

A STUDY OF CARDIOPULMONARY FUNCTION AND WORKING CAPACITY OF SOJOURNERS ACUTELY EXPOSED TO DIFFERENT ALTITUDE LEVELS IN ETHIOPIA

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ABSTRACT: Forced vital capacity (FVC, L/sec), forced expiratory volume (FEV₁, L/sec), FEV₁%, forced expiratory flow rate (FEF₂₀₀₋₁₂₀₀ ml, L/sec), forced mid-expiratory flow rate (FMF₂₅₋₇₅ %, L/sec), peak expiratory flow rate (PEFR, L/min), minute ventilation (VE, L/min), oxygen saturation of arterial blood (SaO₂%), working capacity (Kg_m/min/m² body surface area), pulse rate (beats/min) and arterial blood pressure (mmHg) were studied in eight male sojourners descending to 1370-490 m and ascending to 2550-3300 m from their altitude of residence (2440 m). Of the lung function indices, mean FEF₂₀₀₋₁₂₀₀ ml increased from 6.54 L/sec at 2440 m to 7.18 L/sec and 8.33 L/sec, respectively, at 2550 m and 3100 m. It was not measured at 3300 m for lack of electric power supply. Mean values of FEF₂₀₀₋₁₂₀₀ ml were found to be correlated to the altitudes of investigation significantly ($r=0.7316$). The increase in FEF₂₀₀₋₁₂₀₀ ml probably reflects that large airways tend to open up more widely perhaps concomitant with enlargement of alveolar spaces at 2550 m and above to compensate for the reduced PAO₂ and reduced O₂ uptake. Mean VE showed a 4-5-fold increase with exercise essentially at all levels of altitude. Acute altitudinal effect became quite evident at 3300 m. At rest, VE increased from 5.62 L/min at 2440 m to 7.78 L/min at 3300 m. During exercise, increase in VE with ascent apparently protects the low O₂ saturation (91.9% at rest and 86.9% in this study). Although the mean of exercise VE was markedly increased only at 3300 m, its overall correlation with altitude ($r=0.6698$) and mean working capacity ($r=-0.6426$) was quite significant (two-tailed $P=0.0236$). SaO₂% was found to be correlated with mean ($r=0.7955$) and individual measurements ($r=0.7628$) of working capacity. Mean working capacity was reduced by about 20% with ascent and increased by about 9% with descent. It was negatively correlated with ascent ($r=-0.9101$). This signifies that physical performance is quite limited by hypoxia with ascent. Mean pulse rate, which was 75 beats/min at 2440 m increased to 82 beats/min at 490 m. It decreased to 72 beats/min at 3300 m. This is probably attributable to the decrease in ambient temperature and relative humidity with ascent and to the increase with descent. Arterial blood pressure (mean systolic/diastolic) was found to be 100.1 / 62.13 mmHg at 2440 m, 93.1/59.4 mm Hg at 490 m, 99.6/59.4 at 1370 m and 101.46-102.5/62.5-63.1 mmHg at 2550-3300 m. This pattern of change seems to be attributable to changes in peripheral vascular resistance which is apparently secondary to changes in ambient temperature and relative humidity. One of the sojourners experienced only generalised fatigue. This suggests that physiological alterations occurring at 3300 m cannot apparently induce acute mountain sickness in residents of medium altitude.

Key words/phrases: Cardiopulmonary functions, high altitude, Sojourners, working capacity

