Life Sciences Vol. 14, pp. 187-198, 1974.
Printed in Great Britain

Pergamon Press

RESPIRATORY RESPONSES TO INTERMITTENT AND PROLONGED EXERCISE IN A HOT-DRY ENVIRONMENT

Kent B. Pandolf[1] and Eliezer Kamon

Human Energy Research Laboratory and Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

(Received 21 February 1973; in final form 15 November 1973)

Summary

Ventilatory gas exchange ratio (R), $\dot{V}O_2$, ventilation ($\dot{V}_E$), respiration rate (RR), rectal temperature (T_{re}), and heart rate (HR) were determined for four acclimatized subjects during intermittent and prolonged exercise on a treadmill at 24° and 45°C (dry) as follows: 1) 8 cycles (10 min. exercise and 5 min. rest), and 2) prolonged exercise lasting for 90 min. While during intermittent and prolonged exercise, $\dot{V}O_2$ and $\dot{V}_E$ did not differ in the heat, RR, T_{re}, HR and the respiratory dead space were higher in the hot ambient environment. After steady-state attainment, exercise R was higher in the initial as compared to the last cycles with higher values in neutral as compared to the hot ambient condition. It was concluded that heat was more effective than time in lowering the R, probably with a greater dependence on fat oxidation in the latter exercise cycles which seemed to be more pronounced in the heat.

Introduction

No study has adequately investigated the time course of the ventilatory reactions to heat while exercising (6). It has been found that the ventilatory gas exchange ratio (R) progressively decreased during prolonged moderate to heavy exercise performed in neutral (10,19) as well as in hot environments (3). But the R values found for exercise in hot environments were higher than those reported for the same exercise level under neutral environmental conditions (3,14). These reported reversed effects of heat and prolonged exercise on R do not reconcile with other reports in the literature which suggest that the effect of heat stress should resemble that of prolonged exercise in the sense that both activate agents, such as catecholamines, which mobilize free fatty acids (FFA) (4,16). Consequently, the increased plasma FFA serves as an

---

[1]Present Address: Military Ergonomics Laboratory, U.S. Army Research Institute of Environmental Medicine, Natick, Massachusetts 01760.

available metabolic substrate at the submaximal work level (10) which should finally be reflected in a lower R for both exercise in the heat and the latter part of prolonged exercise as compared to that at neutral and at the early part of the exercise, respectively.

As a preliminary step in elucidating the effect of heat on ventilatory and metabolic events during exercise, we followed the ventilatory gas exchange for prolonged exercise at about 50% of the maximal aerobic capacity of each individual at neutral (24°C) as well as hot (45°C) environments. Assuming that changes in R will also affect the transient ventilatory gas exchange, we also followed the ventilatory gas exchange during repeated intermittent exercise hoping to gain better insight into the mechanism(s) of bodily adjustment to stress.

Methods

Two subjects participated in both the steady-state prolonged exercise and the intermittent exercise. The other two subjects participated in either the prolonged exercise (L.T.) or intermittent exercise (M.S.) only (Table 1). The participation schedule, age, physical characteristics, maximal oxygen uptake ($\dot{V}O_{2max}$) and relative $\dot{V}O_2$ during exercise are given in Table 1 for each subject. Maximal oxygen uptake was obtained on an inclined treadmill (17). The subjects were acclimatized to the heat by 10 days of daily walking for 2 hours at 3.5 mph at 45°C. Mondays were utilized for reinforcement of acclimatization. All subjects wore gym apparel consisting of shorts, socks and sneakers.

Each test was repeated at 24°C and 45°C with the vapor pressure at about 12 mm.Hg. and the air velocity at about 0.5 m/sec for both temperatures. All testing was carried out in the afternoon. The intermittent testing sessions lasted 130 minutes. The first 10 minutes of pre-exercise (seated in the climatic testing room) data collection was followed by eight cycles of 10 minutes exercise and 5 minutes recovery. All subjects walked at 3.5 mph at 7-9% grade which yielded approximately 50% of their $\dot{V}O_{2max}$ (Table 1). Prolonged exercise which followed a 10 minute pre-exercise period was conducted continuously for 90 minutes and was therefore comparable to the total exercise completed during the eight cycle intermittent sessions (80 min.).

