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Oxygen uptake during swimming in a hypobaric hypoxic environment

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Summary. The purpose of this study was to determine oxygen uptake ($\dot{V}O_2$) at various water flow rates and maximal oxygen uptake ($\dot{V}O_{2max}$) during swimming in a hypobaric hypoxic environment. Seven trained swimmers swam in normal [N; 751 mmHg (100.1 kPa)] and hypobaric hypoxic [H; 601 mmHg (80.27 kPa)] environments in a chamber where atmospheric pressure could be regulated. Water flow rate started at 0.80 $m \cdot s^{-1}$ and was increased by 0.05 $m \cdot s^{-1}$ every 2 min up to 1.00 $m \cdot s^{-1}$ and then by 0.05 $m \cdot s^{-1}$ every minute until exhaustion. At submaximal water flow rates, carbon dioxide production ($\dot{V}CO_2$), pulmonary ventilation ($\dot{V}_E$) and tidal volume (V_T) were significantly greater in H than in N. There were no significant differences in the response of submaximal $\dot{V}O_2$, heart rate (f_c) or respiratory frequency (f_R) between N and H. Maximal $\dot{V}_E$, f_R, V_T, f_c, blood lactate concentration and water flow rate were not significantly different between N and H. However, $\dot{V}O_{2max}$ under H [3.65 (SD 0.11) $l \cdot min^{-1}$] was significantly lower by 12.0% (SD 3.4)% than that in N [4.15 (SD 0.18) $l \cdot min^{-1}$]. This decrease agrees well with previous investigations that have studied centrally limited exercise, such as running and cycling, under similar levels of hypoxia.

Key words: Hypobaric hypoxic condition – Swimming – Maximal oxygen uptake – Tidal volume

Introduction

The effect of hypobaric hypoxic environments on oxygen uptake ($\dot{V}O_2$) during various types of submaximal and maximal exercise in man has been previously reported (Pugh et al. 1964; Stenberg et al. 1966; Fagraeus et al. 1973; Gleser 1973; Hartley et al. 1973; Horstman et al. 1980; Fulco et al. 1988). In general, $\dot{V}O_2$ during submaximal exercise on land has not been found to change because of increased cardiac output ($\dot{Q}_c$) and increased pulmonary ventilation ($\dot{V}_E$) (Pugh et al. 1964; Ekblom et al. 1975; Hartley et al. 1973; Vogel et al. 1974; Roca et al. 1989). In water, in a normal environment, maximal voluntary ventilation has been shown to decrease due to greater hydrostatic pressure when compared with that on land (Ogita et al. 1989). In addition, $\dot{V}_E$ during swimming has been shown to be limited since respiratory frequency (f_R) is synchronized with the arm stroke (Holmér 1974a; Holmér and Gullstrand 1980). Therefore, it is probably incorrect to assume that results obtained on land are directly applicable to those of swimming. Furthermore, during exercise on land under the influence of hypoxia, maximal oxygen uptake ($\dot{V}O_{2max}$) has been shown to decrease significantly due to a reduced arterial oxygen content (Pugh et al. 1964; Stenberg et al. 1966; Fagraeus et al. 1973; Gleser 1973; Hartley et al. 1973; Horstman et al. 1980; Fulco et al. 1988). However, $\dot{V}O_{2max}$ during swimming under the influence of hypoxia has not yet been reported. The purpose of this study was to measure submaximal $\dot{V}O_2$ during swimming in water at various flow rates, and $\dot{V}O_{2max}$ during swimming in a hypobaric hypoxic environment.

Methods

Subjects. The subjects were seven trained male college swimmers. Their mean age, height, body mass and % body fat were 20 (SD 1) year, 174.0 (SD 1.1) cm, 64.8 (SD 3.7) kg, and 9.2% (SD 2.6)%, respectively. The mean personal best time of the subjects in the 100-m free style swimming event was 57.2 (SD 2.5) s. Before participating in this experiment, each subject was fully informed of the purposes, protocol, and procedure of the experiment, and any associated risks.

