

## THE PHYSIOLOGICAL EFFECTS OF A MID-SHIFT FEED OF SUCROSE\*

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### SUMMARY

When men work continuously at a moderate (1.0 litres/min oxygen consumption) or hard rate (1.5 litres/min oxygen consumption) for 6 hours, the respiratory quotient falls from 0.94 to 0.80. In classical concepts this indicates that 68% of the caloric requirement comes from carbohydrate initially and that only 30% comes from that source at the end of the work period. This change in metabolism is associated with an increase in oxygen consumption and heart rate, indicating that the men were being less effective mechanically towards the end of the work period when they were using predominantly fat metabolism.

A mid-shift feed of 100 and of 200 g of sucrose in water caused an immediate rise in RQ and a higher level of RQ than in the fasting men which persisted for the rest of the work period. This was associated with a lower rate of oxygen consumption than in the fasting state. While there may be some doubt about the immediate increase in RQ being due to a shift towards carbohydrate metabolism after the ingestion of sucrose, there can be little doubt that this is the case from the end of the first hour onwards, after the ingestion of sucrose.

The doubt about whether the rapid increase in RQ after the ingestion of sucrose is due to a change from fat to carbohydrate metabolism or whether it is due to an alteration in blood insulin level following the ingestion of sucrose could be resolved by ingesting  $^{14}\text{C}$  sucrose and studying the output of  $^{14}\text{C}$   $\text{CO}_2$  in expired air. In view of the practical importance to industry of showing whether or not carbohydrate given during a mid-shift feed is actually metabolized during the remainder of the shift, a study with  $^{14}\text{C}$  sucrose appears to be fully justified; its scientific value in solving the above problems is not in doubt.

The energy required for the re-synthesis of adenosine triphosphate (ATP) in the muscle during exercise is generated principally by the oxidation of phosphorylated glucose derived from the glycogen of the muscles, as well as from the rapid oxidation of free fatty acids (FFA). Both processes are involved during muscular exercise, but the proportions of energy from these two sources at different phases of exercise and at different levels of effort are not well established.

There is indirect evidence<sup>20,21</sup> that the proportion of energy for muscular work from the oxidation of FFA increases during prolonged exercise in the fasting subject. This conclusion was based upon an observed fall in respiratory quotient over the work period. Recently there has been more direct evidence on this point. Seven hours of treadmill running at 5.6 km/h caused a 5-fold increase in the blood FFA.<sup>24</sup> Measurement, by means of muscle biopsy, of the amount of glycogen in working muscle, has shown that the glycogen stores are depleted by prolonged heavy exercise.<sup>7,25</sup> Moreover, the capacity to continue strenuous exercise is affected when the glycogen stores are depleted.<sup>2,7,25</sup>

Studies have been made to determine whether the proportions of energy for muscular work from carbohydrate and fat sources can be altered by different dietary regimes and whether the subjects benefit from these dietary regimes. Mager *et al.*<sup>23</sup> showed that during prolonged muscular work, metabolism of fat could be decreased by repeated intake of carbohydrate. Havel *et al.*<sup>26</sup> have reported that the increase in the quantity of FFA in plasma during prolonged exercise can be avoided by administering glucose in excess of calorie requirements. Data obtained by Bergström *et al.*<sup>7</sup> indicate that the glycogen content of muscle after exhausting work is restored to higher values by a prior carbohydrate-rich diet than it is by a high-fat or high-protein diet and that the length of time for which men can endure exhausting work, after 3 days on these different dietary regimes, is very significantly greater after the carbohydrate-rich diet.

The Bantu worker does most of the heavy muscular work in industry in South Africa. Following tradition and personal choice he has one large meal in the late afternoon after work. It can be assumed that on this dietary regimen his carbohydrate reserves would be depleted after 3 to 4 hours of physical work and that the energy for muscular work in the latter part of the shift would be obtained predominantly from the fat stores laid down during the previous 12 hours. The question has been raised as to whether a pre-shift or mid-shift feed of carbohydrate would improve the Bantu's ability to do prolonged muscular work. This would be so only if the ingested food-stuff is absorbed and metabolized during the shift. In order to examine this question, respiratory quotients, which give some indication of whether carbohydrates or fats are predominantly metabolized, were measured during a 6-hour period of work in fasting men and in men fed a mid-shift feed of 100 g and 200 g of sucrose. The subjects worked at 2 rates which gave oxygen consumptions of 1.0 and 1.5 litres/min. These oxygen consumptions are equivalent to moderate and hard rates of work, respectively.

### METHODS

Four well-trained Bantu males were used as subjects. No attempt was made to alter their standard compound diet. Their physical characteristics are given in Table I.

TABLE I. PHYSICAL CHARACTERISTICS OF SUBJECTS

Subject	Weight (kg)	Height (cm)	Max. $\text{O}_2$ (ml/kg)
J .. .. .	71.5	173.3	46.8
Fa .. .. .	59.0	165.0	45.0
A .. .. .	60.0	163.0	45.0
Fe .. .. .	65.0	167.4	46.6

Studies were made of each subject at 2 work loads, one requiring an oxygen uptake of 1.0 litre/min, and the other an oxygen uptake of 1.5 litre/min. These loads corresponded, respectively, to 30% and 50% of the groups' average maximum oxygen uptake. Studies were conducted

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on pairs of subjects on 4 days per week according to the following schedule:

Week	Work rate	Sucrose administration	Total number of observations
1	1.5 litres O <sub>2</sub> /min	None	16
2	"	100 g	16
3	"	200 g	16
4	1.0 litres O <sub>2</sub> /min	None	16
5	"	100 g	16
6	"	200 g	16

This experimental approach was used rather than isolated studies on the effect of sucrose administration during exercise, because, firstly, any day-to-day variation in diet would have been compensated for during the 6 weeks' duration of the investigation, and, secondly, all results were comparable on a weekly basis. By conducting the studies on 4 days of the week except Mondays, any possible 'Monday effect' was eliminated. Subjects performed the usual work load for 6 hours on Mondays, but no observations were made on that day.