TABLE 1

Physical Characteristics of the Subjects

Subject	Prolonged Exercise	Age (yrs)	Wt (kg)	Ht (cm)	$\dot{V}O_{2max}$ l/min	$\dot{V}O_{2max}$ ml/kg·min	% of $\dot{V}O_{2max}$ Submaximal Work
L.T.* (Trained)	+	19	61.5	171.1	3.83	62.23	55%
K.C. (Trained)	+	20	66.3	167.5	4.11	62.02	51%
M.S. (Trained)	-	22	67.2	178.2	4.64	69.01	49%
K.P. (Physically Active)	+	25	87.3	175.9	4.05	46.41	48%

*Participated in prolonged exercise only.

During intermittent exercise and recovery, breath to breath expired air volume, O_2 and CO_2 concentration, heart rate and rectal temperature were continuously monitored as follows: during the last minute of the resting period and the first 5 minutes of the exercise and then during the last minute of exercise and the 5 minutes of the resting period of each cycle. Not all the cycles in a session were monitored. In the eight cycle session (10 minutes exercise and 5 minutes of rest) the first, fourth, fifth and eighth cycles were monitored. During prolonged exercise the same circulo-respiratory measurements were conducted as follows: during rest and the first minute of exercise and then during the 4th, 9th, 19th, 39th, 59th, 69th, 79th and 89th minutes of exercise.

To measure O_2 and CO_2 concentrations during intermittent and prolonged exercise, the Westinghouse Pulmonary Oxygen Monitor (model 211) and the Beckman LB-1 infrared CO_2 analyzer were used respectively. Three breaths were measured and averaged during each 30 second measurement period for the computation of end-tidal O_2 and CO_2. The recorded peak value of each breath for both O_2 and CO_2 was taken as the end-tidal measurement. Expired gas was sampled continuously at the mouth with end-tidal measurements of O_2 and CO_2 used to approximate their respective alveolar concentrations. Both the CO_2 and O_2 analyzers were fast-response instruments with the 90% response time being 0.16 sec. for the CO_2 analyzer and 0.20 sec. for the O_2 analyzer. Tidal

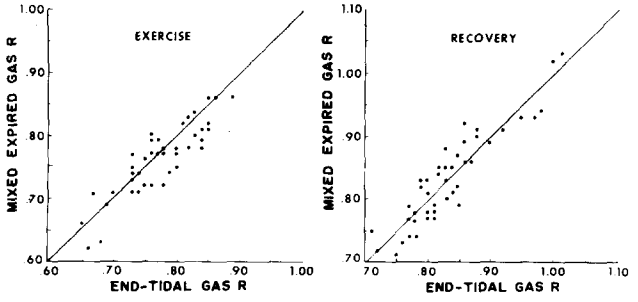
volumes were measured using a Fleisch Pneumotachograph with a Sanborn transducer. A Beckman Integrator was utilized to integrate the expired airflow curve in order to obtain volume. Gas concentrations and expired air volume can be integrated and multiplied at each measurement period to give O_2 uptake ($\dot{V}O_2$) and CO_2 production ($\dot{V}CO_2$) and therefore the ventilatory gas exchange ratio for each breath considering appropriate correction factors (STPD). Both analyzers were calibrated before and after each test with tank gases of known concentration. Similar methods presented in greater detail were recently reported (2). Heart rate was continuously recorded by integrations of R-R time intervals. A 30 gauge copper constantan thermocouple was used to measure rectal temperature. All the above mentioned parameters were continuously monitored on a 6 channel Beckman type R dynograph.

The R values calculated from end-tidal O_2 and CO_2 concentrations were compared with R determined by analysis of mixed expired gas during both exercise and recovery. Mixed expired gas was collected in a Tissot spirometer in order to measure ventilation. The O_2 concentration of the mixed expired gas was measured with a Beckman C_2 oxygen analyzer employing an expanded scale. The CO_2 concentration of the mixed expired gas was measured with the same type of CO_2 analyzer previously mentioned. The results obtained by these two methods of determining R agree quite well and are illustrated in Fig. 1.

The two-way analysis of variance was utilized to test for differences between cycles and environments. Since the variance between the three subjects was found to be small, the results will be presented using the mean response.