Experimental procedures. Using front crawl as the stroke style for this experiment, the subjects swam in both a normal [N; 751 (SD 2) mmHg, 100.1 (SD 0.27) kPa] and a hypobaric hypoxic environment [H; 601 (SD 0) mmHg, 80.27 (SD 0) kPa]. Measurements in N and H were performed 1 week apart. Since the subjects who

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participated in this experiment were often exposed to experiments and training in hypobaric hypoxic environments, the results obtained in both environments were not considered to be affected by the nonrandom order of their exposure to the two environments.

Swimming flume. The swimming flume was located in a chamber where atmospheric pressure could be regulated and water flow rate (0–2.0 $m \cdot s^{-1}$) could be controlled. To ensure accuracy, water flow rate was measured at depths of 10, 20, 30, and 50 cm from the surface with a calibrated flow meter (Nakaasasokki, Tokyo, Japan). The variations in each water flow rate experienced by the subjects were less than ±2%. This accuracy was reproducable during both increasing and decreasing flow rates. Water temperature was maintained at 27.8 (SD 0.3) °C throughout the experiment.

Protocol. The protocol to measure $\dot{V}O_{2max}$ was identical in both environments. The water flow rate was first set at 0.80 $m \cdot s^{-1}$ and was increased by 0.05 $m \cdot s^{-1}$ every 2 min to 1.00 $m \cdot s^{-1}$; then by 0.05 $m \cdot s^{-1}$ every minute until exhaustion (test 1). In test 2, each subject swam for 2 min at 0.80 $m \cdot s^{-1}$, which corresponded to 40%–60% of the subject's $\dot{V}O_{2max}$, when the water flow rate was quickly increased to supramaximal values. This supramaximal flow rate was 0.05 $m \cdot s^{-1}$ faster than that attained during the last minute in test 1. Each subject was allowed to rest for at least 20 min between test 1 and test 2. When heart rate (f_c) and blood lactate concentration ($[la^-]_b$) failed to regain resting values, the subject was allowed more recovery time until these values were achieved.

Measurements. The $\dot{V}O_2$, carbon dioxide production ($\dot{V}CO_2$), $\dot{V}_E$, respiratory exchange ratio (R), tidal volume (V_T), and f_R were all measured with an automatic analyser (Mijnhardt OXYCON4, Bunnik, The Netherlands). The automatic analyser was put above the swimmer on a movable platform which stretched across the swimming flume (see Fig. 1). The $\dot{V}O_2$, $\dot{V}CO_2$ were calculated at standard temperature and pressure (dry), and $\dot{V}_E$ and V_T were calculated in body temperature and pressure (saturated with water vapour). Gas was expired through a low resistance valve and tubing (inspired side: 1.0 m length, inner diameter 36 mm; expired side: 2.0 m length, inner diameter 36 mm) connected to the automatic analyser where the O_2 and CO_2 fractions in expired gas were determined by a paramagnetic O_2 analyser and an infrared CO_2 analyser, respectively. The gas analyser system incorporated a mixing volume and the time constant given for the whole system was 10 s. The gas analysers were calibrated with standard gas (O_2, 16.7%; CO_2, 3.92%) prior to testing under both N and H. Expired gas volume was measured by a dry-gasmeter and f_R was counted from measurements of temperature within the automatic analyser. The V_T was determined from expired gas volume divided by f_R. The $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, V_T, f_R, and R were calculated and printed out every 30 s and values for the last 30 s of each stage were used for further analysis. In earlier experiments it had been determined that $\dot{V}O_2$ and $\dot{V}_E$ obtained with the automatic analyser yielded the same values as those which were obtained by the Douglas bag method. The face mask used during the experiment for collecting expired gas was made in our own laboratory. It was designed to allow unhindered movement of the arms during swimming and to ensure that the inspired and expired airways remained unobstructed at all times. The subject received no specific instructions regarding the synchronising of breathing to arm stroke, therefore they breathed freely during swimming. The f_c was continuously monitored using a telemetered electrocardiogram system, and immediately before the end of each stage, f_c was recorded. The $[la^-]_b$ was analysed with an automatic analyser (Yellow Springs Instrument 23L, Yellow Springs, USA) from blood samples taken from the fintertip immediately and 5 min after the subjects were exhausted. The higher value was accepted as maximal.

