subjects were highly motivated and were instructed to cycle maximally from the start of the test; they were all verbally encouraged throughout each 6-s sprint. After the last sprint the subjects rested for 10 min on an examination couch adjacent to the cycle ergometer.

On a separate day, maximum oxygen uptake (\( \dot{V}O_{2\max} \)) was determined using a graded cycling test. The first work load was high enough to ensure that exhaustion would occur within 6–12 min, the load being increased by 0.5 kg at the end of every 3 min. Expired air samples were collected during the final minute of each load increment and at the last 60 s of exercise. Heart rate was monitored continuously by cardiac oscilloscope during all the exercise tests and was visible to the experimenters throughout the tests.

**Blood samples.** Venous blood samples were taken from an antecubital vein by using an intravenous cannula (16 gauge, Venflon) fitted with an injection diaphragm. The cannula was inserted under local anesthesia (0.5 ml of 1% lidocaine), and patency was maintained by displacing the blood contained within the cannula with isotonic saline containing 100 U/ml of heparin. Blood samples were drawn at rest with the subjects in a recumbent position; immediately after the first, fifth, ninth, and tenth sprints; and at 3, 5, and 10 min after completion of the last sprint (Fig. 1). The volume of blood taken for each sample in the above procedure was 10 ml and was placed in calcium-heparinized tubes to prevent coagulation. Blood pH was determined immediately after collection on a pH-blood gas analyzer (model BMS3 Mk2, Radiometer). The percent change in plasma volume was estimated from the resting and postexercise hematocrit and hemoglobin values (4). In addition 20 μl of venous blood were collected and immediately deproteinized in 200 μl of 2.5% perchloric acid. The precipitate was separated by centrifugation. These samples were then frozen at −70°C and analyzed fluorimetrically (model 8-9, Locarte) for blood lactate concentration (22) and photometrically for blood glucose (29). The remaining blood (8–9 ml) was centrifuged at 6,000 rpm for 15 min at 3°C (Koolspin, Buckhard). The plasma obtained was treated with 200 μl of an oxidizing and chelating agent that was made up of 100 mmol/l ethylene glycol-bis(β-amino ethyl ether)-N,N,N',N'-tetraacetic acid and 100 mmol/l reduced glutathione. The treated plasma was stored at −20°C and analyzed at a later date for epinephrine and

![FIG. 1. Schematic illustration of study protocol. rec, recovery; post, postexercise.](image-url)
norepinephrine by using high-performance liquid chromatography with electrochemical detection (3).

Muscle samples. Muscle biopsy samples were taken under local anesthesia (2 ml, 1% lidocaine) at rest before warm-up and immediately after the first sprint from one leg and 10 s before and immediately after the last sprint from the other leg (Fig. 1). The order of sampling was randomly assigned. The biopsies were obtained by a percutaneous needle biopsy technique (1) with suction being applied. The sampling site selected for the muscle biopsies was the lateral portion of the quadriceps muscle (vastus lateralis). This muscle was selected because it has been shown to be heavily involved when knee extensions are performed during maximal cycling and is easily accessible with minimal risks. Muscle biopsies were taken within 3 s from cessation of the sprints and were immersed in liquid nitrogen within 2 s of sampling. The samples were then left to dry, weighed, and stored at -70°C before and after the muscle was obtained (9). The neutralized extract was assayed enzymatically for PCr, ATP, ADP, AMP, creatine (Cr), glucose, glucose-1-phosphate (G-1-P), glucose-6-phosphate (G-6-P), fructose-6-phosphate (F-6-P), fructose-1,6-diphosphate (F-1,6-DP), triose phosphates (TP), pyruvate, lactate, and the acid-soluble fraction of glycogen. The determination of all these muscle metabolites involved fluorometric analysis (18). The acid-insoluble fraction of glycogen was also determined on the precipitated muscle pellet after the extraction procedure by prior hydrolysis in 1 M HCl. The reference base for muscle metabolite concentrations is dry weight to avoid changes in concentration due to water shift during exercise. In addition, the development of PPO generated during the early stages of each sprint did not change with successive bouts of exercise. The time taken to achieve MPO in all sprints was within the first 2 s of exercise.