FIG. 1

Comparison of R from end-tidal gas concentrations with that determined by analysis of mixed expired gas during exercise and recovery.

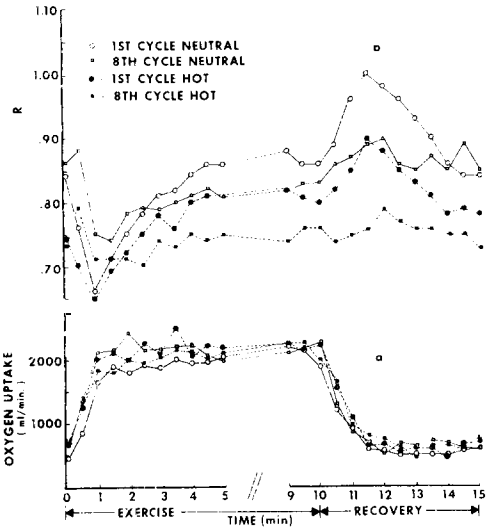


Results

Figures 2 and 3 represent the results yielded for the 8 cycles of intermittent exercise and recovery, and Fig. 4 for the prolonged continuous exercise. Figure 2a describes the time course of the oxygen uptake (1/2 minute values averaged for the 3 subjects) during the first and the eighth exercise-rest cycles of the 2 hours intermittent exercise in both neutral and hot ambient environments. It can be seen that during the transience and the steady state the $\dot{V}O_2$ for the 2 cycles under the different ambient environments is practically the same. However, as Fig. 2b shows the time course of the ventilatory gas exchange (R) computed and averaged at the same intervals as the $\dot{V}O_2$, is different for the two ambient environments during transience (exercise onset to 3 min.) and during steady state (3 1/2 min. to exercise termination) ($P < .01$). At steady state the difference between the separate cycles was also significant ($P < .01$).

FIG. 2

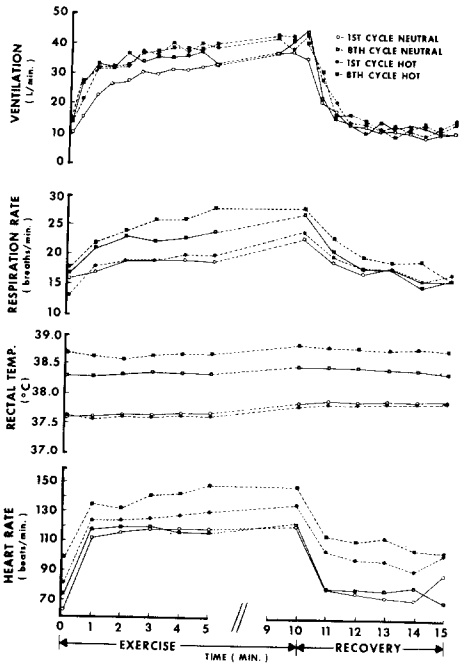
Oxygen uptake (a) and the ventilatory gas exchange - R (b) during the 1st and 8th exercise-rest cycles at 24°C and 45°C.



At the initiation of each cycle in the heat, the R is lower than that in the neutral conditions. It should be recalled that the initial value for the first cycle is followed by 10 minutes sitting in the testing ambient conditions.

FIG. 3

Ventilation, respiration rate, rectal temperature and heart rate during the 1st and 8th exercise-rest cycles at 24°C and 45°C.



The exercise transience of R for the first cycle at both environments can be separated into the following phases: (a) a drop during the first minute and then (b) rise toward steady state within the next 2 1/2 minutes. However, the first phase in each of the last cycles shows a rise prior to the fall in R. Thus, the last cycles can be separated into three phases with the first phase being an initial increase followed by the two phases seen in the first cycles. The last phase of the exercise transience has a faster rise as follows: in the first cycle as compared to the last and during exercise in the neutral as compared to that in the hot ambient environment. These differences are further reflected in the remaining period of the exercise cycle ("steady state") where the R for the first cycle is higher than for the last ($P < .01$), and in the neutral condition it is higher than in the heat ($P < .01$).