Statistical analysis. Conventional statistical methods have been used for data analysis. Changes of cardio-respiratory responses between N and H at submaximal water flow rates were tested by two-way ANOVA with repeated measurements. Although some swimmers were able to swim at flow rates faster than 1.00 $m \cdot s^{-1}$, some were not; therefore only data from 0.80 $m \cdot s^{-1}$ to 1.00 $m \cdot s^{-1}$ was included in these analyses. Student's paired t-test was used to evaluate the differences between maximal values for N and H. Results were regarded as statistically significant at $P<0.05$.

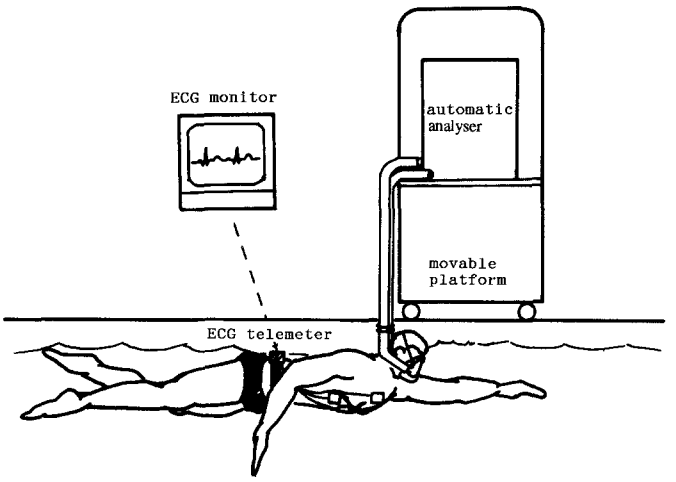

Fig. 1. Experimental setup. Respiratory parameters were measured by an automatic analyser which was put on a movable platform above the swimmer across a swimming flume. The electrocardiogram (*ECG*) to determine heart rate was continuously monitored using a telemetry system

Results

Responses during submaximal swimming

Figure 2 demonstrates $\dot{V}O_2$ during front crawl in N and H at various water flow rates. The $\dot{V}O_2$ increased linearly with increasing water flow rate, and $\dot{V}O_2$ in N tended to be higher than that in H at a given flow rate. However, since individual differences at the same water flow rate in N were great, there were no significant differences. The $\dot{V}CO_2$ and $\dot{V}_E$ in H were significantly higher than those in N ($P<0.05$; Fig. 3, 4). The same result was found for V_T ($P<0.05$; Fig. 5); however, there were no significant differences in f_R (Fig. 6). The f_c in H was not significantly different from that in N (Fig. 7).

Responses during maximal swimming

Peak $\dot{V}O_2$ in test 1 and test 2 was not significantly different, which would indicate that $\dot{V}O_2$ was attained in both tests. Mean $\dot{V}O_{2max}$ were 4.15 (SD 0.18) $l \cdot min^{-1}$ in N, and 3.65 (SD 0.11) $l \cdot min^{-1}$ in H. The $\dot{V}O_{2max}$ in H was significantly lower by 12.0% (SD 3.4)% than that in N ($P<0.01$; Table 1). The $\dot{V}CO_2$ and R in H were significantly higher than those in N ($\dot{V}CO_2$, $P<0.05$; R, $P<0.01$); however, f_c, $\dot{V}_E$, f_R, V_T and $[la^-]_b$ were not significantly different between the envi-

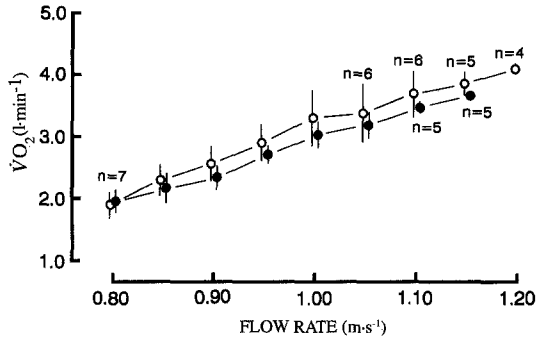

Fig. 2. Oxygen uptake ($\dot{V}O_2$) at various water flow rates in normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa). Values are expressed as mean and SD. There was no significant difference between normobaric normoxia and hypobaric hypoxia. O—O Normobaric hormoxia; ●—● hypobaric hypoxia