#### No-Sucrose Weeks

Observations were made on the subject after he had been resting for at least 1 hour. Expired air was collected in Douglas bags for 5 minutes. A Collins mask, fitted with a low-resistance breathing valve and having a dead space of 100 ml was used. The subject was then weighed, and his rectal temperature measured with a clinical thermometer inserted to a depth of about 8 cm.

Work consisted of pedalling a bicycle ergometer at a continuous fixed work load for 50 minutes of every hour for 6 hours. Recording of heart rate were made with a standard ECG during the 45th minute of exercise of every hour. Expired air was collected from the 46th to 49th minutes of exercise. The subject stopped at the 50th minute of exercise to be weighed and for his rectal temperature to be measured. Work was resumed at the 60th minute. An attempt was made to prevent subjects from becoming dehydrated by replacing weight loss other than that due to voided urine, by administering an equivalent amount of drinking water.

#### Sucrose Weeks

During the second, third, fifth and sixth weeks, 100 g and 200 g of sucrose were administered orally immediately after work was resumed for the fourth hour. The sucrose was dissolved in the amount of water required to compensate for the hourly weight loss. The solution was flavoured with lime juice which was also used during the first and fourth control weeks. Observations were conducted in the same way as during the no sucrose weeks. Additional samples of expired air were collected 15, 25 and 35 minutes after administration of the sucrose.

#### Additional Observations

**Blood-lactic acid content.** Blood samples were collected from the subject's preheated fingertip at the end of the first and sixth hours of work during the first, second and third weeks.

**Blood-glucose content.** Blood samples were collected from the fingertips of 2 subjects on 2 occasions when no sucrose had been given and another 2 when 200 g of

sucrose had been given at the work load requiring 1.5 litres O<sub>2</sub>/min. Samples were taken during resting, and during the second, fourth and sixth hours of work.

**Urinary ketone excretion.** On 2 days during both the first and third weeks urine samples were collected from 2 subjects for ketone-body content determinations. Samples were taken at the end of the second, fourth and final hours. The subjects emptied their bladders every hour.

**Cardiac output.** Cardiac output was measured on 2 subjects, twice weekly during the first, second and third weeks. Observations were made at the end of the second, fourth and sixth hours of work.

#### Analytical Procedures

**Expired-air samples.** Douglas bag volumes were measured with a chain-compensated spirometer. Samples of expired air were collected in 2 butyl rubber bags of 2-2½ litres capacity and analysed on a Beckman LB 1 infrared medical gas analyser for carbon dioxide content. The apparatus was calibrated with high purity nitrogen and about 4% carbon dioxide in air. The remainder of the air was then analysed for oxygen content on 2 Beckman Model E<sub>2</sub> paramagnetic oxygen analysers according to the method described by Strydom *et al.*<sup>42</sup> From the carbon dioxide and oxygen contents of the samples and the corrected volume of expired air at STPD, oxygen uptake and RQs were calculated. RQs determined in this way agreed well with those obtained by accepted chemical methods.<sup>5</sup>

**Blood lactic acid** was assayed by the method of Barker and Summerson<sup>4</sup> with some modifications by Hullin and Noble.<sup>24</sup>

**Blood glucose** content was determined by the o-toluidine method as reported by Hyvarinen and Nikkilä.<sup>27</sup>

**Urinary ketones** were determined according to the method described by Michaels *et al.*<sup>25</sup>

**Cardiac output** was measured by means of a carbon dioxide re-breathing procedure described by Defares.<sup>25</sup> A rapid infrared carbon dioxide meter was used for following the changes in PCO<sub>2</sub> during re-breathing. Arterial PCO<sub>2</sub> was determined by using the Bohr formula, and dead space was estimated by the method of Asmussen and Nielsen.<sup>3</sup>

#### RESULTS

Summaries of the results are given in Tables II and III.

In order to determine whether there were differences between the reactions for no-sucrose and 100 g, no-sucrose and 200 g, an analysis of variance was performed on the results. The results of analysis of variance of pulmonary ventilation, oxygen consumption, respiratory quotient and heart rate are given in Tables IV - VII.

#### Heart Rates (Fig. 1)

**Work rate 1.5 litres O<sub>2</sub>/min.** Heart rate changed little during the first 3 hours of all experiments. When no sucrose was given, average heart rates showed a continuous increase during the second 3-hour period from 118 to 128 beats per minute. When 100 g of sucrose was given, heart rate continued to increase towards the fourth and then remained constant until the sixth hour. Values for the fifth and sixth hours were statistically significantly different from those of the corresponding hour of the non-sucrose study.

