Increases in submaximal cycling efficiency mediated by altitude acclimatization

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Among the many changes proposed to occur with altitude acclimatization is a change in net mechanical efficiency (16). According to this hypothesis, at a given level of submaximal exercise, the net energy cost, as measured by \( \text{O}_2 \) consumption (V\( \text{O}_2 \)), is reduced, and, consequently, mechanical efficiency is enhanced (16). Although this hypothesis is appealing, because the adaptation could potentially allow more work to be performed with the limited \( \text{O}_2 \) available, the evidence is not impressive.

One experimental model that has been commonly utilized to investigate the effects of altitude acclimatization involves initial sea level testing of lowlanders, followed by additional testing soon after arrival at altitude or during acute exposure to simulated altitude, and after a period of residence either at altitude or in hypobaric hypoxic conditions (17, 39). In general, studies examining the response of lowlanders to both acute and chronic hypoxia have reported either no effect (2, 39) or an increase (31) in whole body steady-state V\( \text{O}_2 \) during constant-load exercise. When an increase in whole body V\( \text{O}_2 \) has been found, increases in resting V\( \text{O}_2 \) appear responsible (31).

One factor that could mitigate against reductions in whole body V\( \text{O}_2 \) is the additional energy costs associated with ventilation (4). The increase in minute ventilation (V\( \text{E} \)) that occurs with acute hypoxia (4), and which may be potentiated with sustained exposure (4), could mask changes in efficiency in the working leg muscles. This possibility has been examined by measuring V\( \text{O}_2 \) across the working leg muscles in conjunction with measures of whole body V\( \text{O}_2 \). Using this approach, Wolfel et al. (39) have found evidence of a lower leg V\( \text{O}_2 \) with unchanged total body V\( \text{O}_2 \) as a result of a lower leg blood flow and a lower arteriovenous \( \text{O}_2 \) difference during both acute and chronic hypoxic conditions. These results have not been confirmed by others (2, 31).

Another approach that has been used to examine the effects of acclimatization is to measure the exercise response under normoxic conditions. Under this strategy, the energy costs associated with ventilation are considerably reduced and the more persistent effects of chronic hypoxia on increased red cell volume and arterial \( \text{O}_2 \)-carrying capacity can be examined (35). Surprisingly, this model has rarely been employed. Of the few studies available, exercise in normoxia after acclimatization, although reducing the ventilatory response (2), has not resulted in either lower total body V\( \text{O}_2 \) or V\( \text{O}_2 \) in the working legs (2, 3).

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This issue of mechanical efficiency has also been examined in high-altitude residents during a brief sojourner to near-sea level conditions (16). On the basis of comparisons with sea level residents, both trained and untrained, it was found that the \( \dot{V}O_2 \) measured during progressive exercise at sea level was lower in the highlanders (16). The difference in \( \dot{V}O_2 \) costs could be explained by a lower resting metabolic rate.

In this study, we report on the effects of a 21-day expedition to an altitude of 6,194 m (Mount Denali). By having the mountaineers report to the laboratory a few days before and within 3 days after the expedition, we were able to avoid many of the problems associated with deacclimatization that have characterized other studies (27). Under such conditions, we hypothesized that steady-state submaximal exercise performed at the same absolute power both before and after the expedition will result in lower \( \dot{V}O_2 \) costs that cannot be explained by changes in resting \( \dot{V}O_2 \). As a consequence, net efficiency is increased with acclimatization.

METHODS

Subjects

The data are from five men who participated in an expedition to Mount Denali. On average, the age (mean \( \pm \) SE) was 28 \( \pm \) 2 yr, weight was 76.9 \( \pm \) 4.3 kg, and height was 173.6 \( \pm \) 3.6 cm. An additional member of the expedition, a woman, was not included because we did not have a complete data set. All male climbers were regularly active, including during the months immediately preceding the expedition, and all had a history of mountain climbing. The testing protocols were approved by the Office of Human Research and Animal Care (University of Waterloo, Waterloo, Ontario, Canada), and written consent was obtained from each participant as required. The expedition itself was self-initiated without involvement of the University of Waterloo or any of the scientists involved.