Power output. The MPO and PPO values recorded during each of the ten 6-s sprints are shown in Fig. 2. The PPO generated during the first sprint (1,253.3 ± 334.8 W) was approximately five times greater than that which elicited the subjects' Vo2max (253.6 ± 57.8 W), indicating the maximal intensity of the test. The highest mean power was reached in the first sprint (870.1 ± 159.2 W), after which there was a decline in power output. The highest mean power was reached in the first sprint (870.1 ± 159.2 W), after which there was a decline in power output. At the fifth and final sprints MPO was decreased by 12.6% (P < 0.01) and 26.6% (P < 0.01) of the maximal value, respectively. It was calculated that 47.5% of the fall in MPO over the 10 sprints occurred in the first 5 sprints. Moreover, the fall in MPO was significantly less (P < 0.01) from sprints 4–10, indicating that only four 6-s bouts of maximal cycling is demanding enough to elicit fatigue-induced decrements in performance with 30 s of recovery. The decrease in PPO after 5 and 10 sprints averaged 15.9% (P < 0.05) and 33.4% (P < 0.01), respectively. It is also of interest to note that the amount of work completed over the first five sprints was 54.5% of the total work done over the entire sprint test (44,760 ± 7,610 J). In addition, the development of PPO generated during the early stages of each sprint did not change with successive bouts of exercise. The time taken to achieve PPO in all sprints was within the first 2 s of exercise.

Blood lactate and blood pH. The first sprint resulted in a small increase in blood lactate concentration (2.5 mmol/l), but after sprint 5 lactate concentration had increased 15-fold from 0.6 ± 0.1 mmol/l at rest to 9.2 ± 1.5 mmol/l (P < 0.01). After completion of sprint 9, blood lactate had increased further to 12.6 ± 1.5 mmol/l (P < 0.01) and did not change during the 10 min of postexercise recovery (Fig. 3). A strong correlation (r = 0.88, P < 0.05, n = 6) was found between the increase in blood lactate at the end of sprint 5 and the total amount of work done over the first five sprints. The relationship for the second five sprints, during which 45.5% of the total work done was performed, was not as strong. However, those individuals who showed the greatest fall in MPO after 10 sprints tended to have the largest changes both in blood lactate at 5 min postexercise (r =
values, also produced the largest change in blood lactate concentration at 3 min postexercise ($r = 0.944, P < 0.01, n = 6$).

**Blood glucose.** Blood glucose increased from $3.9 \pm 0.2$ mmol/l at rest to $4.6 \pm 0.5$ mmol/l after nine sprints ($P < 0.01$) and continued to rise until 5 min into the recovery period ($5.6 \pm 0.6$ mmol/l, $P < 0.01$).

**Plasma catecholamines.** The first sprint resulted in a fivefold increase in plasma epinephrine concentration from $0.4 \pm 0.2$ nmol/l at rest to $1.9 \pm 1.0$ nmol/l ($P < 0.05$) and in a twofold increase in plasma norepinephrine from $1.7 \pm 0.4$ nmol/l at rest to $3.3 \pm 1.1$ nmol/l. Both plasma epinephrine and norepinephrine concentrations increased after sprint 5 to $4.2 \pm 1.4$ nmol/l ($P < 0.01$) and $15.7 \pm 2.7$ nmol/l ($P < 0.01$), respectively. The peak values occurred after completion of sprint 9, representing a 13-fold increase from resting values in epinephrine to $5.1 \pm 1.5$ nmol/l ($P < 0.01$) and a 13-fold increase in norepinephrine to $22.3 \pm 6.5$ nmol/l ($P < 0.01$). The norepinephrine concentration remained above the preexercise value throughout the recovery period ($P < 0.01$), whereas epinephrine concentrations were not different from resting after the immediate postexercise sample (Fig. 5).