INTRODUCTION

The physiology of man at high altitude is still a subject of great interest and exploration. The emphasis given to the study of natives of different altitudes and sojourners moving to high altitudes is now very considerable. On ascending to 2300 meters and above, physiologic adjustments occur in lowlanders to compensate for the thinner air and accompanying low alveolar oxygen level (McArdle *et al.*, 1991). Lowlanders ascending to even 1600-2000m have been observed to develop some physiological adjustments (Anderson *et al.*, 1953; Banchero, 1987). For instance, maximum O₂ uptake (VO₂ max) is reduced by 10-40% in individuals ascending to 2000-4000m above sea level (Andersen, 1973). VE measured in sea level residents is much lower than the one measured in residents living at an altitude of one mile above sea level, the variation being about 1.8 L/m² body surface area (Anderson *et al.*, 1953). A mild degree of dyspnoea and dizziness was observed by the author in a few visitors who came to Addis Ababa (about 2440 m above sea level) at different times from places in Europe where the altitude is close to sea level. According to confirmed observation to-date, effects of altitude are physiologically significant at 3000 m and above to produce acute mountain sickness (Heath and Williams, 1981; West and Lahiri, 1984; Banchero, 1987). At 2850-3000 meters and above oxygen deprivation starts to become increasingly more significant at rest and during physical activity, alveolar spaces enlarge, signs and symptoms of acute mountain sickness become evident in many subjects with the incidence being higher in the young than in the old (Hacket and Rennie, 1976; Heath and Williams, 1981). The immediate and important physiological responses to an altitude of 3000 m and beyond are: (1) an increase in respiratory drive leading to hyperventilation due to stimulation of chemoreceptors by hypoxia, and (2) an increase in heart rate and blood volume flow at rest and during submaximal exercise (Heath and Williams, 1981; Schoene, 1984). These responses are different in lowland and highland residents. High altitude imposes certain difficulties in lowlanders whose reflex attempt will be to maintain and coordinate bodily functions on arrival at high altitude (Hurtado, 1971; Monge and Leon-Velarde, 1991). The physiological and biochemical features of high altitude acclimatization begin to become more pronounced with prolonged stay (Hacket and Rennie, 1976; Winslow, 1984; Jurgens *et al.*, 1986). The short-term or homeostatic responses are, however, expected to occur in minutes to hours.

The study made on certain ranges of altitude below and above the threshold of acute mountain sickness (3000 m) in Ethiopia, is inadequate. Besides, there are regional differences and whether or not 3000 m altitude is the threshold point at which most physiological variables invariably begin to alter for acute mountain sickness to develop in residents of medium altitude is not depicted. Observations on cardiopulmonary responses to low altitude with high ambient temperature and relative humidity in this part of the

world are also lacking. There are Ethiopians who occasionally travel and work in some parts of the Danakil and close to the peaks of Semien mountains (> 4,000 m above sea level). We do not know whether or not the physiological status of these people can be considerably affected during the initial period of their stay (before natural acclimatization) at altitudes higher or lower than their altitude of residence. Apparently, the heat in the lowland and the hypoxia and cold weather at high altitudes are liable to hamper man's working capacity and cardiopulmonary function. It is understood that people living at the same altitude but in different parts of the world appear to adjust to the environment differently. For instance, arterial blood pressure is greater in highland Ethiopians than it is in the lowlanders and this is the reverse of what is observed elsewhere in the world (Marticorena *et al.*, 1962; Lancet Annotations, 1975; Clegg and Harrison, 1976). We do not know if other physiological variables could show the same peculiarity in Ethiopians who are living not only at different levels of altitude but are also travelling between different altitudes as sojourners. It is not also documented if the status of the airways could be altered when one acutely changes one's altitude of residence. Therefore, the present study was conducted in sojourners staying for about three hours at a given altitude to: 1) determine the effect of high and low altitudes with markedly different ambient conditions on lung function indices, minute ventilation, saturation of arterial blood with O₂, arterial blood pressure, pulse rate and working capacity, 2) see if Ethiopian sojourners living at medium altitude would have a different altitude threshold level for developing acute mountain sickness.

MATERIALS AND METHODS

Subjects and sites of study

Cardiopulmonary variables and working capacity in eight healthy young male Ethiopian sojourners, ages 20–26 years, height 169.13±4.54 cm, residing at 2440 m above sea level, were studied in relation to acute exposure to descending and ascending altitudes. The study was conducted during a three-hour stay at altitude levels of 2440 m (Addis Ababa), 1370 m (Shewarobit), 490 m (Dubti), 2550 m (Dessie), and 3100–3300 m (peak of Tarmaber, Debresina). These altitudes were measured using a pocket altimeter (Revue Thommen Ag, Switzerland) calibrated at a known site of altitude (Geophysical Observatory, Addis Ababa University).