Experimental Design

To examine the effects of the expedition to Mount Denali, testing was conducted at the University of Waterloo (altitude = 317 m). Approximately 1 wk before the beginning of the climb, the mountaineers were transported to the laboratory, where a series of tests (Pre) was conducted over a 3-day period. On day 1, the participants performed a progressive cycle test to fatigue for measurement of peak \( \dot{V}O_2 \) (\( \dot{V}O_2 \) peak) and related parameters. Also on day 1, a short submaximal test was performed for measurement of basal \( \dot{V}O_2 \) and blood flow. This test preceded the progressive cycle test. Approximately 2–3 h were allowed between tests. On day 2, a two-step prolonged cycle test was performed to measure gas exchange and selected blood properties. The results of the progressive and prolonged tests are reported in this paper. Three days after the expedition (Post), the climbers were transported back to the laboratory to perform the same series of tests in the same order.

The expedition to Mount Denali extended over a 21-day period. The mountaineers flew to Talkeetna, Alaska (300 m), and established a base camp (2,160 m) on day 1. Ascent to the summit (6,194 m) was achieved on day 18. By day 8, the climbers achieved an altitude of \( \sim \)50% of the summit, and, by day 14, 75% of the objective was realized. By day 20, the volunteers had arrived at the initial base camp and returned to Talkeetna. Three days later, the group arrived at the University of Waterloo for posttesting.

Testing Protocols

Progressive exercise. Approximately 60 min before the progressive test, the participants reported to the laboratory. During this period, weights and heights were recorded, and preparations were made for blood sampling and the monitoring of respiratory gas exchange and heart rate. For blood sampling, a small Teflon catheter with a three-way stopcock was placed in a dorsal vein of a prewarmed hand and kept warm throughout the exercise with a heating pad. Heart rate was determined by standard electrocardiograph techniques. Gas exchange [\( \dot{V}O_2 \), CO\(_2\) production (\( \dot{V}CO_2 \)), and \( \dot{V}E \)] was measured by use of an open-circuit system as previously described (18). An electrically braked cycle ergometer (Quinton 870) was calibrated on each testing day using standardized weights, was used for all exercise tests. In addition, the gas analyzers (Beckman OM-11 and LB-2) and the pneumotachograph (Hewlett Packard 4730 A) were calibrated on each testing day (both before and after the expedition). Common reference gases, with the gas percentages precisely determined, were used for the calibration. The pneumotachograph was calibrated by using a 3-liter syringe, emptied to produce a flow rate similar to that found in exercise.

The progressive test protocol consisted of 4 min of cycling at 25 W followed by a step increase in power output of 25 W every second minute until the subject could continue no longer. Sampling of the ventilatory volumes and respiratory gases was continuous, with the average volume and gas concentrations obtained during the last 20 s of each stage used for analysis. Heart rates and blood samples were also obtained during the same period.

Prolonged exercise. The prolonged cycle exercise protocol consisted of 40 min of exercise performed for 20 min at each of two power outputs, designed to elicit \( \sim \)60% and \( \sim \)75% of prealtitude \( \dot{V}O_2 \) peak. As with the progressive test, subjects were instrumented for respiratory, heart rate, and blood sampling. These measurements were performed at rest before the exercise and, in the case of the heart rate and respiratory gas exchange measurements, during the final 4–5 min segment of each power output. Resting measurements for all variables were obtained after the subjects had been sitting quietly on the cycle for \( \sim \)15-min period. Blood samples were collected over a 30-s period before 3, 20, and 40 min of exercise. All procedures, including calibration and data collection, were essentially as reported for the progressive test. For the submaximal tests, all subjects consumed a liquid supplement consisting of one can of Ensure (1.045 kJ: 14.8% protein, 31.5% fat, and 53.7% carbohydrate; Ross Laboratories, Montreal, PQ, Canada) \( \sim \)4 h before the beginning of exercise. No other supplements, including coffee, were permitted on the day of the test.

Both tests, progressive and prolonged, were performed under normal and relatively standard environmental conditions (24–26°C dry bulb temperature and 50–60% relative humidity). Water was not permitted during either test.