**Change in plasma volume.** The exercise test resulted in an average decrease in plasma volume of $12.1 \pm 4.5\%$ ($P < 0.01$). The changes in the concentrations of blood lactate, glucose, and plasma catecholamines, in response to the exercise test, were severalfold higher than those in plasma volume. Therefore, changes in blood and plasma metabolites cannot be explained by changes in plasma volume.

**Muscle metabolites.** The muscle metabolite concentrations at each sampling time are shown in Table 1. After the first sprint there was a 57% fall in PCR concentration ($P < 0.01$), which was accompanied by a 13% fall in ATP ($P < 0.01$). No accompanying changes were observed in ADP and AMP concentrations. Total glycogen concentration decreased by 14% ($P < 0.01$) after sprint 1, whereas there were dramatic increases ($P < 0.01$) in G-6-

![FIG. 4. Blood pH during exercise and recovery. Values are means ± SD; $n = 7$ except as indicated.](image)

![FIG. 5. Plasma norepinephrine and epinephrine concentrations during exercise and recovery. Values are means ± SD; $n = 7$ except as indicated.](image)

### TABLE 1. Muscle metabolites before and after first and last 6-s sprint of multiple sprint test on cycle ergometer

<table>
<thead>
<tr>
<th>Metabolites</th>
<th>Pre-1st 6 s ($n = 8$)</th>
<th>Post-1st 6 s ($n = 8$)</th>
<th>Pre-10th 6 s ($n = 7$)</th>
<th>Post-10th 6 s ($n = 7$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycogen</td>
<td>316 ± 87.4</td>
<td>273 ± 79.3</td>
<td>221 ± 90.3</td>
<td>201 ± 90.1</td>
</tr>
<tr>
<td>Acid-soluble glycogen</td>
<td>26 ± 8.15</td>
<td>14 ± 18.1</td>
<td>7 ± 15.2</td>
<td>5 ± 12.6</td>
</tr>
<tr>
<td>Acid-insoluble glycogen</td>
<td>290 ± 27.1</td>
<td>259 ± 68.3</td>
<td>214 ± 76.2</td>
<td>196 ± 78.4</td>
</tr>
<tr>
<td>ATP</td>
<td>24 ± 2.7</td>
<td>20 ± 2.5</td>
<td>16 ± 2.4</td>
<td>14 ± 2.8</td>
</tr>
<tr>
<td>ADP</td>
<td>3 ± 0.3</td>
<td>3 ± 0.6</td>
<td>2 ± 0.4</td>
<td>3 ± 0.5</td>
</tr>
<tr>
<td>AMP</td>
<td>0.1 ± 0.0</td>
<td>0.1 ± 0.0</td>
<td>0.1 ± 0.0</td>
<td>0.1 ± 0.0</td>
</tr>
<tr>
<td>PCr</td>
<td>73.6 ± 3.7</td>
<td>73.6 ± 22.6</td>
<td>37.5 ± 15.0</td>
<td>12.2 ± 5.7</td>
</tr>
<tr>
<td>Cr</td>
<td>43.5 ± 4.8</td>
<td>43.5 ± 12.6</td>
<td>81.9 ± 13.1</td>
<td>107.5 ± 15.0</td>
</tr>
<tr>
<td>Glucose</td>
<td>1.4 ± 0.4</td>
<td>2.1 ± 1.0</td>
<td>7.9 ± 1.9</td>
<td>8.2 ± 1.3</td>
</tr>
<tr>
<td>G-1-P</td>
<td>0.4 ± 0.1</td>
<td>0.7 ± 0.4</td>
<td>0.8 ± 0.6</td>
<td>0.8 ± 0.5</td>
</tr>
<tr>
<td>G-6-P</td>
<td>0.6 ± 0.2</td>
<td>0.9 ± 0.2</td>
<td>5.7 ± 1.8</td>
<td>5.9 ± 1.0</td>
</tr>
<tr>
<td>F-6-P</td>
<td>0.2 ± 0.2</td>
<td>0.3 ± 0.1</td>
<td>0.3 ± 0.2</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>TAT</td>
<td>0.2 ± 0.2</td>
<td>0.2 ± 0.2</td>
<td>0.2 ± 0.2</td>
<td>0.2 ± 0.2</td>
</tr>
<tr>
<td>F-1,6-16-DP</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.4 ± 0.3</td>
<td>0.5 ± 0.1</td>
</tr>
<tr>
<td>Lactate</td>
<td>2.8 ± 1.1</td>
<td>2.8 ± 5.7</td>
<td>11.2 ± 5.9</td>
<td>11.2 ± 5.0</td>
</tr>
<tr>
<td>Pyruvate</td>
<td>0.6 ± 0.3</td>
<td>0.6 ± 0.3</td>
<td>1.6 ± 0.3</td>
<td>1.6 ± 0.3</td>
</tr>
</tbody>
</table>

