Physiological responses to prolonged exercise in ultramarathon athletes

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DAVIES, C. T. M., AND M. W. THOMPSON. Physiological responses to prolonged exercise in ultramarathon athletes. J. Appl. Physiol. 61(2): 611-617, 1986.—The physiological responses of 10 ultramarathon athletes to prolonged exercise at the highest intensity level they could sustain for 4 h have been examined. Energy expenditure for the 4 h of exercise was 14,146 ± 1,789 kJ, of which 63% was provided by the oxidation of fat. Plasma free fatty acids rose, but the changes in blood lactate concentration (Δ0.2 mmol/l) and exchange ratio (Δ0.05) were small, and the postexercise glycogen content (130 ± 42 μmol/g) of the vastus lateralis muscles was estimated to be 37-53% of normal resting values. During exercise O2 intake (VO2) increased with time from the 50th to 240th min, the rise becoming significant (P < 0.01) after 110 min of work. The change in VO2 was equivalent to a rise in relative intensity (%VO2 max) of +9.1% and a change of speed of 1.49 km/h. A rise in cardiac frequency compensated for a fall in stroke volume (SV), so that cardiac output was maintained, and the increases in the laboratory have usually been limited to exercise of 1- to 2-h duration, but little is known of the relative importance of these physiological variables when work time is extended to cover ultramarathon distances. This paper examines the metabolic, cardiovascular, thermal, and muscle function adjustments to 4 h of continuous running on a motor-driven treadmill in 10 ultra-long-distance athletes at the highest level of sustainable %VO2 max.

MATERIALS AND METHODS

The physical characteristics of the 10 ultramarathon athletes were as follows: age, 35.8 ± 7.9 yr; weight, 63.89 ± 5.76 kg; height, 170.7 ± 7 cm; and estimated lean body mass (using skinfold thickness measurements) 56.8 ± 5.5 kg. Their mean maximal aerobic power (VO2 max) was 4.27 ± 0.51 l/min. The athletes were all in regular training (100-200 km/wk) during their period of measurement in the laboratory. During preliminary visits to the laboratory the O2 cost (VO2) of running on the treadmill at various speeds was determined and measurements were made of each athlete's VO2 max. The athletes were then required to exercise continuously at the highest level of work they could tolerate for 4 h on the treadmill. A number of factors were taken into account for selecting this level of work. First, we felt that the intensity for a given duration should be such as to stress the aspects of physiological regulation to be investigated. A period of 4 h of exercise at the maximum load that could be tolerated was considered potentially capable of severely taxing energy reserves, producing large fluid losses and placing the cardiovascular and thermoregulatory systems under extreme stress. Since intersubject differences in endurance capacity are minimized when duration is expressed as a function of work load (9), the athletes were exercised at the same upper limit of relative (%VO2 max) that could be sustained for 4 h. This was estimated as between 65 and 70% VO2 max from our previous studies (7-9) of the relationship between sustainable %VO2 max and exercise duration in athletes. For each athlete this was determined on the basis of his VO2-to-speed relationship and his measured VO2 max.

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Before exercise commenced, tests of quadriceps muscle function were made using electrically evoked voluntary contractions after the method of Edwards et al. (14). During the muscle function tests the athlete was seated and secured in an adjustable straight-backed (Tornvall) chair with knee flexed at 90°. An inextensible taut strap passed around the ankle under the chair to a steel bar transducer, on which were hobbled two strain gauges to form one half of a Wheatstone bridge. Force produced by contraction of the quadriceps was transmitted to the bar and recorded (after amplification) using a storage oscilloscope and ultraviolet recorder. Contraction of the quadriceps was evoked percutaneously using two large pad electrodes placed proximally and distally to the anterolateral thigh. Two tests of electrically evoked muscle function were used. The first test measured the evoked tetanic tension of the right quadriceps at frequencies of 10, 20, 50, and 100 Hz, using a submaximal voltage 8-s stimulus train (2 s at each frequency) and a pulse width of 50-μs duration. The force produced were expressed as a percentage of the maximal force recorded. In the second test the left quadriceps was stimulated continuously for 18 s at a frequency of 30 Hz, and the percentage decline of force was recorded. After appropriate rest periods the subjects performed three maximal voluntary contractions (MVCs) with each leg, the highest recorded force being used for subsequent analysis, and sustained 30% of their right leg MVC until the required force could no longer be maintained. The time taken was recorded and used as an indication of the voluntary isometric endurance of the quadriceps muscle. The subjects were weighed nude and clothed and then had a resealable Butterfly needle placed in the antecubital vein for blood sampling and a thermistor probe inserted into the rectum for the measurement of core temperature.