TABLE II. MEAN VALUES FOR 4 SUBJECTS EXERCISING AT  $\dot{V}_{O_2}$  1.5 LITRES/MIN

	Sucrose dose (g) admin. end 3 hours	Time of observation (hour)									
		Rest	1	2	3	3:15	3:25	3:35	4	5	6
Heart rate (beats/min)	0	72	118	115	118				121	124	128
	100	66	117	112	114				120	116	119
	200	66	114	111	114	116	117	118	122	121	120
$\dot{V}_{O_2}$ (litre/min)	0	0.33	1.50	1.53	1.57				1.61	1.64	1.64
	100	0.32	1.51	1.51	1.54				1.55	1.53	1.55
	200	0.32	1.46	1.51	1.53	1.57	1.57	1.57	1.57	1.57	1.53
$\dot{V}_{CO_2}$ (litre/min)	0	0.30	1.35	1.32	1.31				1.29	1.30	1.29
	100	0.30	1.35	1.32	1.28				1.40	1.31	1.29
	200	0.30	1.33	1.33	1.29	1.39	1.40	1.42	1.43	1.43	1.32
VE (litre/min)	0	10.3	32.2	32.2	31.8				31.3	31.8	32.4
	100	9.9	32.3	31.2	30.7				32.7	31.4	31.7
	200	10.2	31.7	31.7	31.2	32.7	33.6	33.9	34.3	34.4	32.0
RQ	0	0.9180	0.8983	0.8644	0.8348				0.8009	0.7922	0.7868
	100	0.9516	0.8972	0.8718	0.8293				0.8991	0.8568	0.8319
	200	0.9498	0.9060	0.8795	0.8381	0.8852	0.8948	0.9008	0.9087	0.9122	0.8621
Cardiac output (litre/min)	0			11.78					11.58		11.72
	100			11.15					11.57		12.09
	200			11.90					11.97		11.87
Stroke volume (ml)	0			106.00					99.91		96.07
	100			106.84					100.08		110.07
	200			111.51					102.07		102.40
Pco <sub>2</sub> (arterial)	0			34.8					34.8		33.8
	100			34.4					34.5		34.5
	200			34.8					35.0		34.2
Pco <sub>2</sub> (mixed venous)	0			60.2					60.3		57.8
	100			60.7					62.3		58.2
	200			60.4					63.7		60.0
Lactate (mM/litre)	0	1.840	1.188								1.202
	100	1.957	1.167								0.930
	200	1.792	0.973								0.984
Blood glucose (mg/100 ml)	0	86.4		82.0					80.5		72.4
	100										
	200	84.8		82.5							76.0
Total ketones in urine (mg excreted/h)	0			11.00					10.45		10.08
	100										
	200			11.85					11.83		13.35
b-OH butyrate in urine (mg excreted/h)	0			8.40					7.51		7.30
	100										
	200			8.09					1.48		2.50
Aceto-acetate in urine (mg excreted/h)	0			2.60					2.94		2.78
	100										
	200			3.76					10.35		10.85
Grams carbohydrate utilized	0			163				87			
	100			173				153			
	200			177				183			
% calories from carbohydrate	0		66	52	42				32	29	29
	100		66	56	42				66	52	42
	200		69	59	46	63	63	66	69	69	52

TABLE III. MEAN VALUES FOR 4 SUBJECTS EXERCISING AT  $\dot{V}_{O_2}$  1.0 LITRES/MIN

	Sucrose dose (g) admin. end 3 hours	Time of observation (hour)									
		Rest	1	2	3	3:15	3:25	3:35	4	5	6
Heart rate (beats/min)	0	63	95	93	92	93	93	93	95	93	94
	100	61	92	92	92	94	94	95	97	95	93
	200	66	95	94	95	97	100	101	104	103	98
$\dot{V}_{O_2}$ (litre/min)	0	0.28	0.94	0.98	0.99	1.00	1.00	1.01	1.03	1.00	1.04
	100	0.28	0.96	1.00	1.01	1.04	1.05	1.05	1.05	1.02	1.02
	200	0.27	0.97	1.00	1.05	1.05	1.05	1.06	1.08	1.07	1.05
$\dot{V}_{CO_2}$ (litre/min)	0	0.26	0.85	0.85	0.84	0.86	0.85	0.85	0.84	0.80	0.83
	100	0.26	0.85	0.87	0.84	0.92	0.96	0.97	0.97	0.90	0.88
	200	0.30	0.87	0.87	0.87	0.97	0.99	0.99	1.02	1.01	0.94
VE (litre/min)	0	9.0	20.9	20.6	20.4	21.1	21.0	20.9	21.0	20.4	21.6
	100	8.4	20.3	20.7	20.5	21.3	22.1	22.4	21.8	21.7	20.8
	200	8.4	20.6	20.9	20.8	22.8	23.0	23.2	24.1	24.0	22.2
RQ	0	0.9054	0.8907	0.8678	0.8499	0.8575	0.8491	0.8372	0.8193	0.7955	0.7995
	100	0.9242	0.8902	0.8680	0.8381	0.8845	0.9141	0.9282	0.9241	0.8916	0.8556
	200	0.9260	0.8963	0.8711	0.8284	0.9186	0.9369	0.9410	0.9468	0.9413	0.8926
Grams carbohydrate utilized	0			110					73		
	100			108					120		
	200			108					148		
% calories from carbohydrate	0		69	56	49				39	32	32
	100		63	56	46	59	69	76	73	63	52
	200		66	56	42	73	80	80	83	80	63