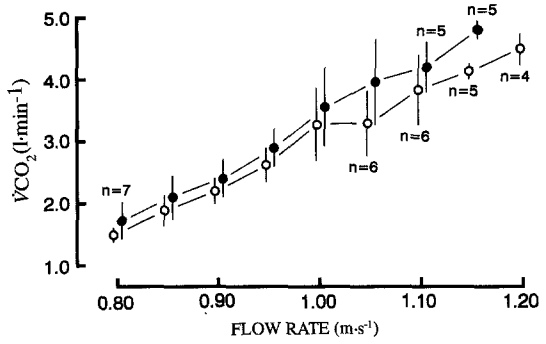

Fig. 3. Carbon dioxide production ($\dot{V}CO_2$) at various water flow rates in normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa). Values are expressed as mean and SD. The $\dot{V}CO_2$ in hypobaric hypoxia was significantly higher than that in normobaric normoxia, which was tested by two-way analysis of variance with repeated measurements. For explanation of symbols see Fig. 2

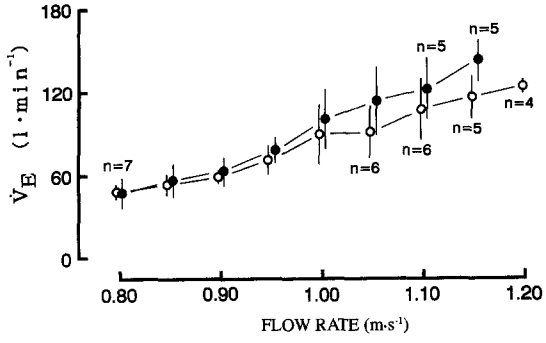

Fig. 4. Pulmonary ventilation ($\dot{V}_E$) at various water flow rates in normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa). Values are expressed as mean and SD. The $\dot{V}_E$ in hypobaric hypoxia was significantly higher than that in normobaric normoxia, which was tested by two-way analysis of variance with repeated measurements. For explanation of symbols see Fig. 2

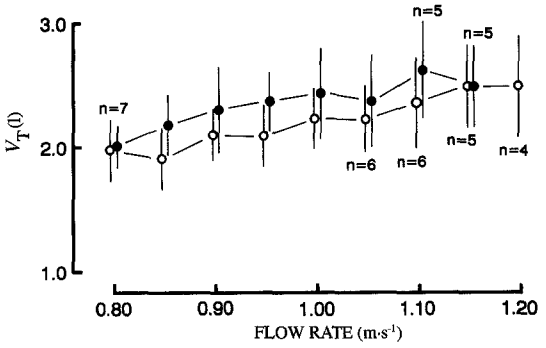

Fig. 5. Tidal volume (V_T) at various water flow rates in normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa). Values are expressed as mean and SD. The V_T in hypobaric hypoxia was significantly higher than that in normobaric normoxia, which was tested by two-way analysis of variance with repeated measurements. For explanation of symbols see Fig. 2

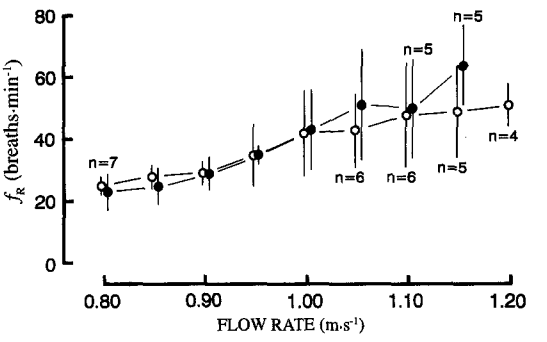

Fig. 6. Respiratory frequency (f_R) at various water flow rates in normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa). Values are expressed as mean and SD. There was no significant difference between normobaric normoxia and hypobaric hypoxia. For explanation of symbols see Fig. 2