During the 4 h of exercise the treadmill speed was held constant and O2 intake (VO2) and cardiac output (Q) were measured at the 20th min and then every 30 min throughout the work period. For the measurement of VO2 a low-resistance open-circuit technique was used, and Q was determined by the indirect (CO2) Fick procedure. This involved the measurement of oxygenated mixed venous PCO2 using a rebreathing method (19) in which the athlete was given the appropriate volume and concentration of CO2 in O2 to rebreathe for 12–15 s. The end-tidal PCO2 was recorded continuously to identify the plateau representing equilibrium between mixed venous PCO2, alveolar PCO2, and the rebreathing plateau. If a perfect plateau (i.e., one that appeared within 3–4 s and broke with recirculation at 10 s) was not obtained, the extrapolation procedure as described by Denison et al. (10) was applied.

Blood samples were obtained on six subjects at rest and during and immediately following exercise. During exercise the samples were taken while the athlete was running at the following times: 15, 30, 45, 60, 105, 135, 160, 225, and occasionally 240 min. The blood sample was usually 10 ml. The plasma was analyzed for concentrations of free fatty acids (FFA), glycerol, glucose, lactate, and proteins, and the hematocrit ratio was noted. Plasma FFA concentration was determined by the method described by Duncombe (12), and plasma glycerol, glucose, and lactate were determined enzymatically, using material kits obtained from Boehringer-Mannheim.

Rectal temperature (Tm) was measured every 2 min from the thermistor probe, which was inserted 8 cm above the anal sphincter. Skin temperatures were measured every 15 min using a thermocouple placed on the skin at 13 sites: hand, upper and lower arm, forehead, chest, sternum, abdomen, scapula and lumbar regions of the back, and anterior and posterior regions of the thigh and calf. The recordings were weighted, and the average value of the 13 sites was taken as the mean skin temperature (Tsk, see Ref. 8). At the end of each 30 min, exercise was stopped momentarily (<30 s) to enable the athletes to be weighed.

Fluid in the form of water was available to the athletes on demand during the experiments, and intake was recorded. The ambient temperature of the exercise room was maintained at a constant level [dry-bulb temperature (Tm) = 21°C, wet-bulb temperature (Twb) = 17°C, relative humidity (rh) = 48%], and convective airflow (2.5–4.0 m/s) was always sufficient to allow for the evaporation of sweat. From the raw (thermal) data calculation of the metabolic heat production (M), heat storage (S), evaporative sweat loss (msw), and body heat conductance (K) were calculated using conventional equations. Immediately on the cessation of exercise the muscle function tests were repeated, and 1 h postexercise a muscle biopsy sample was taken from the vastus lateralis. The muscle fibers of the biopsy sample were classified as type I or type II on the basis of their histochemical reaction to myosin adenosinetriphosphatase after preincubation at pH 9.4 (11). The “lesser fiber diameter” (see Ref. 11) of 100 fibers of each type was measured by using an eyepiece micrometer. It was assumed that each fiber had a circular cross section of diameter equal to the lesser fiber diameter, and the mean fiber cross-sectional area was calculated for each type. Muscle glycogen was analyzed by acid hydrolysis, followed by subsequent enzymatic analysis of the glucose residues (20).