TABLE IV. PULMONARY VENTILATION

Work rate	Hour	Between no sucrose and 100 g of sucrose	Between no sucrose and 200 g of sucrose	Between 100 g and 200 g of sucrose
		5 000	4	S
	5	N.S.	S	S
	6	N.S.	N.S.	N.S.
3 000	4	S	S	S
	5	S	S	S
	6	N.S.	N.S.	S

TABLE V. OXYGEN CONSUMPTION

Work rate	Hour	Between no sucrose and 100 g of sucrose	Between no sucrose and 200 g of sucrose	Between 100 g and 200 g of sucrose
		5 000	4	S
	5	S	S	S
	6	S	S	N.S.
3 000	4	N.S.	S	S
	5	N.S.	S	S
	6	N.S.	N.S.	S

TABLE VI. RESPIRATORY QUOTIENT

Work rate	Hour	Between no sucrose and 100 g of sucrose	Between no sucrose and 200 g of sucrose	Between 100 g and 200 g of sucrose
		S	S	N.S.
5 000	4	S	S	N.S.
	5	S	S	S
	6	S	S	S
3 000	4	S	S	S
	5	S	S	S
	6	S	S	S

TABLE VII. HEART RATE

Work rate	Hour	Between no sucrose and 100 g of sucrose	Between no sucrose and 200 g of sucrose	Between 100 g and 200 g of sucrose
		N.S.	N.S.	N.S.
5 000	4	N.S.	N.S.	N.S.
	5	S	N.S.	S
	6	S	S	N.S.
3 000	4	S	S	S
	5	N.S.	S	S
	6	N.S.	N.S.	S

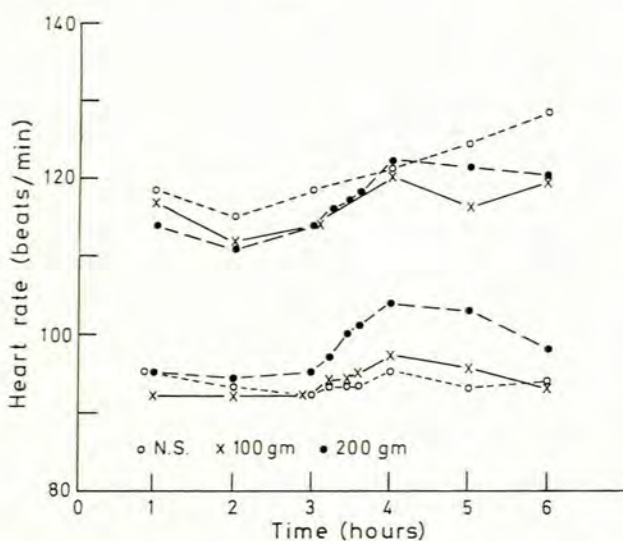


Fig. 1. The influence of different quantities of sucrose on the heart rate.

The responses obtained by the 200 g dose were very similar to those for the 100 g dose. Although heart rate remained unchanged after the fourth hour, results for no-sucrose and 200 g of sucrose were significant only for the final hour. The results obtained with 100 g and 200 g were significantly different only for the fifth hour. The net results with the 2 doses seem to be the same.

*Work rate 1.0 litre  $O_2$ /min.* Average heart rates remained practically unchanged throughout the whole 6-hour period during which no sucrose was given. The over-all result of a 100-g sucrose dose was one of no change. On the other hand, a dose of 200 g caused a significant cardio-acceleration in the fourth and fifth hours. Sixth-hour values, although still elevated, did not, however, differ significantly from those of the no-sucrose experiment. There was a significant difference between the results for 100 g and 200 g of sucrose in the fourth, fifth and sixth hours.

### Oxygen Uptake (Fig. 2)

*Work rate 1.5 litres  $O_2$ /min.* When no sucrose was given, a steady increase in oxygen uptake during the 6-hour work period was observed. When sucrose was given, oxygen uptake remained practically unchanged after the third hour. Oxygen uptakes for the fourth, fifth and sixth hours were significantly lower compared with those obtained in the experiments during which no sucrose was administered. A significant difference was observed between the results obtained after administration of 100 g and 200 g of sucrose during the fifth hour.

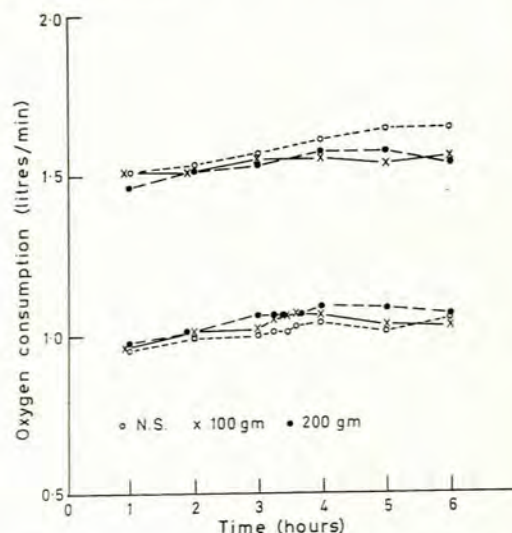


Fig. 2. The influence of different quantities of sucrose on oxygen consumption.