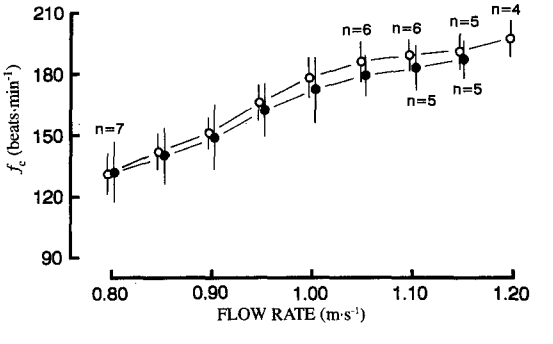

Fig. 7. Heart rate (f_c) at various water flow rates in normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa). Values are expressed as mean and SD. There was no significant difference between normobaric normoxia and hypobaric hypoxia. For explanation of symbols see Fig. 2

Table 1. Maximal values of oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), pulmonary ventilation ($\dot{V}_E$), respiratory frequency (f_R), tidal volume (V_T), heart rate (f_c), blood lactate concentration ($[la^-]_b$), and water flow rate (v_{water}) under normobaric normoxia (751 mmHg, 100.1 kPa) and hypobaric hypoxia (601 mmHg, 80.27 kPa)

		Normobaric normoxia		Hypobaric hypoxia	
		mean	SD	mean	SD
$\dot{V}O_2$	($l \cdot min^{-1}$)	4.15	0.18	3.65	0.11**
$\dot{V}CO_2$	($l \cdot min^{-1}$)	4.47	0.47	4.85	0.18*
$\dot{V}_E$	($l \cdot min^{-1}$)	134.2	13.5	141.8	13.4
R		1.08	0.09	1.32	0.06**
V_T	(l)	2.34	0.32	2.36	0.29
f_R	(breaths $\cdot min^{-1}$)	59	13	61	10
f_c	(beats $\cdot min^{-1}$)	190	9	184	6
$[la^-]_b$	($mmol \cdot l^{-1}$)	10.7	0.8	9.0	1.2
v_{water}	($m \cdot s^{-1}$)	1.19	0.07	1.14	0.03

Significant differences between normobaric normoxia and hypobaric hypoxia, which were tested by Student's paired t-test, are indicated by the following: ** $P<0.01$, * $P<0.05$

ronments. Water flow rates at which $\dot{V}O_{2max}$ were obtained in N tended to be higher than those in H; however, no significant difference was found since one subject could swim at the same water flow rate in both environments.

Discussion

The present study determined $\dot{V}O_2$ at different water flow rates and $\dot{V}O_{2max}$ during swimming in a hypobaric hypoxic environment. Test 2 was carried out in addition to test 1 to ensure that $\dot{V}O_2$ had levelled off. The method used here for measuring $\dot{V}O_{2max}$ was first described by Ekblom (1969). Åstrand and Saltin (1961) have also reported that $\dot{V}O_{2max}$ can be obtained during exercise in which the subjects reach exhaustion within 2 to 3 min provided the subjects perform an extensive warm up. Therefore, the peak $\dot{V}O_2$ obtained in this study was considered to be the maximal value for each subject during swimming.