The differences in R observed between the cycles and between exercise at the different ambient environments are further seen during the recovery (Fig. 2b); the highest initial rise followed by the sharpest rate of decrease is

seen in the first cycle at the neutral condition as compared to the other cycles ($P < .01$).

Figure 3 describes the exercise-recovery time course for the same cycles shown in Fig. 2 but for the following physiological responses: ventilation ($\dot{V}_E$), respiration rate (RR), rectal temperature (T_{re}) and heart rate (HR). $\dot{V}_E$ was not significantly different between cycles or under different ambient conditions although in the heat it was slightly higher. RR and T_{re} were the same during the first cycle in the heat and in the neutral ambient environments but both rose progressively to the point where during the last cycle they were higher than in the first cycle more so in the hot than in the neutral ambient environment ($P < .01$). HR in the heat was higher than in the neutral conditions and while it stayed at the same level throughout the 8 cycles in the neutral it rose progressively in the heat so that the 8th cycle yielded higher HR during transient as well as during the steady-state phases as compared to the first cycle ($P < .01$).

FIG. 4

R, oxygen uptake, ventilation, respiration rate, rectal temperature and heart rate during prolonged exercise of 90 minutes duration at 24°C and at 45°C.

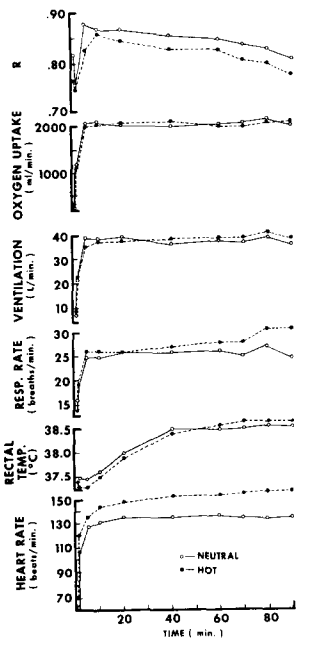


Figure 4 describes the time course of the physiological responses to continuous exercise lasting 90 minutes at the same level and in the same two ambient conditions as has been used for the intermittent exercise. As in Figs. 2 and 3 the values shown in Fig. 4 are the means for 3 subjects. Unless otherwise mentioned steady state will be assumed between about the 4th and the 90th minute. It can be seen that the steady-state R, for work in the heat is lower as compared to that in the neutral ambient environment. The mean values were found to differ significantly ($P < .05$). The $\dot{V}O_2$ and $\dot{V}_E$ values that were obtained throughout the 90 minutes of exercise were the same for the neutral and the hot-dry environments. While steady-state RR was achieved during the prolonged exercise in the neutral conditions it started to progressively rise after 30 minutes of work in the heat. This rise renders a significant RR difference between the two ambient conditions ($P < .05$). T_{re} for the two ambient environments did not differ until an hour after the onset of the work, when in the heat it starts to progressively rise. The elevated T_{re} for exercise in the heat was significantly higher than the corresponding responses in the neutral condition during the last 1/2 hour of experimentation ($P < .01$). Expectedly, steady-state HR in the heat is higher than in the neutral conditions ($P < .01$).

Discussion

This study was attempted to compare the effect of time on exercise ventilatory gas exchange (R) to the effect of heat during continuous and during intermittent exercise. Indeed the consequences of these two factors were similar in decreasing R although heat was more effective in lowering R. These two observed factors, time and heat, showed no effect on the $\dot{V}_E$ and on the $\dot{V}O_2$, thus the observed decrease in R with time or due to the hot ambient environment could be attributed mainly to changes in the elimination rate of CO_2 and partially to changes in the respiration rate (RR).

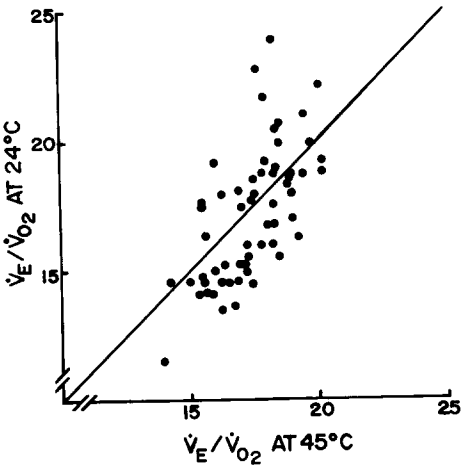
The rapid changes in $\dot{V}_E$ during the initiation of exercise at both ambient environments (64% of mean steady-state $\dot{V}_E$ achieved at 15 seconds of exercise onset) lend support to a strong neutral component and are in agreement with those reported by others (5). Pre-exercise anticipation was not considered to be a factor affecting the rapid changes in ventilation at the onset of exer-

cise because all subjects were highly acquainted with treadmill walking and the experimental protocol.