Net efficiency, defined as the work rate divided by the energy expended above that at rest, was calculated during steady state for the submaximal cycle test. For this calculation, work rate is expressed in watts and the \( \dot{V}O_2 \), used as a measure of energy expended, was converted to joules per milliliter (20.93 J/ml), assuming a respiratory exchange ratio (RER) of 1.0. It was not possible to calculate an energetic equivalent based on the actual ratio because this value was >1, particularly during the Post tests.
Table 1. Effects of acclimatization on resting blood hematocrit, hemoglobin, total protein, and arterial oxygen saturation

<table>
<thead>
<tr>
<th></th>
<th>Hct, %</th>
<th>Hb, g/100 ml</th>
<th>Sat, %</th>
<th>ΔPV, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre</td>
<td>5.98 ± 0.16</td>
<td>43.7 ± 0.6</td>
<td>15.0 ± 0.49</td>
<td>96 ± 0.6</td>
</tr>
<tr>
<td>Post</td>
<td>5.61 ± 0.14</td>
<td>47.4 ± 0.6</td>
<td>15.8 ± 0.41</td>
<td>96 ± 0.6</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 5. Pro, total protein; Hct, hematocrit; Hb, hemoglobin; Sat, arterial oxygen saturation. Pre, preacclimatization; Post, postacclimatization; ΔPV, change in plasma volume using Hct and Hb values. *Significantly different from Pre (P < 0.05).

Analytical techniques. Whole blood was extracted and used for the measurement of lactate, hematocrit (Hct), and Hb. For lactate, the blood was deproteinized by using ice-cold perchloric acid, centrifuged to remove the pelleted proteins, and neutralized with ice-cold KHCO₃. Samples were stored at −80°C until analyzed by fluorometric techniques (21). Hct was measured in triplicate using standard techniques (38) and corrected for trapped plasma (0.96) and venous-to-whole body Hct difference (0.91). Hb was also measured in triplicate using standard cyanmethemoglobin methods. Changes in plasma volume (PV) before and after the expedition were determined using both Hct and Hb (38) obtained during rest before the prolonged exercise test. Changes in PV during the exercise were calculated using only the Hct values (37).

Where possible, blood measurements (samples obtained before and after the expedition) were performed after the expedition on blood that had been stored at −80°C. During a given analytical session, all samples for a given subject and for a given property were performed in duplicate during the same analytical session.

Measurements of arterial O₂ saturation (SaO₂) were made from the fingertip using oximetry (Ohmeda model 3700). Fingertips were carefully cleaned with alcohol before the probe was attached. The validity of the oximeter was previously established (29). Moreover, we have also found a close relationship between SaO₂ obtained from arterial blood obtained from the radial artery and the oximeter during exercise (unpublished observations).

Statistical Procedures

The effect of the altitude expedition was determined by both one-way and two-way ANOVA procedures for repeated measures. One-way ANOVA was employed with a single measurement (i.e., V˙O₂peak, peak V̇E, and so forth) obtained before and after the expedition. Two-way ANOVA procedures were used when several measurements were made during exercise (i.e., V˙O₂, V˙E, lactate, and so forth), both before and after acclimatization. When significance was found, Newman-Keuls procedures were applied to locate differences between specific means. Significance was set at the 0.05 level.

Table 2. Effects of acclimatization on peak aerobic power and related measures

<table>
<thead>
<tr>
<th>V˙O₂peak</th>
<th>V˙CO₂peak</th>
<th>HRpeak</th>
<th>V̇Epeak</th>
<th>RERpeak</th>
<th>PO, W</th>
</tr>
</thead>
<tbody>
<tr>
<td>l/min</td>
<td>ml·kg⁻¹·min⁻¹</td>
<td>beats/min</td>
<td>l/min</td>
<td>BTS</td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>3.99 ± 0.17</td>
<td>52.3 ± 2.1</td>
<td>4.98 ± 0.17</td>
<td>189 ± 7</td>
<td>156 ± 15</td>
</tr>
<tr>
<td>Post</td>
<td>3.81 ± 0.18*</td>
<td>50.6 ± 2.2</td>
<td>4.72 ± 0.23</td>
<td>185 ± 6</td>
<td>155 ± 16</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 5. V˙O₂peak, peak O₂ consumption; V˙CO₂peak, peak CO₂ production; HRpeak, peak heart rate; V̇Epeak, peak ventilation; RERpeak, peak respiratory exchange ratio; PO, power output. *Significantly different from Pre (P < 0.05).