Study variables

The variables measured were forced vital capacity (FVC, L/sec), forced expiratory volume in one second (FEV₁, L/sec), FEV₁%, forced expiratory flow rate (FEF₂₀₀₋₁₂₀₀ ml, L/sec), forced mid-expiratory flow rate (FMF₂₅₋₇₅ %, L/sec), peak expiratory flow rate (PEFR, L/min), minute ventilation (VE, L/min), saturation of arterial blood with oxygen (SaO₂%), arterial blood

pressure (BP, mmHg), pulse rate (PR, beats/min) and working capacity (Kgm/min/m² body surface area). All instruments used for measuring these variables were standardised and calibrated before measurements were made. Physiological variables were measured at the beginning and at end of the three-hour stay at each altitude between 10:00 a.m. and 3:00 p.m. The study was conducted over a period of four consecutive days. Each subject rested for 30–40 minutes before measurements were made. Ambient temperature and relative humidity were recorded at each altitude.

Lung function indices

Procedures of lung function measurements were explained and demonstrated to subjects for better co-operation and accuracy. FVC, FEV₁, FEF_{200–1200} ml and FMF_{25–75}% (Table 1) were extrapolated from an expiratory spirogram (vitalogram) produced twice by a vitalograph (PFT II PLUS) calibrated using a one-litre syringe (plunger). Vitalography was performed at 2440 m, 1370 m, 490 m, 2550 m and 3100 m above sea level. All readings were taken at BTPS. FEV₁% was calculated using the formula: FEV₁/FVC×100. Two readings of PEF_R were also made using a Wright Peak flow meter. For lack of electric power supply, expiratory spirogram could not be recorded at 3300 m where all other variables were measured.

Table 1. The Effect of Acute Exposure to Altitude on Lung Function Indices (M±SD) of Eight Male Sojourners. Baseline measurements given in bold.

Altitude	FVC L/sec	FEV ₁ L/sec	FEV ₁ %	FEF _{200–1200} ml	FMF _{25–75} %	PEF _R L/min
490 m	3.37±0.45	3.04±0.38	90.5±1.77	6.44±1.80	4.73±0.76	394.09±37.24
1370 m	3.69±0.58	3.38±0.54	90.75±1.58	5.71±1.10	4.63±0.67	356.89±39.10
2440 m	3.45±0.38	3.12±0.41	90.1±2.31	6.54±1.23	4.67±0.82	350.21±32.73
2550 m	3.36±0.41	2.97±0.35	88.75±4.20	7.18±1.56	4.93±1.10	349.43 ±41.52
3100 m	3.41±0.38	2.99±0.37	87.6±3.38	8.33±0.63	4.54±1.15	332.38 ±25.7

FVC, Forced vital capacity; FEV₁, Forced expiratory volume in 1 sec.; FEF_{200–1200} ml, Forced expiratory flow rate; FMF_{25–75}%, Forced mid expiratory flow rate; PEF_R, Peak expiratory flow rate

Minute ventilation (VE)

This was measured using a Douglas bag connected to the study subject through a corrugated tube with a respiratory valve and a mouthpiece. A clip was put on the nose of each subject during tests. At rest and during exercise, expired air was collected in the Douglas bag at different altitudes for a period of 5 minutes and was measured by passing it through a gas meter. The value obtained was divided by 5 to get the minute ventilation in L/min. To determine the maximum workload, each subject was instructed to work on a bicycle ergometer at each altitude until a point of exhaustion was reached. Cessation of exercise depended on the tolerance of each subject to a workload fixed for each of them (3.0–4.5 kp). The duration of exercise was

1.5–3 minutes at 490–3100m and only 70–90 sec at 3300 m for all subjects. All values of VE obtained were corrected to BTPS.