RESULTS

The morphological characteristics of the vastus lateralis determined from the postexercise muscle biopsy samples showed that the 10 athletes had a mean of 80 ± 10% type I and 20 ± 10% type II fibers, and respective mean fiber areas of 3,178 ± 763 and 2,041 ± 322 μm². The relationship between speed (V) and VO2 during running was given by the equation

\[ V (\text{km/h}) = 2.103 + 0.244 \times \text{VO2 (ml·kg}^{-1}·\text{min}^{-1}) \]; \ r = 0.94

During the 4 h of exercise the mean VO2 was 2.91 ± 0.09 l/min, but with respect to time the VO2 increased gradually, the rise becoming significant (P < 0.01) after 110 min of exercise. However, during the last 3 h of exercise the rise in VO2 (6.1 ml·kg}^{-1}·\text{min}^{-1}) was effectively linear

\[ \text{VO2 (ml·kg}^{-1}·\text{min}^{-1}) = 42.73 + 0.031 \times t (\text{min}); \ r = 0.98, n = 80 \]
and increased the relative work load from 67.0 to 76.1% (+9.1%). A rise of $\text{VO}_2$ of 6.1 ml kg$^{-1}$ min$^{-1}$ over the period of 3 h is equivalent to an increase of speed of 1.49 km/h. The rise in $\text{VO}_2$ was associated with an increased minute ventilation ($\text{Ve}$) mainly due to a rise in respiratory frequency, tidal volume remained fairly constant during the exercise period. The changes in respiratory exchange ratio (R) were small, R increased from 0.8 at rest to 0.84 at the 30th min, and then declined. After 3 h of exercise R was 0.79 ± 0.05, and at the 225th min of exercise it was 0.80 ± 0.05. Blood lactic acid concentration (LA) remained relatively unchanged during the 4 h of exercise and close to resting values. After 45 min of exercise LA was 0.6 ± 0.2 mmol/l compared with 0.7 ± 0.2 mmol/l at the 165th min and 0.9 ± 0.3 mmol/l at the end of the exercise period (Fig. 1). Similarly, blood glucose concentration varied little during exercise, with only a marginal ($P > 0.1$) decrease from 5.6 ± 1.1 to 5.2 ± 1.1 mmol/l being observed from the 45th to the 225th min. Plasma glycerol was significantly ($P < 0.05$) raised after 15 min of exercise and continued to rise during the work and the immediate postexercise period. The FFA concentration tended to decline initially, and it was not until the 105th min of exercise that the mean FFA level was significantly ($P < 0.05$) elevated above resting levels. However, beyond this time FFA increased continuously during work and showed a marked rise during the recovery period. The increased FFA concentration during work and the immediate postexercise period was (negatively) associated with R ($r = -0.94$ and -0.97, respectively). Estimates of the metabolic mixture from the R data suggested that 70% of the energy demands during the final 2 h of exercise were supplied by the oxidation of fat. The mean postexercise muscle glycogen content determined from vastus lateralis needle-biopsy samples was 179.8 ± 42.2 μmol/g dry wt.

Cardiac frequency ($f_H$) rose as a linear function of time from the 80th to the 240th min

$$f_H (\text{beats/min}) = 143 + 0.124 \text{ t (min)}; \ r = 0.99$$

but $\dot{Q}$ remained essentially constant during exercise. After 20 min of running $\dot{Q}$ was 18.9 ± 1.5 l/min compared with 18.4 ± 2.2 and 18.6 ± 2.5 l/min at the 140th and 240th min, respectively, ($P > 0.1$). Thus the rise in $f_H$ was a reciprocal function of stroke volume ($SV$), which decreased ($P < 0.001$) from 130 ± 23 ml at the 10th min to 107 ± 16 ml at the 240th min of exercise.

The rise in $f_H$ during the final 100 min of exercise was also associated with $T_{re}$ ($r = 0.99$). During exercise $T_{re}$ rose from 38.43 ± 0.31°C at the end of the 1st h to 39.09 ± 0.50°C at the 4th h. During the same period $T_{sk}$ fell from 28.60 ± 1.43 to 27.38 ± 1.42°C. The rise in $T_{re}$ was associated with an increase in $M$ and the resulting change in %$\text{VO}_2_{\text{max}}$. The gradient between core and skin temperatures increased markedly after 90 min of work and continued to rise throughout the remaining exercise period. Thus, despite the increase in $M$, $K$ remained effectively constant during the latter part of work. After the 1st h of exercise $\text{Hct}_{\text{max}}$ remained constant until the end of work. The cumulative sweat loss of the subjects was a linear function of time

$$\Delta \text{sweat loss (g)} = 101.1 + 14.70 \text{ t (min)}; \ r = 0.99$$

At the conclusion of the 4 h of exercise mean sweat loss accounted for 5.5 ± 0.8% of the subjects' body weight, the total weight loss in absolute terms being 3.49 ± 0.50 kg.