*Work rate 1.0 litre  $O_2$ /min.* Oxygen uptake increased with time when no sucrose was given. Administration of sucrose after the fourth hour prevented a further increase in oxygen uptake. The significantly higher oxygen uptake observed during administration of 200 g was already present before sucrose administration. The difference between work rates when 100 g and 200 g of sucrose was administered was significant during the fourth, fifth and sixth hours of work.

### Carbon Dioxide Output

Carbon dioxide output remained fairly unchanged during both work loads for the whole period during which no sucrose was given.

Sucrose caused a marked increase in carbon dioxide production at both work loads. The effect was more pronounced when 200 g was administered.

### Pulmonary Ventilation

*Work rate 1.5 litres  $O_2$ /min.* Pulmonary ventilation remained relatively constant for the whole 6 hours of work when no sucrose was given. One hundred grams of sucrose caused a slight but significant increase in ventilation during the fourth hour. There was no significant difference between the values for the fifth and sixth hour observed for no-sucrose and 100 g of sucrose. Administra-

tion of 200 g of sucrose caused a significant increase in ventilation during the fourth and fifth hours with the sixth-hour value not significantly different from that for the no-sucrose experiment.

*Work rate 1.0 litre O<sub>2</sub>/min.* Pulmonary ventilation during the no-sucrose experiment remained relatively unchanged during the whole 6 hours. Administration of 100 g or 200 g of sucrose resulted in a significant increase in ventilation during the fourth and fifth hours. Last-hour values were close to those observed when no sucrose was given. There was a significant difference between the ventilation responses following administration of 100 g and 200 g of sucrose in that the latter produced higher values.

#### Respiratory Quotient

*Work rate 1.5 litres O<sub>2</sub>/min.* (Fig. 3). The decrease in RQ when no sucrose and sucrose was given was similar during the first 3 hours of work. During this period RQ decreased from about 0.90 to about 0.83. During the no-sucrose experiment, RQ continued to decrease the fourth-hour value of 0.80 after which little change occurred.

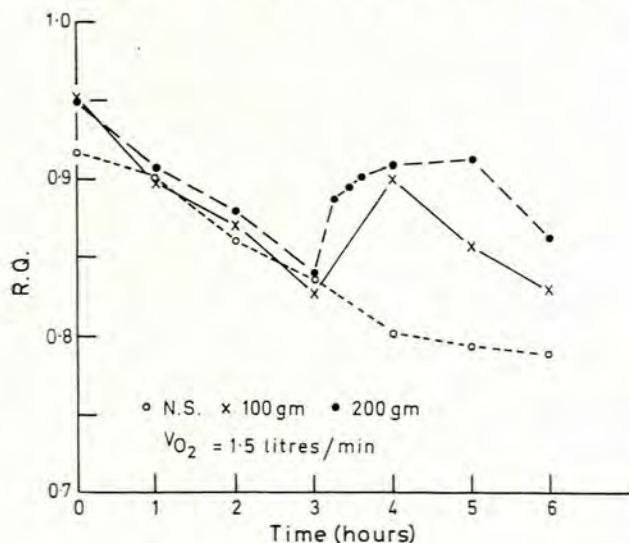


Fig. 3. The influence of different quantities of sucrose on the RQ at an oxygen uptake of 1.5 litres/min.

When 100 g of sucrose was given, RQ increased significantly during the fourth hour and although it declined thereafter it remained significantly higher during the fifth and sixth hours compared with the value in the no-sucrose experiment. Administration of 200 g of sucrose caused a rapid increase in RQ to reach a peak during the fourth hour of about the same value as that obtained with 100 g. This increase was maintained until the fifth hour, after which the RQ decreased during the final hour. The response obtained during the fifth and sixth hours with 200 g was significantly different from that obtained after administration of 100 g. The peak RQ value after administration of sucrose usually equalled the value observed during the first hour of work.

*Work rate 1.0 litre O<sub>2</sub>/min* (Fig. 4). When no sucrose was given, RQ decreased continuously until the fifth hour,

after which there was no change. A dose of 100 g of sucrose after the third hour caused a rapid and significant increase in RQ during the first 15 minutes, and a peak value was reached at the fourth hour. Thereafter the RQ value decreased towards the sixth hour but still remained significantly higher than the corresponding value for no sucrose. A similar effect was achieved after administration of 200 g of sucrose, but again the RQ increased until the fifth hour after which it decreased. The difference between the reactions to 100 g and 200 g of sucrose was statistically significant for all hours.

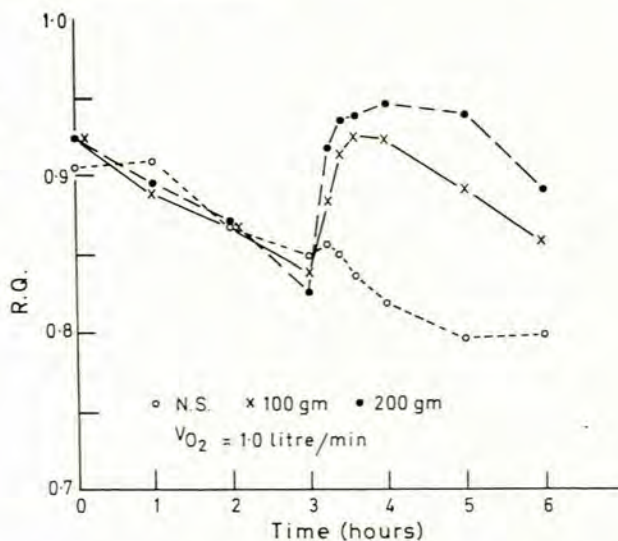


Fig. 4. The influence of different quantities of sucrose on the RQ at an oxygen uptake of 1.0 litres/min.