RESULTS

Resting Blood Measurements

After the expedition to Mount Denali, both resting Hct and Hb concentration were elevated (Table 1). PV, calculated using both Hct and Hb changes, decreased by ~10% after acclimatization. Acclimatization also resulted in an ~6.2% decrease in total protein. At rest, arterial Hb saturation with O₂ was estimated at 96% both before and after the expedition.

Progressive Exercise

After the expedition, a small but significant decline was observed in V˙O₂peak obtained during progressive exercise to fatigue (Table 2). The decline was observed only when V˙O₂peak was expressed in absolute (l/min), and not relative (ml·kg⁻¹·min⁻¹) terms. Body mass, measured before (76.9 ± 3.7 kg) and after the expedition (75.5 ± 2.9 kg), was unchanged. Acclimatization did not result in a lower peak V˙CO₂. The peak values for the other variables examined, namely heart rate, V̇E, RER, and power output, were all unchanged with acclimatization. Also unchanged with acclimatization were the peak concentrations of blood lactate (7.21 ± 0.35 vs. 5.65 ± 0.70 mM) and the blood catecholamines epinephrine (246 ± 53 vs. 216 ± 56 pg/ml) and norepinephrine (1,796 ± 210 vs. 1,674 ± 266 pg/ml).

V˙O₂, measured during the final 20 s of each step increase in power output, was lower after the expedition (Fig. 1A). The lower value was not specific to any time point but rather represented a general effect. Unlike V˙O₂, V˙CO₂ was not changed before and after acclimatization (Fig. 1B). In contrast to V˙O₂, V̇E was higher during exercise after acclimatization (Fig. 1C). For all variables, a general progressive increase was shown with each increment in power output. Blood lactate concentration was also found to be altered with acclimatization (Fig. 2A). At rest and during initial 25-W load pedaling, lactate concentration was higher after acclimatization than before. With progressive exercise, a lower percent SaO₂ was observed (Fig. 2B). In general, SaO₂ remained higher during exercise after the altitude expedition than before; however, the difference was not significant. No effect of the expedition was observed on heart rate, regardless of the exercise intensity (Fig. 2C).

Changes in the catecholamines, norepinephrine and epinephrine, were found for both exercise and acclima-
tization. With exercise, both norepinephrine and epinephrine increased with increases in power output (Fig. 3, A and B). However, these hormones reacted differently to acclimatization. For norepinephrine, a generally higher value was found that was not specific to any time point. In contrast, the concentration of epinephrine was unchanged after acclimatization.

Submaximal Exercise

As expected, the two-stage prolonged exercise test resulted in increases in \( V_{\text{O}_2} \), \( V_{\text{CO}_2} \), and \( V_{\text{E}} \) (Table 3). After acclimatization, \( V_{\text{O}_2} \) was depressed by 9.7% at 20 min and by 7.7% at 40 min of exercise. A similar effect...

![Fig. 1](image1.png)

**Fig. 1.** Effects of progressive exercise and acclimatization on \( O_2 \) consumption (\( V_{\text{O}_2}; A \)), \( CO_2 \) production (\( V_{\text{CO}_2}; B \)), and minute ventilation (\( V_{\text{E}}; C \)). Values are means ± SE; \( n = 5 \). Pre, preacclimatization; Post, postacclimatization. For both \( V_{\text{O}_2} \) and \( V_{\text{E}} \), a main effect (\( P < 0.05 \)) of condition was found. For \( V_{\text{O}_2} \), Pre > Post. For \( V_{\text{E}} \), Post > Pre. Main effects for exercise (\( P < 0.05 \)) were shown for all variables. With the exception of the initial power outputs, progressive increases were observed for all variables.