The causes for the changes in transient R values seem to differ from those affecting the steady-state values. Actually the exercise and recovery transient R values revealed some differences in trend only between the ambient environments in the intermittent exercise, and between cycles and environments during recovery. The rise towards steady state was slower in the last as compared to the first cycle under the two ambient conditions. According to previous observations by others (11,12) these differences reflect the changes in the storage of the gases in the body; oxygen intake in the exercise and ridding of CO_2 in the recovery transience. The initial rise in transient R observed in both 8th exercise cycles may be a compensatory mechanism for the less pronounced elimination of CO_2 during the prior recovery period.

FIG. 5

The $\dot{V}_E$ / $\dot{V}O_2$ from 4 subjects while exercising at 24 and 45°C. These data represent transient and steady-state responses about a line of identity.



While, as we could show, heat affected the transient R values it was also clearly effective in lowering the steady-state R as compared to the values yielded under neutral ambient environments. This was in disagreement with previous reports showing higher R values under hot as compared to neutral am-

bient conditions (14). However, of the 27 subjects described in the work of Rowell and co-workers approximately half were not heat acclimatized, while the others were heat acclimatized and had been reported in previous papers (13,15). Hyperventilation was apparent in the heat and became most pronounced at the highest work loads. Although the work loads for these 27 subjects elicited up to 87% $\dot{V}O_{2max}$, none of heat acclimatized subjects exceeded 60% $\dot{V}O_{2max}$ (13,15). Since hyperventilation became pronounced at the higher work loads, we hypothesized that the effect is attributable mainly to the unacclimatized subjects. Figure 5 illustrates the $\dot{V}_E$ / $\dot{V}O_2$ from each of the 4 acclimatized subjects in our study while exercising at 24 and 45°C. These data do not indicate a trend to hyperventilate in the heat. Thus, the pronounced hyperventilation previously reported (14), attributed mainly to the unacclimatized subjects, may further account for an increased CO_2 elimination and the higher R values presented in that paper.

In this study the lower steady-state R values in the heat were observed in the prolonged as well as in the intermittent exercise. Despite the apparent lower R value from the beginning of exercise in the heat it continued to gradually decrease with time parallel to the decrease observed under the neutral ambient environment. During prolonged or intermittent exercise, the quantitative difference between the initial peak exercise R and that at exercise termination was the same for each of the two ambient environments, but the absolute difference was slightly greater during exercise in the heat (.06) than the neutral condition (.05). Such a gradual decrease in R during continuous prolonged work was also reported by others (8,19); however, it is interesting to note that this time course decrease was apparent also during the intermittent exercise, performed in this study, when 5 minutes of rest was allowed between the 10 minute exercise bouts. It was suggested that the gradual decrease in CO_2 elimination rate during exercise under neutral ambient environments was a result of the shift from carbohydrate to free fatty acid (FFA) metabolism as the store of carbohydrate is reduced (10,19). This might explain the further decrease in the R value during exercise in the heat since hot ambient environments were found to enhance the excretion of the catecholamines, norepinephrine and epinephrine, (4,16) which may result in a greater

mobilization of FFA.