In general, $\dot{V}O_{2max}$ observed during exercise using large muscle groups, such as cycling and running, has been shown to be limited by factors of the central circulation such as cardiac pumping capacity and inadequate venous return (Horstman et al. 1976; Reybrouk et al. 1975; Andersen and Saltin 1985; Saltin 1985; Shephard et al. 1988). Consequently, $\dot{V}O_{2max}$ in hypoxia shows a decrease proportional to the decrease in arterial O_2 content. In the present study, $\dot{V}O_{2max}$ during swimming in H, which corresponded to 2000 m above sea level, was significantly lower by 12% than that in N. Furthermore, this decrease of $\dot{V}O_{2max}$ was in good agreement with the values of previous studies of whole body exercise on land, which were limited by factors of the central circulation, with similar levels of hypoxia (Fagraeus et al. 1973; Squires and Buskirk 1982; Wagner et al. 1986; Roca et al. 1989). Furthermore, we would like to suggest that $\dot{V}O_{2max}$ during exercise using large muscle masses, such as running and swimming is mainly limited by the maximal work rate of the heart, which might be an index of the central circulation. Theoretically, work is calculated from the product of P and ΔV, where P is pressure and V is volume. Therefore, the work rate of the heart can be expressed as a product of blood pressure, stroke volume and f_c. Since $\dot{Q}_c$ is a product of the last two factors, the work rate of the heart should consequently be expressed as a product of blood pressure and $\dot{Q}_c$. It has been reported by many investigators that the cardio-respiratory response to exercise varies with variations in body position (Bevegard et al. 1966; Stenberg et al. 1967). For example, studies of swimming in a prone position and running upright have shown maximal f_c to be generally lower during swimming (Holmér et al. 1974a, b; Holmér 1972); however, differences in stroke volume have not been found (Holmér et al. 1974a). A similar result was observed during a combined leg and arm exercise, as in swimming, in both supine and upright positions (Stenberg et al. 1967). In addition, no marked differences in arterial blood pressure between supine and upright exercise were reported. However, Holmér et al. (1974a) has shown arterial blood pressure during prone swimming to be higher than that during running. This difference might be attributed to the effect of hydrostatic pressure and the vasoconstriction of blood vessels in the skin. Consequently, it has been suggested that the maximal work rate of the heart during swimming may be similar to that during running. On the other hand, the arteriovenous O_2 difference, an index of peripheral factors, in exercise in a supine position, has been reported to be lower than in upright combined exercise on land (Stenberg et al. 1967). This similar response in prone swimming and running is not easy to understand since one study has shown the arteriovenous difference to be lower during swimming (Holmér et al. 1974a) while another has shown no difference (Dixon and Faulkner 1971). Arteriovenous O_2 differences for supine and prone position exercise might be affected by changes in blood flow due to the position of the active limbs in relation to the heart. Overall, it seems likely that $\dot{V}O_{2max}$ during swimming may be mainly limited by factors of the central circulation such as cardiac pumping capacity, which is equivalent to maximal work rate of the heart, and that the decreased $\dot{V}O_{2max}$ in H might be attributed to a decrease in arterial O_2 content. However, since no measurements of parameters such as stroke volume, blood pressure, and arteriovenous O_2 difference were made in this experiment, it is difficult to understand clearly the limiting factors in $\dot{V}O_{2max}$ during swimming. Further experiments measuring those parameters simultaneously are needed.

In this experiment, $\dot{V}CO_2$ and $\dot{V}_E$ in H at submaximal water flow rates were found to be significantly higher than in N and this was attributed to a significantly higher V_T in H. The $\dot{V}_E$ during swimming was done against the forces of hydrostatic pressure. Therefore, respiratory work in H may be higher than that in N. However, $\dot{V}O_2$ in H tended to be lower than that in N at

a given water flow rate but no significant difference was found. This might be explained in part by significantly greater V_T in H. Holmér (1974b) has shown that functional residual capacity during swimming was almost constant regardless of increased V_T. If functional residual capacity in H was not different from that in N, greater V_T in H would increase buoyancy and hence would reduce body resistance. Mechanical work during swimming is expressed by a production of water flow rate and body water resistance. To calculate energy demand during swimming, work should be divided by mechanical efficiency. If mechanical efficiency is assumed to have been similar in N and H, the lack of a significant difference in $\dot{V}O_2$ may mean that a greater V_T had induced a decrease in mechanical work which would have compensated for the increase in respiratory work.

In summary, we measured $\dot{V}O_2$ at different water flow rates and $\dot{V}O_{2max}$ in H during swimming. At submaximal water flow rates, $\dot{V}O_2$ in H was not significantly different from that in N. The $\dot{V}O_{2max}$ in H decreased by 12% from that in N. The decrease in $\dot{V}O_{2max}$ in H during swimming agrees well with the values from previous studies on whole body exercise limited by the central circulation at similar altitudes or levels of H. This decrease of $\dot{V}O_{2max}$ in H might have been a result of the reduction in arterial O_2 content induced by a decrease in atmospheric pressure.

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