#### Circulatory Responses

Cardiac output measured in 2 subjects at the higher work load showed the following:

1. Cardiac output had no time trend and was not influenced by either 100 g or 200 g of sucrose.
2. Stroke volume decreased with time. This trend was eliminated by administration of sucrose.
3. The calculated arterial PCO<sub>2</sub> was not influenced by time or administration of sucrose.
4. Mixed venous PCO<sub>2</sub> was increased after administration of sucrose with 200 g causing a bigger rise than 100 g.

#### Blood Lactic Acid

Lactic acid had no time trend and was not influenced by a dose of 200 gm of sucrose. When 100 g of sucrose was given, lactic acid values for the sixth hour were lower than those for the first hour.

#### Blood Glucose

Average values for blood glucose on 2 subjects showed a decrease from 85 mg/100 ml during resting to about 73 mg/100 ml after 6 hours of work. Two hundred g of sucrose increased blood glucose levels to 88 mg/100 ml during the fourth hour of work and these levels eventually dropped to about 78 mg/100 ml during the sixth hour.

### Urinary Ketones

**Total ketones.** Total urinary ketones expressed as milligram of acetone per hour showed a decrease during 6 hours of work. Administration of 200 g sucrose resulted in a higher excretion for the sixth hour.

**B-OH butyric acid.** This fraction of the ketone bodies decreased with time when no sucrose was given. Administration of sucrose caused a marked reduction in the excretion of this metabolite.

**Aceto-acetic acid.** Aceto-acetic acid showed little change during 6 hours of work when no sucrose was given. When 200 g of sucrose was given it was increased almost 3-fold during the fourth and sixth hours.

### Utilization of Carbohydrates

Calculations based on RQ and assuming a calorific value of 4.0 cal/g of carbohydrate gave the following information:

**Work rate 1.5 litres  $O_2$ /min.** When no sucrose was given, a total of 260 g of carbohydrate was utilized during the 6 hours of work. During this period 170 g was utilized during the first 3 hours and 90 g during the second 3-hour period. When 100 g of sucrose was administered, utilization of carbohydrate during the second 3-hour period increased to 155 g, causing a rise of 65 g compared with the results of the no-sucrose study. Calculations for the 200 g sucrose dose showed that the carbohydrate utilization was increased to 185 g during the second 3-hour period thus resulting in an increase of 95 g.

**Work rate 1.0 litre  $O_2$ /min.** A total of 185 g of carbohydrate was utilized during the 6 hours of which 110 g was used during the first 3 hours and 65 g during the second 3 hours. A dose of 100 g of sucrose caused a utilization of 120 g in the second 3-hour period, while 200 g caused a utilization of 150 g for this period. Thus utilization was increased by 55 g and 80 g when 100 g and 200 g of sucrose, respectively, were given.

### Calories from Carbohydrate and Fat

The percentage of calories derived from carbohydrate declined from about 70% in the first hour to 30% in the fifth and final hours of work. The decrease was steepest during the first 3 hours of work, namely, from about 70% to about 40%. A dose of 100 g of sucrose caused a return to first-hour values with a subsequent decline to reach the third hour or higher values at the end of the sixth hour.

### DISCUSSION

#### *Does the Respiratory Quotient Indicate the Proportions of Fat and Carbohydrate Metabolized?*

The question of whether or not the RQ during exercise is a valid indication of the relative proportions of fat and carbohydrate used by the muscle during exercise, was examined by Christensen and Hansen.<sup>11</sup> On the basis of measurement of  $CO_2$  and lactate content of the blood they concluded that after 10 to 15 minutes of exercise the RQ values at high, but not maximum, work levels gave a true representation of the fuels utilized. The same investigators found, in agreement with the results of Courtice and Douglas,<sup>12</sup> that the RQ during prolonged work in the

fasting state showed a clear tendency to decrease during the course of work and assumed this to be due to the progressive decrease in glycogen stores.

Results obtained on the glycogen content of working muscles, by the muscle-biopsy technique, as well as the evidence from studies with  $^{14}C$ -labelled glucose and on fatty acid contents in blood, support the conclusion of Christensen and Hansen that a low RQ during prolonged muscular exercise is indicative of a decrease in the proportion of muscular energy derived from carbohydrate metabolism.

There is good evidence that during prolonged muscular exercise there is an increase in the concentration of FFA in plasma<sup>20,21,23</sup> and an increase in the turnover rate of FFA as measured by palmitate- $1-^{14}C$ .<sup>20,21</sup> When the release of FFA from fat deposits is blocked by nicotinic acid,<sup>8,9</sup> muscle glycogen was depleted<sup>5</sup> at a faster rate during muscular exercise and RQ values were significantly higher. Oral administration of carbohydrate is known to block the mobilization of FFA from fat deposits<sup>20</sup> and the higher RQs and lower plasma FFA reported by Havel<sup>20</sup> in exercising subjects who received carbohydrate, compared with subjects receiving water only, are clear indications of a shift towards carbohydrate metabolism. Young *et al.*<sup>43</sup> after studying prolonged exercise at a moderate rate (1.2 litres/min oxygen consumption) state that the quantity of carbohydrate utilized, as determined by  $^{14}C$  studies of glucose rate of turnover and metabolism, is less than they would have predicted from RQ values during exercise. They, however, were not confident about the accuracy of their RQ values during work.