Values are means ± SD given in mmol glucosyl units/kg dry wt for glycogen and mmol/kg dry wt for other metabolites; $n$, no. of subjects; PCr, phosphocreatine; Cr, creatine; G-1-P, glucose 1-phosphate; G-6-P, glucose 6-phosphate; F-6-P, fructose 6-phosphate; TP, triose phosphate; F-1,6-16-P, fructose 1,6-diphosphate. * Significant difference from previous sample, $P < 0.01$; ** significant difference from previous sample, $P < 0.05$; † significant difference from post-1st 6 s, $P < 0.01$; ‡ significant difference from preexercise, $P < 0.01$; § significant difference from preexercise, $P < 0.05$. 

There was only a small change in blood pH by the end of the first sprint ($pH = 0.02$). However, it decreased by 0.27 unit (from $7.37 ± 0.03$ at rest to $7.10 ± 0.03$) by the end of the exercise test ($P < 0.01$) and remained low for the first 3 min of passive recovery (Fig. 4). During the subsequent 7 min of recovery blood pH increased to $7.16 ± 0.07$ ($P < 0.01$), indicating that the time course of the recovery during this period was different from that for blood lactate. The decrease in blood H+ concentration occurred at a faster rate than the decrease in lactate after exercise. Those subjects who exhibited the greatest change in pH immediately after exercise, from resting
TABLE 2. Estimates of ATP production from anaerobic sources during first and final 6-s sprint of exercise test

<table>
<thead>
<tr>
<th></th>
<th>ATP Production, mmol/kg dry wt</th>
<th>ATP Production Rate, mmol·kg dry wt(^{-1})·s(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sprint 1 (n = 8)</td>
<td>Sprint 10 (n = 7)</td>
</tr>
<tr>
<td>Total</td>
<td>80.3±13.8</td>
<td>31.6±14.7</td>
</tr>
<tr>
<td>From glycolysis</td>
<td>39.4±9.5</td>
<td>5.1±8.9</td>
</tr>
<tr>
<td>From PCr</td>
<td>44.3±4.7</td>
<td>25.3±9.7*</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. * Significant difference from sprint 1, \(P < 0.01\).

P (1,500%), F-6-P (1,100%), and lactate (653%) concentrations. There was a similar change \((P < 0.01)\) in F-1,6-DP (600%) but a much more modest increase \((P < 0.01)\) in muscle glucose content (80%) and G-1-P concentration (75%). Before the last sprint, the 32% decrease in ATP concentration was accompanied by a 30% fall in glycogen \((P < 0.01)\). F-6-P and F-6-P were now only nine times greater than their preexercise values, whereas F-1,6-DP had almost fallen to resting levels. In contrast, there was a further threefold increase in muscle glucose and a further fourfold increase in lactate compared with the values observed after the first sprint. Although the glycogen concentration fell a further 6% during the last sprint (NS), there was almost no change in the concentration of ATP, the glycolytic intermediates, glucose, or lactate. The PCr concentration before sprint 10 was 51% lower than the resting value and fell to 16% during this last bout of exercise. The ADP and AMP concentrations, by comparison, remained unchanged. Similarly, after the initial modest increase in TP and pyruvate \((P < 0.01)\) observed after the first sprint, the concentration of these metabolites was maintained constant during the last sprint. The inosine 5'-monophosphate content was not determined in this study, but it has been shown to increase by an amount equal to the sum of the decreases in the concentrations of ATP + ADP + AMP (25).