![Fig. 2](image2.png)

**Fig. 2.** Effects of progressive exercise and acclimatization on blood lactate (\( A \)), saturation (Sat; \( B \)), and heart rate (HR; \( C \)). Values are means ± SE; \( n = 5 \). An effect of acclimatization was only found for blood lactate. *Pre > Post (\( P < 0.05 \)). †Post > Pre (\( P < 0.05 \)). Main effects (\( P < 0.05 \)) of exercise were found for all variables.
was not found for $V_{CO_2}$. Both $V_E$ and RER were higher at postacclimatization compared with preacclimatization. In the case of RER, higher values were observed at rest and at 20 and 40 min of exercise. For $V_E$, the higher values observed were not specific to any time point. Exercise heart rates were reduced at both 20 and 40 min with acclimatization (Fig. 4A). In contrast, resting heart rates were higher after the expedition than before. Blood lactate concentration, although increased with exercise, was not affected by the expedition (Fig. 4B).

Net mechanical efficiency, calculated during steady state for both exercise intensities, was 25.9 ± 1.6% and 24.4 ± 1.3% during the first and second step, respectively, before the expedition. After the expedition, the respective values were 31.3 ± 1.3% and 29.5 ± 1.5%. Acclimatization resulted in an increase in net efficiency.

Of the plasma catecholamines norepinephrine and epinephrine, only norepinephrine was blunted after acclimatization (Fig. 5, A and B). The lower norepinephrine was observed only during exercise and not at rest. As with norepinephrine, epinephrine increased with exercise. Although lower epinephrine values were observed after the expedition, the differences were not significant.

Declines in arterial oxyhemoglobin saturation were initially observed at 20 min of exercise and persisted at 40 min of exercise (Fig. 6A). The decline in saturation was not affected by the state of acclimatization.

As expected, reductions in PV occurred with exercise (Fig. 6B). Before acclimatization, the decline was progressive, averaging 4.0% at 20 min and 6% at 40 min. The magnitude of the PV reduction was not altered with acclimatization.

**DISCUSSION**

As hypothesized, we found a lower $V_{O_2}$, measured during identical progressive and prolonged work protocols after the expedition compared with before the expedition. Because resting $V_{O_2}$ was unchanged (before submaximal exercise), increases in net efficiency are indicated with acclimatization. For the submaximal test, net efficiency was found to increase ~21% regardless of power output. This study appears to be the first report of changes in mechanical efficiency in lowlanders after an altitude expedition or after acclimatization when the exercise is performed under sea level conditions. A higher mechanical efficiency has been suggested in highland residents during submaximal exercise at sea level compared with conditioned lowland residents (16); however, the differences do not appear to be due to differences in $V_{O_2}$-work rate relationship (9).

It should be emphasized that there is little chance that measurement error could explain the lower $V_{O_2}$...
that we observed after acclimatization. In an accompa-
nying paper that addresses muscle blood flow and \( V\dot{O}_2 \) kinetics (22), a similar result was found. This observa-
tion was made while using a different gas-exchange
system that was independently calibrated, a different
ergometer, and a different test protocol.

Because we measured whole body \( V\dot{O}_2 \), it was not
possible to determine whether the lower \( V\dot{O}_2 \) was
mediated by changes in the locomotor muscles used in
cycling or in the nonlocomotor components such as the
muscles of ventilation. However, in this study, the
lower \( V\dot{O}_2 \) did not appear to be due to a decreased cost
of ventilation because \( V\dot{E} \) was generally higher, during
both progressive and prolonged exercise, after acclima-
tization. It would appear probable, given the magni-
tude of the difference between \( V\dot{O}_2 \) measured before
and after acclimatization and the fact that the muscles
involved in cycling account for most of the total body
\( V\dot{O}_2 \) (11, 28), that the decrease in \( V\dot{O}_2 \) is explained, in
large part, by a reduced \( V\dot{O}_2 \) utilization in the working
locomotor muscles. Teleologically, the lower \( V\dot{O}_2 \) could
be explained by a shift toward a greater glycolytic
involvement in ATP regeneration, a shift toward a
greater carbohydrate preference over fats in oxidative
phosphorylation, and/or an increased ability of the
excitation and contraction processes to perform work
at lower energy costs.

An acclimatization-dependent increase in anaerobic
glycolysis during submaximal exercise does not appear
probable given the studies that provide evidence that
just the opposite occurs, namely, a reduction in anaer-
obic-based metabolism. During exercise after acclima-
tization, reductions in lactate production (5), lactate
exchange across the working limbs (3), and lower blood
(10) and muscle (13) lactates have been reported.