There was a considerable variability in the volume of water ingested during the exercise period, ranging from 0 to 1.49 liters (mean 864 ± 454 ml). Although the one subject (LH) who chose not to drink during exercise demonstrated the largest increase in $T_{sk}$, there was no correlation between the volume of water consumed and the 240th min $T_{sk}$ for the group, nor was individual water intake related to sweat loss. Despite the progressive hypohydration, there was no evidence of hemoconcentration; the pre- and postexercise hematocrit (Hct) and plasma protein concentration values were similar. The respective values before and after 4 h of exercise were 43.3 ± 2.0 and 43.6 ± 1.8, and 7.4 ± 1.6 and 7.9 ± 1.4 g/100 ml. The similarity of pre- and postexercise Hct and plasma protein concentration suggests a stable plasma volume (PV).

Finally, the results of the muscle function tests taken immediately before and after the cessation of exercise are summarized in Table 1. None of the electrically evoked contractile and force-generating properties of the quadriceps are significantly ($P > 0.1$) different pre- and postexercise. The only difference in muscle function observed was during voluntary contractions: the force of MVC was reduced ($P < 0.001$) by ~25% in each leg, and the endurance time of a sustained isometric contraction at 30% MVC was decreased ($P < 0.001$) from 198 ± 78 to 82 ± 46 s, a decrement of 60 ± 6.5%.

**DISCUSSION**

The group of ultramarathon athletes studied was active in competition and may be regarded as representative of those who regularly compete in long-distance events in the United Kingdom. Their age, weight, stature and $O_2$ cost of running at given speeds are similar to a group of endurance runners previously studied in the laboratory (9), though their mean $\text{VO}_2_{\text{max}}$ is lower. In addition, the present athletes are characterized by having
a relatively high (80 + 10%) proportion of type I muscle fibers, a reduced mean fiber area of type II fibers, and low body fat content. They were all capable of running the traditional marathon distance in under 2.75 h, which is well within the first class standard for athletic members of the Road Runners Club of Great Britain. At the end of the 4 h of exercise all the subjects were complaining of fatigue, and we had the greatest difficulty in persuading the athletes to continue running during the last 30 min of work. On a rated (Borg) perceived exertion scale the athletes indicated a score of 19 (maximum 20) during the final stages of the exercise, and they had to be assisted from the treadmill at the end of the work period. By any subjective criteria they were distressed and manifestly exhausted at the conclusion of the experiment, and yet, despite the comprehensive nature of our physiological measurements, the etiology of their fatigue still remains unclear. The results provide no evidence of metabolic, cardiovascular, thermoregulatory, or electrically evoked muscle function impairment during (or following) 4 h of continuous exercise performed at the upper limit of their sustainable %VO\textsubscript{2,max}.

During exercise the mean total energy expenditure of the athletes was 14,146 kJ, of which 63% (8,912 kJ) of the metabolic mixture as indicated by the R data was fat and 37% (5,234 kJ) carbohydrate. At 10 min carbohydrate and fat contributed equally to energy needs, but by 170 min to the end of exercise fats were the main fuel supplying 70% of the metabolic requirements. It is interesting to note that despite the high relative exercise intensity (67-73% VO\textsubscript{2,max}) fats contributed at least half of the energy requirements during the early stages of the 4 h run, at a time when carbohydrate (in the form of glycogen) was readily available to the working muscles. The ability to mobilize and utilize a large fraction of fuel needs in the form of fats is well known to characterize endurance athletes (15). Endurance training increases the oxidative capacity of the exercising muscles through increased mitochondrial content (21) and aerobic enzymatic activity (18) and promotes a glycogen sparing effect through the enhanced use of fat as an oxidative substrate (24). Our observations would support the view that the enhanced ability to utilize fat as fuel is an important factor governing performance in prolonged running.