ATP production. Although some lactate efflux from muscle probably occurred during each 6-s sprint, the total minimum ATP production from anaerobic sources during the first and final sprints was considered to be equal to the sum of \(\Delta\text{PCr} + \Delta\text{ATP} + 1.5 \times \Delta\text{lactate} + 1.5 \times \Delta\text{pyruvate}\) as has been previously described (27). The ATP production from glycolysis was estimated from the changes in lactate and pyruvate using the formula \(1.5 \times \text{lactate} + 1.5 \times \text{pyruvate}\). The results of these calculations are shown in Table 2. Glycolysis contributed 44% of the ATP produced during the first sprint with the remainder being derived predominantly from PCr degradation. In contrast, during the last sprint anaerobic ATP production was almost entirely supplied from PCr (80%). Despite an apparent average 20% contribution from glycolysis to ATP production in the last sprint (Fig. 6), it should be noted that in four subjects this was estimated to be zero (range 0–23.1 mmol ATP/kg dry wt, \(n = 7\)).

Glycogenolytic and glycolytic rates. The minimum anaerobic glycogenolytic and glycolytic rates were estimated using the formulas described by Hultman and Sjoholm (12), as follows: glycogenolysis = \(-\Delta G\ 6\ P\ + \ (0.33 \times \Delta G\ 6\ P\) + \(0.5 \times (\Delta\text{lactate} + 0.1 \times \Delta\text{lactate})\); glycolysis = \(0.5 \times (\Delta\text{ATP} + 0.1 \times \Delta\text{ATP})\). Increases in intracellular glucose have not been included in the calculation of glycogenolysis because the free glucose in muscle has probably been released by the action of the debranching enzyme and not the action of phosphorylase. The results of these calculations are shown in Table 3. In the first sprint anaerobic glycogenolysis had exceeded glycolysis by 91% with the rate of total glycogen breakdown surpassing the rate of anaerobic glycogenolysis by almost 65%. After 10 sprints, there was an 11-fold decrease in glycogenolysis and an 8-fold decrease in glycolysis, although the total glycogen breakdown had only fallen by 37%.

Interestingly, those individuals who had the greatest fall in MPO over the ten 6-s sprints were found to have the greatest glycolytic rate during the first sprint \((r = 0.893, P < 0.01; n = 7)\). Moreover, despite a high negative correlation between the rates of anaerobic glycogenolysis between the first and the last sprints \((r = -0.842, P < 0.05; n = 7)\), those who achieved the greatest amount of work in sprint 1 also performed more work in sprint 10 \((r = 0.851, P < 0.05; n = 7)\).

DISCUSSION

Despite the brief duration of each exercise bout, there were significant decrements in the power output over the
ten 6-s sprints. These decrements in power output were accompanied by a considerable reduction in the rate of ATP production by anaerobic processes. This was mainly reflected by the 10-fold decrease in the rate at which glycogen was degraded to lactate in the final sprint compared with the values calculated for the first sprint.

Previous studies from this laboratory have proposed that intermittent maximal exercise of brief duration (6 s) with short rest intervals (30 s) leads to an increasing demand on anaerobic glycolysis to maintain the rate of energy production because of incomplete resynthesis of PCr stores (31). This was not the case in the present study, which employed a similar work-to-rest ratio.

The initial ATP production rate, estimated from the changes in the concentration of muscle metabolites, was 14.9 ± 2.2 mmol·kg dry wt$^{-1}$·s$^{-1}$. This value is close to the theoretical maximum rate of 17 mmol·kg dry wt$^{-1}$·s$^{-1}$ calculated by McGilvery (21). Degradation of PCr accounted for one-half of the ATP resynthesized (49.6%) from anaerobic sources, with the remainder being derived predominantly from glycolysis (44.1%).