Ahlborg *et al.*,<sup>2</sup> Bergström *et al.*,<sup>5</sup> Hermansen *et al.*,<sup>23</sup> and Hultman<sup>24</sup> using muscle-biopsy techniques to measure the glycogen content of working muscle, found a good correlation between the amount of glucose utilized during work, as calculated from the RQ and the oxygen consumption, and the decrease in muscle glycogen in the thigh muscles. This applied over a wide range of RQ values and glycogen concentrations.

#### *Fat and Carbohydrate Metabolism During Exercise in the Fasting State*

The results on RQs of fasting men obtained in this study during prolonged exercise agree with those reported by a number of workers. It was found that the RQ decreased from 0.94 at the beginning of exercise to 0.80 after 6 hours of work at both levels. From these RQ values it was concluded that 68% of the caloric requirements for the first hour was supplied by the metabolism of carbohydrate and that only 30% came from this source in the sixth hour. It is interesting to note that the RQ fell to the same level at the end of the sixth hour at both work levels. This low level of RQ was reached at the end of the fourth hour at the high rate of work and 1 hour later at the lower rate of work. It can be concluded that the working muscle has to rely more and more upon fat metabolism for its oxidative processes towards the end of prolonged exercise. There is indirect support for this contention in the 5-fold increase in blood FFAs after 7 hours of exercise at 5.6 kmh on the treadmill in the fasting state<sup>24</sup> and in the high turnover rates of palmitate- $1-^{14}C$ , given by intravenous infusion, during muscular exercise.<sup>20</sup>

The continuous increase in fat metabolism with time, as indicated by the decrease in RQ was associated with a continuous increase in oxygen uptake as well as heart rate. This finding is in accord with that of Krog and Lindhard,<sup>36</sup> who showed that the relative change from carbohydrate to fat metabolism reduces the mechanical efficiency by 5-10%. Using the first-hour values as a baseline, it was estimated that mechanical efficiency fell by 2% at the end of the third hour and 9% at the end of the sixth hour.

The increase in utilization of fat during prolonged exercise was accompanied by a decrease in the excretion of ketone bodies in urine. Skeletal muscles as well as heart muscles have been shown to have a high affinity for these substrates. Normally  $\beta$ -hydroxybutyrate and aceto-acetate are not major sources of energy because of their low concentration in blood plasma, but the situation may be quite different during prolonged exercise in the fasting state and effective utilization of these substances in working muscle may account for the decreased excretion of ketone bodies in the urine.

It will be noted that the initial RQs before exercising averaged 0.94 in these experiments. This is higher than is usually found in the postabsorptive state. It is, however, consistent with the report of Courtice *et al.*<sup>34</sup> in which they showed that the RQ after 6 days of a carbohydrate-rich diet was 0.95 compared with an RQ of 0.75 after 6 days of a high-fat diet. The Bantu subjects of these experiments are fed a carbohydrate-rich diet in the mine compounds due to the very high proportion of mealie meal in the diet.

#### *Effects of Mid-shift Feed of Sucrose on Fat and Carbohydrate Metabolism*

Within 15 minutes of ingestion of sucrose at the end of the third hour of the 6-hour period of work there was a significant rise in RQ. The peak value of the RQ occurred an hour after ingestion and the RQ remained significantly higher than that for the control and fasting values for the remainder of the work period. The rise in RQ was significantly higher after administration of the 200-g dose than after administration of the 100-g dose and it was maintained at the higher level for a longer period.

The question that must be answered is whether the rise in RQ after the ingestion of sucrose really means that a change occurred in the proportions of carbohydrate and fat metabolized. Courtice *et al.*<sup>34</sup> showed that an intravenous injection of 1 to 2 units of soluble insulin caused the RQ to rise during rest and exercise, but that the increase was not as rapid nor as consistent as after a subcutaneous injection of 0.5 mg of adrenalin. The ingestion of glucose leads to an increase in insulin level in blood in association with a rise in blood sugar level.<sup>30</sup> The results of these experiments showed that blood sugar concentration rose during exercise after the ingestion of sucrose for a period of one hour and it can be assumed that blood insulin levels also rose in that period.

Whether a significant increase in blood insulin occurs during exercise after the ingestion of sucrose and whether this could account for the rise in RQ observed, is open to conjecture. Intravenous infusion of glucose did not result in as significant, or as consistent, an increase in blood insulin during exercise as it did in men at rest.<sup>37</sup> Further-

more, it is generally agreed that insulin is not the only factor which facilitates the uptake of glucose and its oxidation in cells during muscular exercise.<sup>32,35,37,38,43</sup> For example, there is evidence that the lowering of blood sugar by exercise still occurs in pancreatectomized animals and in diabetic humans.<sup>37,43</sup> At present, and until measurements of blood insulin are made in experiments similar to those reported in the present study, it must be accepted that the part insulin plays in the rise in RQ after the ingestion of sucrose during muscular exercise, is still an open question.