The ratio of carbohydrate to fat (30:70) remained virtually unaltered during the final 2.5 h of the treadmill run, suggesting that the fuel flux was constant and adequately maintained. At least without a further shift in R during the final stages of exercise it can be assumed that carbohydrate oxidation, although quantitatively small, was being maintained from available (but diminishing) carbohydrate stores. We found no evidence of hypoglycemia; blood glucose concentration was well maintained during exercise and muscle glycogen stores were not exhausted, despite the increased aerobic requirements of the exercise from 50 to 240 min (Fig. 1).

The measured mean glycogen content of postexercise biopsy samples taken from the vastus lateralis muscle of six of the athletes was 130 \(\mu\)mol/g dry wt muscle. This value represents an estimated 47-63% reduction over normal resting values (see Ref. 13, 17), suggesting that glycogen depletion per se was not a factor leading to the state of exhaustion observed at the end of exercise in the athletes studied. However, such a conclusion must be tentative, since other muscles known to be active in running (e.g., gastrocnemius and soleus) were not sampled, preexercise biopsies were not taken, nor was glycogen content determined with respect to specific fiber types. Costill et al. (6) have shown that selective glycogen depletion of type I fibers is an important factor in prolonged running, and thus our glycogen content determination may not accurately reflect the specific state of the active fibers involved in the exercise. Further studies would be required before definitive conclusions can be made.

The increased plasma FFA concentration was accompanied by a rise in plasma glycerol concentration, suggesting that the rate of release from adipose tissue exceeded the rate of FFA uptake by the skeletal muscle. Theoretically, the ratio of glycerol (resulting from the splitting of triglycerides) and FFA should be 1:3, but the increase in glycerol was more pronounced. Since glycerol is not utilized by the muscles, the difference in this ratio suggests a rapid utilization of FFA. If the rise in plasma glycerol is attributed to the mobilization of fats, then it is difficult not to avoid the conclusion that during the 1st h of exercise FFA is being utilized at an increased rate relative to the resting condition. This may be due to a disparity existing between the initial uptake by the exercising muscles and the onset of lipolysis and release of FFA from adipose tissue. Although triglycerides were not determined in the present investigation, studies during exercise (16) have shown that intramuscular and plasma triglycerides are reduced in response to sustained effort, though this source of fuel is quantitatively less important than FFA. Why fat oxidation cannot be used exclusively for the support of energy needs of the muscle

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**TABLE 1. Electrically evoked and voluntary quadriceps muscle function before and after 4 h continuous exercise**

<table>
<thead>
<tr>
<th>Force, % (Tetanus\textsubscript{100}/Tetanus\textsubscript{90})</th>
<th>Relaxation Times, ms</th>
<th>MVC, N</th>
</tr>
</thead>
<tbody>
<tr>
<td>SF\textsubscript{60}</td>
<td>SF\textsubscript{50}</td>
<td>Right leg</td>
</tr>
<tr>
<td>Preexercise</td>
<td>82.3±6.7</td>
<td>119.4±5.6</td>
</tr>
<tr>
<td>Postexercise</td>
<td>81.7±6.2</td>
<td>112.1±5.1</td>
</tr>
<tr>
<td>Normal values</td>
<td>76.9±6.5</td>
<td>104.7±9.4</td>
</tr>
</tbody>
</table>

Values are means ± SD. See Ref. 13 for description of normal values. Ratio of tetanic forces at 20 and 50 Hz, relaxation times at the cessation of exercise as described by Edwards et al. (13), and maximal voluntary contraction (MVC). *P < 0.001. † Predicted from body weight.
during prolonged severe exercise is not clear. Rose and Goresky (25) have indicated that the barrier in the endothelial cells of the capillaries limit the translocation of FFA from the blood into the active muscle, suggesting that the FFA utilization is not limited by the processes of oxidation but by the rate of transport from the plasma into the muscle.