SaO₂% and pulse rate

These were approximated by NELLCOR N-180 pulse oximeter checked for accuracy at the altitude of residence. The working of this pulse oximeter with automatic oximetry calibration is based on the principles of spectrophotometry and plethysmography. A clip of the oximeter containing a light source and a photocell was used to enclose one of the subject's fingers from which SaO₂% and pulse rate were sensed and read on the oximeter.

Arterial blood pressure

The conventional Riva-Rocci method was used to estimate arterial blood pressure. Two readings were taken in each subject at the different altitudes to ascertain reproducibility. The mean±SD of systolic and diastolic blood pressure was considered. The overall mean BP was not considered; instead systolic and diastolic BP were considered because it is understood that systolic and diastolic BP could show different responses to the change in testing conditions.

Working capacity

All subjects were instructed to work on the Monark bicycle ergometer (Astrand, 1960) at a workload or tension fixed for each of them for as long as possible. At the prescribed tensions given above, subjects were able to tolerate exercise for 1.5–3 min at 490–2550 m and for only 70–90 sec at 3300 m. Exercise was maximal. Cycling was synchronised with a metronome beat (100 beats/minute). The bicycle had a pedal wheel of 6 meters revolving at 50 rpm, i.e. the brake wheel travels at 300 m/min. The work done per minute was calculated using the formula, $W = \text{Force (KP)} \times \text{distance (m)}$ of brake wheel. The total work done in kgm/min was divided by the body surface area of the subject to get the total working capacity in kgm/min/m² body surface area.

Statistical analysis

All data are presented as mean±SD and compared by using the paired and unpaired t-tests. The softwares INSTAT2 and Graph pad prism were used for the above and other statistical analyses. A p value of <0.05 was taken as significant.

RESULT

Lung function indices

Descending towards sea level or ascending beyond the altitude of residence (2440 m), the eight sojourners in this study did not seem to show considerable changes in lung function indices including FVC, FEV₁, FEV₁%, FMF₂₅₋₇₅%, and PEF_R (Table 1). However, mean FEF₂₀₀₋₁₂₀₀ ml increased from 6.54 L/sec at 2440 m to about 8.33 L/sec at 3100 m altitude. Analysis relating the different study altitudes with mean values of FEF₂₀₀₋₁₂₀₀ ml showed a correlation value of $r=0.7316$. The linear regression is as shown in Fig. 1.

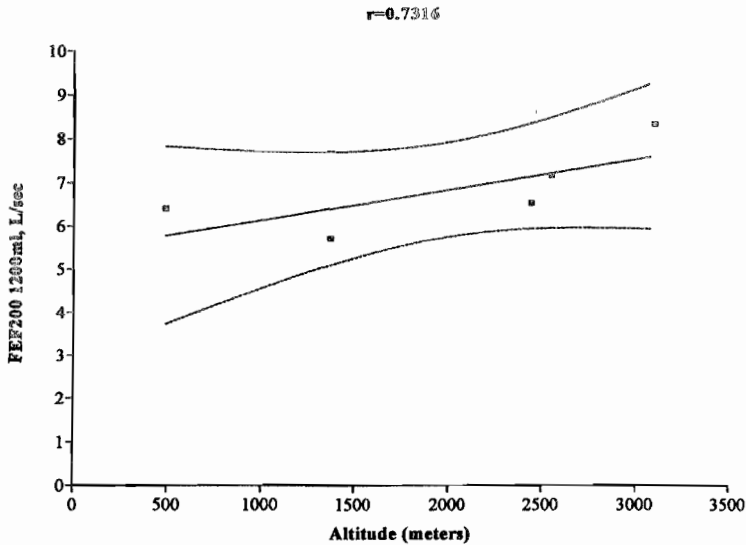


Fig. 1. Linear regression and correlation of FEF 200–1200 ml with altitudes of 490 m, 1370 m, 2440 m, 2550 m, and 3100 m above sea level. (Linear regression; 95% confidence interval.)