There is some doubt, therefore, whether the rise in RQ during the first hour after the ingestion of sucrose indicates an increase in the rate of carbohydrate metabolism and a decrease in the rate of fat metabolism. This is not so as regards the last 2 hours after the ingestion of sucrose because the oxygen consumption in that period does *not* increase with time as it does when the men were fasting. This indicates, as mentioned in the previous section, that the mechanical efficiency of the work decreases on prolonged exercise in the fasting state with the change to fat metabolism. When sucrose is administered the decrease in mechanical efficiency is prevented. The improvement in mechanical efficiency following administration of sucrose is good evidence that the working muscles are metabolizing a higher proportion of carbohydrates and that this is reflected in the rise in RQ. It should be stated that the oxygen-sparing effect of sucrose ingestion is not due to anaerobic metabolism because there was no difference between the concentration of lactic acid in blood in the first and sixth hours.

There is little in the physiological literature which has a direct bearing on this subject. Mager *et al.*<sup>34</sup> who studied men over a 7-hour period of exercise at 5.6 kmh on a treadmill, noted that FFA level in blood rose 5-fold when the men were in the fasting state and that intermittent ingestion of carbohydrate during the period of exercise prevented this rise in FFA level. Havel *et al.*<sup>30</sup> showed that in a shorter period of work 25 g of sucrose fed at the end of each half hour suppressed the significant rise in blood FFA observed in the fasting state. They measured the turnover rate of palmitate-1-<sup>14</sup>C and concluded that oxidation of FFA entering tissues accounted for 41-49% of energy metabolism during exercise in fasting subjects and 7-10% in subjects fed glucose. They stated further that 'decreased mobilization of FFA after the ingestion of carbohydrate seemed to account almost entirely for their decreased oxidation during muscular exercise'.

Administration of sucrose caused an increased excretion of urinary ketones as well as a change in ratio of  $\beta$ -OH butyrate/aceto-acetate. It was suggested that the ratio of these two substances in blood reflects the intramitochondrial NADH/NAD ratio.<sup>30</sup> Depending on this ratio part of the aceto-acetate is reduced to form  $\beta$ -OH butyrate.

It was also suggested that the redox pair  $\beta$ -OH butyrate/aceto-acetate acts as a hydrogen shuttle transferring reducing equivalents from cytoplasmic NADH to the intramitochondrial respiratory chain.<sup>30,31</sup> It is obvious that the ketone bodies fulfil an important role in cellular metabolism and cannot merely be regarded as breakdown products of fat metabolism. The significance of change in

ratio in the excretion of the ketone bodies is not known but could be indicative of a change in glucose and fructose metabolism at cellular level.

From the above it seems safe to conclude that the ingestion of 100 g or 200 g of sucrose leads to a definite shift to carbohydrate metabolism, but whether this shift also occurs in the first hour after ingestion is an open question. This question can be answered by experiments in which <sup>14</sup>C-labelled sucrose is fed and serial measurements are made of <sup>14</sup>C in carbon dioxide in expired air.

These experiments also showed that there is a significant dose-response effect with the ingestion of 100 g and 200 g of sucrose. Calculation of carbohydrate utilization in the last 3 hours of work, from the rates of oxygen consumption and RQs, shows that, at the higher rate of work, 90 g of carbohydrate was oxidized during fasting, 155 g after administration of 100 g sucrose and 185 g after administration of 200 g sucrose. Comparable figures for carbohydrate utilization at the lower work rate were 65, 120 and 150 g of carbohydrate, respectively. Thus, although there is a definite dose-response effect, the increase in carbohydrate utilization at both levels of work was less when the dose of sucrose was increased from 100 g to 200 g than when it was increased from 0 g to 100 g. Therefore in practice the major effect of a mid-shift feed of sucrose on carbohydrate/fat metabolism is obtained when a dose of 100 g of sucrose is fed. On present evidence there would be no justification for increasing the feed from 100 g to 200 g.

#### Circulatory Changes

The reduced carbohydrate utilization during prolonged exercise in the fasting state is associated with a slow, continuous increase in heart rate and oxygen consumption. Cardiac output, measured in 2 subjects, was unchanged and the increase in oxygen consumption must therefore have been due to an increase in arterio-venous oxygen difference. The increase in heart rate, in conjunction with no change in cardiac output, points to a decrease in stroke volume. The reason for the decrease in stroke volume is not obvious. Although these subjects ended the 6-hour period with a water deficit of  $\pm 1.5$  litres it is unlikely that the water deficits were responsible for the increase in heart rate because the men who were given a mid-shift feed of sucrose attained the same degree of water deficit but their heart rates did not increase. The most reasonable explanation for the increase in heart rate appears to be that it is due to the increase in metabolism. Such an association is well documented.<sup>1, 27, 40, 43</sup>

As has been indicated above, heart rate did not increase, nor did oxygen consumption, when sucrose was ingested midway during the 6-hour period of work. As there was no significant difference between the cardiac outputs in the fasting state and after feeding of sucrose, stroke volume was higher after sucrose administration than in the fasting state.

#### Ventilatory Changes

Minute ventilation during exercise was significantly increased after the ingestion of sucrose. The increase in minute ventilation could not, *per se*, have been responsible for the rise in RQ because the RQ remained high even after the minute ventilation returned to control levels.

The reason for the increased minute ventilation after the ingestion of sucrose is not apparent from the results. Arterial PCO<sub>2</sub> was estimated by the Bohr formula and did not show any significant differences from those observed in men in the fasting state. It is also unlikely that pH was changed as there was no evidence of any increase in blood lactate over the period of the exercise at either work rate. Blood lactates were, however, not sampled at the end of the hour when minute ventilation was increased.

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