In general, adaptations to chronic hypoxia in hy-
poxia-intolerant mammals appear to favor increased
exploitation of available \( O_2 \) (14, 15). The second possi-
bility, namely, increasing carbohydrate dependency
during acclimatization, could lower \( V\dot{O}_2 \) costs because
complete oxidation of glycogen yields more ATP per

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**Fig. 5. Effects of submaximal exercise and acclimatization on blood Norepi (A) and Epi (B) concentrations. Values are means ± SE; \( n = 5 \). *Significantly different (\( P < 0.05 \)) from Pre. For both Epi and Norepi, main effects (\( P < 0.05 \)) of exercise were found; 40 > 20 > 0 min.**
mole of O₂ than the complete oxidation of fats (16). Studies using stable isotopes have reported increases in glucose oxidation during exercise while subjects were resident at altitude (6, 32), in conjunction with decreases in fat oxidation (31). However, it is not clear what effect the increased glucose utilization has on $\dot{V}O_2$ during exercise. Studies reporting increases in glucose utilization have not found decreases in $\dot{V}O_2$ with acclimatization (6, 32). Moreover, because a reduction in the muscle glycogen depletion rate also appears to be part of the acclimatization process (13, 40), it is possible that some of the glucose is being diverted to glycogenogenesis. Alternatively, it is possible that the glycogen-sparing effect with acclimatization is due to reductions in anaerobic glycolysis (5, 13).

Regardless of the effect of the acclimatization-induced shift toward increased glucose utilization, a shift in substrate cannot explain the effect of the expedition on the increases in net efficiency reported in this paper. It should be emphasized that, although our calculations of the energetic equivalent were based on a standardized ratio of 1.0, the actual ratio obtained (i.e., 0.95 Pre vs. 1.0 Post) would not alter our conclusions regarding the increase in net efficiency after the altitude expedition.

It would appear that the increase in net efficiency occurs as a consequence of the reduced energy needs of one or more of the processes involved in excitation and contraction. The major energy-consuming processes in the contracting muscle are associated with $Na^+\!\!/K^+$ exchange across the sarcolemma and T tubule, $Ca^{2+}$ pumping into the sarcoplasmic reticulum (SR), and actomyosin cycling (19). The ATP requirements of these processes are supplied by three ATPases, namely the $Na^+\!\!/K^+$-ATPase, the SR $Ca^{2+}$-ATPase, and the myosin ATPase. It is possible that, given the requirements of the exercise tasks, decreased cation or actomyosin cycling could occur without compromise to excitation and contraction events. Alternatively, increased efficiency could result in increases in the coupling between the number of cations pumped per ATP utilized, as in the case of $Ca^{2+}$ cycling (23). Increased efficiency may also result from the metabolic adaptations that occur with acclimatization. The decrease in metabolic by-product accumulation, such as ADP, $P_i$, and $H^+$, that occurs after acclimatization would be expected to increase the amount of free energy released from ATP hydrolysis (26) and, consequently, depress the need to maintain hydrolysis rates at preacclimatization levels. Although the significance remains to be established, it is of interest that the altitude expedition induced a downregulation in muscle $Na^+\!\!/K^+$-ATPase content (12) and supposedly in maximal $Na^+\!\!/K^+$-ATPase activity (7). On the basis of Michaelis-Menton enzyme kinetics, a lower maximal $Na^+\!\!/K^+$-ATPase activity should reduce reaction rates at a given substrate concentration (26). At present, it is unclear how acclimatization affected the SR $Ca^{2+}$-ATPase and the myosin ATPase. It is of interest that Hochachka et al. (16) have reported a higher mechanical efficiency in long-term high-altitude residents (4,200 m) compared with trained lowlanders when measured under sea level conditions, an adaptation that they also attribute to increased efficiency of one or more of the processes involved in excitation and contraction.

It has been shown that the type I (slow twitch), in contrast to the type II (fast twitch), muscle fiber is considerably more efficient in performing isometric tasks or tasks requiring relatively lower velocities (8). Acclimatization-induced transformation of fiber types could conceivably underlie changes in mechanical efficiency. However, in this study, the fiber types and subtypes were unaltered by the expedition (12). Interestingly, the mountaineers contained an abnormally high percentage of type I fibers in the vastus lateralis before the expedition. This observation was made previously in experienced mountaineers (27).