Minute ventilation (VE)

Resting mean VE at the altitude of residence was found to be 5.62 ± 1.5 L/sec. During exercise, it rose to 25.72 ± 4.12 L/sec, almost a fivefold increase. On descending to 1370–490 m, it was about 5.5 L/sec at rest and about 26–27 L/sec during exercise, a fivefold increase again. This means that a descent of such magnitude does not affect minute ventilation. On ascending to 2550–3300 m of altitude, both resting and exercise ventilation increased in general (Table 2). At 3300m of altitude it increased by 38.43% at rest and by 44.25% during exercise. The observed pattern is that \dot{V}_E is quite correlated to ascent ($r=0.6698$) and to mean working capacity ($r=-0.6426$).

Oxygen saturation of arterial blood ($SaO_2\%$)

$SaO_2\%$ ($94.0\pm 1.21\%$ at the altitude of residence) showed no considerable change during exercise at 490–2550 m but it decreased from $92.2\pm 3.14\%$ at rest to $89.6\pm 2.31\%$ during exercise at 3100 m and from 91.9% at rest to 86.9% during exercise at 3300 m (Table 2). Its correlation with altitude was high (Fig. 3).

Working capacity

Working capacity increased by about 9% with descent to 490 m (Fig. 2, Table 2); it decreased by about 20% ($P < 0.001$) with ascent to 3300 m (Table 2). The changes relating mean total working capacity to altitude ($r = -0.9101$) and to $SaO_2\%$ ($r = 0.7955$) were significant ($P < 0.05$). Individual measurements of working capacity and $SaO_2\%$ were also significantly correlated ($r = 0.7628$; Fig. 3) at only 3300.

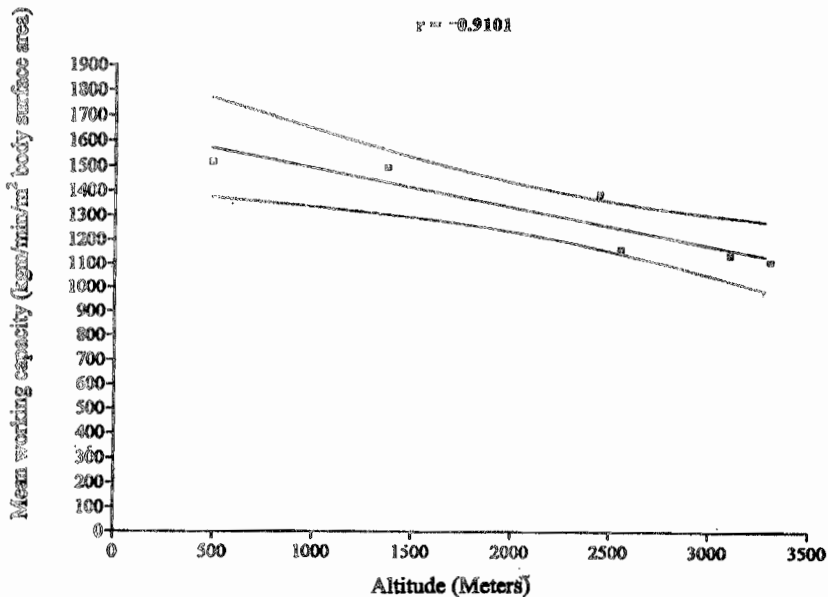


Fig. 2. Linear regression of mean working capacity (kgm/min/m² body surface area) of eight Ethiopian male sojourners (ages 20–26, height, 169.13 ± 4.54 cm) measured at altitudes of 490 m, 1370 m, 2440 m, 2550 m, 3100 m and 3300 m above sea level. (Linear regression; 95% confidence interval.)