Observed plasma lactate concentration in the present study remained virtually unchanged during the 4 h of exercise and close to resting levels. This is in agreement with a number of studies (e.g., Ref. 2) that indicate that plasma lactate accumulation is not a limiting factor in prolonged exercise. Indeed, plasma and muscle lactate may be used as fuel during prolonged exercise in the presence of low glycogen levels (16), and this may account for the low values of lactate we have observed. Thus from the data we have obtained it is difficult to ascribe the degree of exhaustion observed at the end of 4 h of continuous exercise to metabolic factors. Muscle glycogen was present in the postexercise muscle biopsies of vastus lateralis, R remained virtually unchanged for the final 2.5 h of exercise, plasma glucose concentration was well maintained, and there was no appreciable accumulation of plasma lactic acid.

During the 4 h of exercise a gradual rise in aerobic metabolism and cardiac frequency was observed. Similar observations have been previously reported by several authors during prolonged exercise (e.g., Ref. 28). The rise in fH was associated with VO2, though Q was maintained at a constant level throughout exercise. Thus SV decreased and the arterial-mixed venous O2 difference increased gradually during the 4 h of exercise. The mean rise in fH from 10 to 240 min of exercise was 25 beats/min, which represents an increase of 17%, the final value of fH at the cessation of work being 93% of the maximal fH of the athletes. Thus, although these observations provide evidence of severe cardiac stress, it is unlikely that the central circulation was impaired; fH and Q remained within the expected physiological range and were well maintained during the 4-h period of exercise. The mean increase of arterial mixed venous O2 difference from 107 ml/l at 10 min to 158 ml/l at 240 min appeared sufficient for local tissue requirements, the plasma lactate concentration remained relatively unchanged during the duration of the exercise and did not exceed 1 mmol/l. Thus tissue perfusion and oxygenation were adequately served by Q.

The mean rise in absolute VO2 from 50 to 240 min of exercise was 239 ± 118 ml/min and effectively increased the relative workload by 5.5% (66.5–72% VO2 max), despite a constant treadmill speed and a significantly (P < 0.001) reduced body weight. The body weight loss primarily due to sweating accentuated the rise in VO2 when expressed in terms of milliliters per kilogram per minute, the increase being 6.1 ml·kg⁻¹·min⁻¹ (19.1% VO2 max) between 50 and 240 min of exercise. A loss of body weight would be expected to reduce the workload if the weight loss was not integral to the metabolic needs of the exercise. A rise in VO2 of 6.1 ml·kg⁻¹·min⁻¹ is equivalent to an increase in running speed of 1.49 km/h (0.47 km/h for each hour of work), which undoubtedly contributed to the state of exhaustion of the athletes during the concluding stages of the treadmill exercise. However, the underlying mechanisms are not altogether clear.

It is well known that in healthy normal subjects as exercise progresses R declines and the metabolic mixture gradually switches from carbohydrate to fat. The utilization of fat as a substrate is known to increase (~7%) the O2 requirement of exercise. However, in the present study R is effectively constant (Fig. 1) during the final 3 h of work, the period in which the major change of VO2 is observed. It is possible that the changes in VO2 may have arisen through increased work of the respiratory muscles and the rise in body temperature during the course of the exercise, but if so they would be small and unlikely to affect the VO2 significantly. In the present experiments the change in respiratory frequency, VE, and V̇E between the 1st and 4th h were 8 breaths/min, 16.1 l/min, and 0.63°C, respectively. Our own view is that the loss of apparent mechanical efficiency must be sought at the level of the exercising muscles. Bink (4) demonstrated a decrease in mechanical efficiency with exercise extending from 5 to 200 min and explained this in terms of the need to use auxiliary muscle groups to maintain performance. The occurrence of increased shoulder rotation, head movements, and loss of mechanical coordination would undoubtedly give rise to an increased requirement for O2 during exercise. However, our subjects were able to maintain their natural stride frequency (mean, 86 strides/min), pattern, and running form during the major part (3.5 h) of the work period. It was only during the last 15–30 min of work that some of our athletes became noticeably less coordinated and to use more arm, shoulder, and torso movement in their running action. This may have contributed to the rise in VO2 during the latter stages of the exercise but would not explain the changes in VO2 prior to the final hour of running. A more plausible explanation, therefore, may be related to a decline in muscle function per se rather than a loss of muscular coordination. A gradual decrease in muscle strength will place an increased load on the force-generating capability of the muscle and may necessitate an increased recruitment of fibers for force maintenance if speed is to be sustained. Petrofsky (22) has shown that the number of fibers active in a muscle is proportional to VO2 during exercise. Subjects in the present study were unable to reproduce static muscle contractile forces following the 4-h exercise period compared with preexercise values. The force of MVC of the leg extensors was reduced by 25%. If in overcoming this impairment of muscle function more fibers are recruited for the generation of a given force, then one would expect physiological efficiency to fall and VO2 to rise gradually during exercise. However, if this is so, our electrically evoked contraction data (Table 1) would suggest that impairment of voluntary force generation is not due to a failure of the contractile machinery per se, since the mechanical properties of the tetanus remain unchanged following the exercise. The stimulation results clearly implicate the central nervous system and suggests that the loss of force may be due to involuntary inhibition resulting in a decreased central neural drive to the ex-
ercising muscles. Clearly this is an important area for future research.