Acclimatization also resulted in a reduction in exercise heart rate during each step of the two-stage exercise protocol, even though resting heart rate was elevated. The reduction in heart rate could be explained,
at least in part, by the lower exercise \( \dot{V}O_2 \) and, consequently, the reduced cardiovascular demand in conjunction with the lower leg blood flow that probably resulted (2). Unlike the effects of acclimatization measured during exercise in hypoxia (3), in our findings, \( Sa_O_2 \) was not elevated during exercise in normoxia after the expedition to Mount Denali. Exercise, both before and after the expedition, did result in a significant reduction in \( Sa_O_2 \). As in a previous study (34), we have shown that, within 1 wk of deacclimatization, Hb concentration remains significantly elevated. Because resting \( Sa_O_2 \) was not affected by altitude residence, arterial \( O_2 \) content would be elevated (2,39). Given that preservation of arterial \( O_2 \) delivery to working muscle appears to be a high priority, the increases in arterial \( O_2 \) content should have implications to cardiac function and muscle blood flow (2,39).

In addition to the lower exercise \( \dot{V}O_2 \), acclimatization also altered several variables measured during the two-step submaximal exercise test employed in this study. As an example, a higher exercise \( V_e \) and RER were observed after acclimatization, both of which can be explained by the higher resting values. Higher \( V_e \) values have also been reported at rest during acclimatization to 4,300 m (4). However, higher resting \( V_e \) values were not observed at sea level after a 10-day deacclimatization period (34). The increase in \( V_e \) that we observed could result from increased chemoreceptor sensitivity (20,30,36), which still persists for a few days after acclimatization but is lost as the deacclimatization period is extended. Interestingly, the higher exercise RER values that we observed after acclimatization are not due to increases in \( VCO_2 \), because \( VCO_2 \) remained unaltered, but to the reduction in \( \dot{V}O_2 \).

The adaptations that were observed during the progressive exercise are generally similar to those reported for the two-stage submaximal exercise test protocol. As an example, \( \dot{V}O_2 \) was lower, \( V_e \) was higher, and \( VCO_2 \) was unchanged with acclimatization, regardless of the test protocol employed. However, in the progressive test, blood lactates were clearly depressed with acclimatization but only at the higher power outputs. At the lower power outputs, blood lactates were higher after acclimatization. The results that we have observed at the higher intensities are consistent with what was reported by Grassi et al. (10) after a 1-wk deacclimatization from an expedition to 5,050 m. A curious difference was observed between the two exercise protocols regarding the effects of acclimatization on the heart rate response. During the prolonged exercise protocol, heart rate was depressed after the expedition. Such effects were not observed during the progressive exercise. These differences might be explained as a result of differences in the blood catecholamine response to exercise and acclimatization. During the progressive exercise, blood norepinephrine was elevated, an adaptation that could be explained by the progressive increase in resting blood norepinephrine that occurs with acclimatization (1,24,25). During the prolonged exercise session, blood norepinephrine was blunted during exercise. Differences in the effects of acclimatization also occurred between tests for blood epinephrine concentration. Whereas acclimatization resulted in a lower blood epinephrine response during progressive exercise, no significant changes were observed during the submaximal exercise protocol. After acute exposure, chronic altitude residence results in a lower blood epinephrine response during prolonged exercise (25). The most probable reason for the differences in the acclimatization response between the two exercise protocols, notwithstanding the different challenges imposed, is the period of deacclimatization. The progressive test was conducted some 2 days earlier than the prolonged submaximal test. Given the role of the sympathoadrenal system in regulating a wide range of physiological responses, including cardiovascular and metabolic, the differences that we observed on the acclimatization effects between the two tests may be related to the period of deacclimatization.

In summary, we found that, when tested under sea level conditions, a chronic acclimatization to altitude results in a range of adaptations, the most conspicuous being an increase in net efficiency. Although it is appealing to credit the effects observed to chronic hypoxia, other influences may be important. Acclimatization, as experienced by a mountaineering expedition, results in exposure to a variety of other stressors, including cold, food and water restriction, and exhaustive work. The independent and interactive effects of each of these variables remain uncertain.

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