One puzzling observation was that already on the initiation of exercise in the heat the R values were lower than in neutral (see Figs. 2 and 4) while RR or $\dot{V}O_2$ did not change. This value followed the 10 minutes of rest in the climatic chamber and one could expect some changes in alveolar dead space gas interchange. Upon calculation of the respiratory dead space at rest using the BOHR formula, the dead space in the heat was higher (211 cc) than that in the neutral condition (140 cc). But during both exercise and recovery the relative difference (≃70 cc) remained about the same while the absolute dead space during exercise in the heat reached 306 cc and seemed to be in agreement with previous reports (1). The increased respiratory dead space in the heat may explain the reduction in both end-tidal CO_2 and the R, particularly during rest, which may result from a reduced perfusion of the lung in the heat because of a pooling of blood to the skin. These findings may also partially explain the exercise and recovery transient differences in R between ambient environments, especially at lower tidal volumes, where the relative difference in the respiratory dead space might be an important contributing factor. However, the possible effect of the increased RR on the R values in the later periods of exercise cannot be excluded. Despite the fact that the ambient conditions allowed free evaporative cooling by sweating, RR started to rise at about 1/2 an hour after the beginning of the exercise coinciding with the onset of rise of T_{re}. These two parameters, RR and T_{re}, were most likely related to each other. The lower R values for exercise in the heat may also be associated with decreased lactic acid concentrations in the blood as previously seen by others (7).

Expectedly, the HR did not reach steady state in the heat. This suggests some regulatory mechanism identical to most of the physiological responses in which achievement of a steady state at a preset value is possible only if the ambient conditions allow. In this respect HR, T_{re} and RR equilibrated at the neutral ambient environment but departed to a continuous rise when the ambient environment was stressful even during the intermittent exercise.

As a "bloodless" measure, breath to breath changes in R from other studies have been compared to corresponding arterial blood lactate and bicar-

bonate concentrations with the former measure reliably reflecting the metabolic acidosis of exercise (9,18). We have planned further experiments to measure arterial blood lactate, plasma bicarbonate and free fatty acids in conjunction with R during exercise in the heat which will help to further understand the regulatory mechanisms and shifts in metabolic events that result from our observed changes in gas exchange.

References

1. E. ASMUSSEN and M. NIELSEN, Acta Physiol. Scand. 38, 1 (1956).
2. W. L. BEAVER, K. WASSERMAN, and B. J. WHIPP, J. Appl. Physiol. 34, 128 (1973).
3. L. BROUHA, P. E. SMITH, R. DELANNE and M. E. MAXFIELD, J. Appl. Physiol. 16, 133 (1960).
4. T. COOPER, V. L. WILLMAN and C. R. HANLON, Diseases Chest. 44, 88 (1963).
5. E. D'ANGELO and G. TORELLI, J. Appl. Physiol. 30, 116 (1971).
6. P. DEJOURS, Handbook of Physiology, Respiration, American Physiological Society, Washington, D. C. (1964).
7. R. DELANNE, J. R. BARNES, L. BROUHA and F. MASSART, J. Appl. Physiol. 14, 328 (1959).
8. L. G. EKELUND and A. HOLMGREN, Acta Physiol. Scand. 62, 240 (1964).
9. A. NAIMARK, K. WASSERMAN, and M. B. McILROY, J. Appl. Physiol. 19, 644 (1964).
10. E. D. R. PREUTT, J. Appl. Physiol. 29, 809 (1970).
11. H. RAHN and W. O. FENN, A Graphical Analysis of the Respiratory Gas Exchange, American Physiological Society, Washington, D. C. (1955).
12. H. RAHN and A. B. OTIS, J. Appl. Physiol. 1, 717 (1949).
13. S. ROBINSON, E. S. TURRELL, H. S. BELDING, and S. M. HORVATH, Am. J. Physiol. 140, 168 (1943).
14. L. B. ROWELL, G. L. BRENGELMANN, J. A. MURRAY, K. K. KRANING and F. KUSUMI, J. Appl. Physiol. 26, 395 (1969).
15. L. B. ROWELL, K. K. KRANING, J. W. KENNEDY and T. O. EVANS, J. Appl. Physiol. 22, 509 (1967).
16. P. N. SYMBAS, M. JELLINEK, T. COOPER and C. HANLON, Arch. Int. Pharmacodyn. 150, 132 (1964).
17. H. L. TAYLOR, E. BUSKIRK and A. HENSCHEL, J. Appl. Physiol. 8, 73 (1955).
18. K. WASSERMAN, B. J. WHIPP, S. N. KOYAL, and W. L. BEAVER, J. Appl. Physiol. 35, 236 (1973).
19. K. WASSERMAN, A. L. VAN KESSEL and G. G. BURTON, J. Appl. Physiol. 22, 71 (1967).