The raised \( V_{O2} \) and apparent loss of mechanical efficiency increased the amount of metabolic heat to be dissipated for thermal balance to be maintained. In a previous study of marathon runners (7) it was shown that performance at marathon speeds was dependent on the maintenance of a cool skin and minimizing peripheral blood flow. During exercise there was evidence of peripheral vasoconstriction, and the convective transfer of heat from core to skin was enhanced by a rise in deep body temperature. The data from the ultramarathon athletes, who ran the equivalent of 30–40 miles during 4 h on the motor-driven treadmill in the present investigation, would suggest that they adopt a similar thermal strategy to the one described (9) for endurance athletes running shorter distances but at higher relative workloads. Beyond 80 min of exercise in the ultramarathon athletes, \( T_{sk} \) decreases as an approximate linear function of time and \( T_r \) rises. However, in contrast to previous experiments (9) on marathon runners, the increase in \( T_{sk} \) is proportional to the rise in \( M \) and %\( V_{O2_{max}} \); there is no evidence of a spiraling increase in \( T_{sk} \) and loss of thermal control during the latter part of exercise. During running at 66–73% \( V_{O2_{max}} \) for 4 h \( T_r \) remains with the normal physiological range for exercise and at no point exceeds 39.3°C, a value of core temperature usually taken to represent the upper limit of physiological thermal regulation in humans (see Ref. 8). Sweat rate increases cumulatively as a linear function of time, and there is no evidence of saturation of the sweating mechanism. The athletes tolerated sweat losses equivalent to ~5.5% body wt without apparent discomfort. Despite this level of dehydration, the athletes ingested very little water (which was freely available) during the 4 h of exercise (mean 864 ± 454 ml) and plasma protein and Hct remained fairly constant during the work period. By using the method of Van Beaumont et al. (29), which is based on Hct, the maximum calculated decrease in PV is ~<5%. Thus despite the large sweat losses PV appears to be well maintained during exercise, a finding in agreement with earlier studies (27).

Finally, the gradual increase in aerobic metabolism from 50 to 240 min of exercise found in this study has important implications for performance in ultramarathon running. Examination of the records of the British Road Runners Club would suggest that a typical race profile for ultramarathon events is one of declining speed following the first 15 miles of the race. The decrease in speed accords with the increase in \( V_{O2} \) we have found during 4 h of running on a treadmill. To offset the increased aerobic requirements of continuous prolonged running we would have found it necessary to reduce the speed of the treadmill bed by 0.47 km/h each hour after the first 1 h of exercise. This would have effectively maintained \( V_{O2} \) at a constant level and negated the rise in relative workload. The possibility arises, therefore, that the experienced ultramarathon athlete during competition is able to judge pace to such an extent as to maximize the use of his available aerobic power and effectively sustain an iso relative energy expenditure level throughout the whole period of the race.

The experiments were carried out at the London School of Hygiene and Tropical Medicine in 1978/9.

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