There is, of course, the possibility that some glycogen was used aerobically. However, by assuming an ATP energy provision of 100 kJ/mol (21), a mechanical efficiency of 20% (15), a water content of 77%, and an exercising muscle mass of 14.4 kg for the subjects used in this study (muscle mass of the lower extremities calculated from radiographic estimates of lean leg size) (30) and by applying these values to the total work done in the 6-s sprint, it was then estimated that a total ATP production rate of 13.1 mmol·kg dry wt$^{-1}$·s$^{-1}$ was required to sustain the work done. This value is remarkably close to that calculated in the present study from anaerobic sources. Another approach is an examination of the relationship between glycogen utilization and lactate produced. Interestingly, those individuals who exhibited the highest rates of glycogen degradation in the first sprint accumulated more lactate in muscle ($r = 0.79, P < 0.05; n = 8$) and also had the greatest ATP production rates from anaerobic sources ($r = 0.84, P < 0.01; n = 8$), supporting the view that most of the glycogen was metabolized anaerobically. These findings suggest that the MPO generated over the first sprint must have been fueled by energy derived mainly from PCr and anaerobic glycolysis.

These results also support the observations of other authors (2, 14) that glycolgenolytic processes leading to lactate formation are initiated within the first few seconds of maximal dynamic exercise.

The extent to which glycogen was used anaerobically in the first 6-s bout of exercise, as calculated from the accumulation of hexose monophosphates, lactate, and pyruvate, represents one of the highest rates of glycogenolysis (4.4 ± 0.9 mmol glycosyl units·kg dry wt$^{-1}$·s$^{-1}$) recorded for human quadriceps femoris muscle. This high value possibly reflects the great involvement of the vastus lateralis muscle group during maximal cycling. This value is similar to that observed in a previous study from this laboratory (2).

The high glycogenolytic rate calculated from changes in metabolites during the first sprint requires a total phosphorylase activity of 264 mmol glycosyl units·kg dry wt$^{-1}$·s$^{-1}$. This greater phosphorylase activity could be attributed, in part, to a high rate and great transformation of inactive phosphorylase $b$ to active phosphorylase $a$ resulting from the rapid fivefold rise in plasma epinephrine concentration that was observed after the first sprint.

In the final 6 s of exercise the estimated ATP production rate from anaerobic sources was reduced to 35.6% (5.3 ± 2.5 mmol·kg dry wt$^{-1}$·s$^{-1}$) of that estimated for the first sprint. Glycolysis contributed only 16.1% as the glycolytic rate had fallen 7.6 times. In addition there was a 10-fold decrease in the glycogenolytic rate, although it always exceeded glycolysis.

The increase in muscle lactate concentration found at the end of the exercise test is associated with large reductions in muscle pH (11). Blood pH was also decreased to 7.10 units after completion of the test. This blood pH value has been related to decreases in muscle pH to ~6.6 (11). By using the equation described by Sahlin (24) for calculating the muscle pH from lactate and pyruvate changes in muscle and applying it to our values, it is estimated that the muscle pH before the last sprint was 6.59. It seems likely, therefore, that acidosis resulting from maximal anaerobic degradation of glycogen, particularly during the early stages of the test, may be responsible for the observed reduction in the glycogenolytic and glycolytic rates.

In contrast, the contribution from PCr to ATP production was 80% in the final sprint. The PCr concentration measured 10 s before the tenth sprint was still only 49% of the resting value and subsequently fell to 16% during the last sprint. However, if one assumes that PCr concentration after completion of the ninth sprint reached a similar value to that measured after sprint 10, then it can be calculated from the observed PCr values measured 10 s before the last sprint that PCr concentration was restored at a rate of 1.3 mmol·kg dry wt$^{-1}$·s$^{-1}$, which is in agreement with the value of 2 mmol·kg dry wt$^{-1}$·s$^{-1}$ reported by Harris and co-workers (8). On the basis of these calculations it appears that the muscle PCr stores were reduced almost to the same extent during the first and the final sprints. If this were the case, then it suggests that a 30-s recovery interval may be sufficient to allow significant PCr resynthesis and, therefore, considerable contribution from PCr to ATP production during this type of intermittent exercise, even after ten maximal 6-s sprints. This is in contrast to previous suggestions that PCr degradation may be of minor importance in maintaining high rates of ATP production during the latter stages of intermittent exercise of maximal intensity (31).

Therefore, the decline in power output in the present study was accompanied by evidence of decreasing contribution to ATP production from glycogen degradation to lactate formation as indicated by the changes in muscle hexose monophosphates and lactate concentrations before and after the last exercise bout. However, whether the decreased rate of ATP resynthesis in the last sprint, as a result of a low rate of glycogen degradation to lactate, was secondary to inhibited ATP hydrolysis, as previously suggested (13), is not known.

Despite a dramatic reduction in energy yield from anaerobic glycolysis in the last sprint, the MPO achieved was 73% of that in the initial sprint. Several
possibilities exist to explain this difference. First, a greater reliance on aerobic metabolism of glycogen is possible. A detectable fall in glycolygen content was observed in the last bout of exercise, which cannot be accounted for by the accumulation of glycolytic intermediates, glucose, or lactate. It is difficult to account for the amount of lactate that escaped from the muscle into the circulation during that sprint. The increase in blood lactate immediately after exercise may reflect glycolygenolytic activity of all preceeding exercise bouts. Because no differences in blood lactate were seen before and after the last exercise bout, however, it is possible that little lactate left the muscle during this period of exercise. Moreover, the post-exercise hyperglycemia in blood observed in the present study is thought to have been derived from liver glycogonolysis due to the high levels of circulating catecholemynes.

The possibility, therefore, that some aerobic metabolism of glycogen occurred in the last sprint cannot be excluded. Elevated H+ concentrations have been suggested to increase the activity of pyruvate dehydrogenase and so enhance aerobic metabolism. Previous studies on intermittent exercise have shown that intramuscular triacylglycerol stores may also be a potential fuel source during this type of exercise. Additional support for this proposition is provided by a study that reported a 500% increase in plasma glycerol during maximal intermittent exercise.

A significant shift to aerobic metabolism at the latter stages of the exercise test, due to a reduced anaerobic energy yield, seems to be the most plausible explanation for the decrease in power generated in the last sprint. Recruitment of additional muscle groups, that usually have a secondary role in power generation, seems unlikely because of the maximal nature of the exercise test. An increase in efficiency of muscular contractions is also unlikely, as efficiency during dynamic exercise appears to decrease when lactate accumulates, in contrast to continuous stimulation where the energy cost per unit force is decreased.

The decline in power output observed in the present study with successive sprints was greater than the corresponding values previously reported during repeated sprints on the nonmotorized treadmill. These more pronounced decrements in performance observed in the present study could possibly be attributed to the greater leg speeds during cycling compared with running due to the higher intrinsic resistance of the treadmill. It is also possible that during sprint running subjects improve their running stride efficiency with increasing fatigue by shortening their stride length.

The power outputs in the present study during each exercise bout were also higher than those values observed for active male subjects during repeated 6-s bouts of sprint running with 30-s recovery intervals on a nonmotorized treadmill. These differences in performance possibly reflect the lack of load optimization associated with treadmill sprinting. Power output requires an optimal resistance to attain a maximal value. In treadmill sprinting this resistance is determined by the inertia of the belt and the bearings only. In addition, only the propulsive component of the total external power output generated by the subjects is being measured during this type of treadmill sprinting.

In summary, the results of the present study indicate that the average power output over the first sprint was supported by energy that was derived mainly from PCr and anaerobic glycogenolysis. In the final sprint, however, no lactate accumulation was apparent, yet the average power output was still 73% of that in the initial sprint. On the basis of the circumstantial evidence that suggests an inhibition of anaerobic glycolysis at the end of exercise, it is reasonable to propose that ATP resynthesis was mainly derived from PCr degradation and oxidative metabolism.

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