Pulse rate

Mean pulse rate was 75.4 ± 8.9 beats/min at the altitude of residence (ambient temperature= 21° C, relative humidity= 50%), 82 beats/min at 490 m (ambient temperature= 38° C, relative humidity= 70%) and 79 beats/min at 1370 m (ambient temperature= 32° C, relative humidity= 58%). On ascent to 2550 m (ambient temperature= 20° C, relative humidity= 49%), 3100 m (ambient

temperature=17° C, relative humidity =31%), and 3300 m (ambient temperature=16° C relative humidity =30%), the mean pulse rates recorded decreased to about 74, 73 and 72 beats / min, respectively (Table 2).

Table 2. The Effect of Acute Exposure to Altitude on Cardiopulmonary Variables and Working Capacity (M±SD) of Eight Male Sojourners. Base line measurements given in bold.

Altitude	PR	BP	SaO ₂ %		VE		Working capacity	
	Rest	Rest	Rest	Exercise	Rest	Exercise	Average load (kp)	Work done (kgm)
490 m	82.3±9.23	S=93.1±5.94 D=59.4±5.63	96±0.93	95±2.17	5.46±1.56	26.96±4.11	3.56±0.5	1516±526.72
1370 m	79.3 ±15.8	S=99.6±11.78 D=59.4±9.43	95.5±1.26	95±1.31	5.5±1.35	25.84±3.73	3.56±0.50	1493.96±476.26
2440 m	75.4±8.9	S=100.1±10.8 D=62.13±5.2	95.8±2.31	94±1.21	5.62±1.22	25.72±4.21	3.56±0.50	1385.47±387.3
2550 m	74.3±9.41	S=102.5±11.3 D=63.1±7.53	95.5±2.12	94±1.85	6.61±0.71	25.69±5.20	3.56±0.50	1159.9±328.17
3100 m	73.2±9.5	S=101.46±2.21 D=62.8±9.82	92.2±3.14	89.6±2.31	7.1±1.21	27.8±7.23	3.56±0.50	1134.8±302.14
3300 m	72.5±7.84	S=101.6±7.29 D=62.5±10.35	91.9±4.39	86.9±9.58	7.78±1.43	37.1±10.15	3.56±0.50	1106.5±306.56

PR, Pulse rate (beats/min); BP, Arterial blood pressure (mmHg); S, systolic; D, diastolic; VE, Minute ventilation (L./min); SaO₂%, O₂ saturation of arterial blood; Kp, kilopond; Kgm, kilogram-meter.

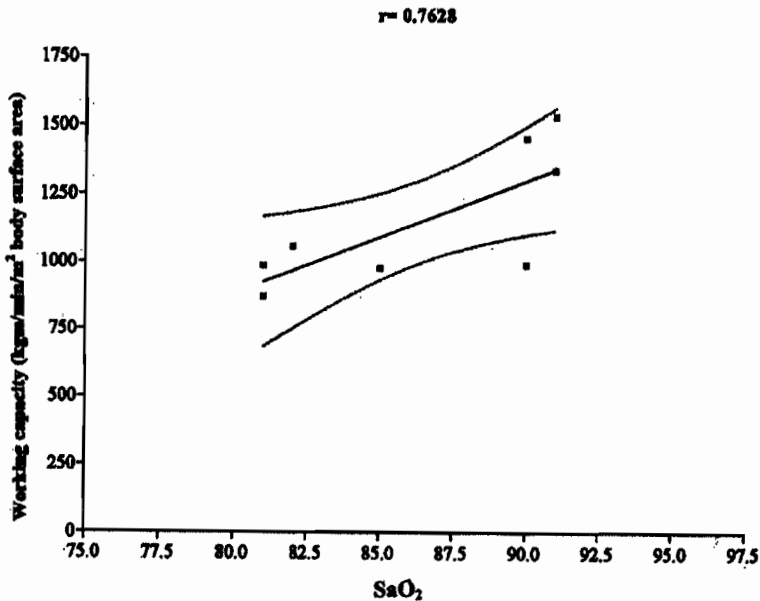


Fig. 3. Linear regression and correlation of working capacity (kgm/min/m² body surface area) with O₂ saturation (SaO₂%) in eight male sojourners studied at 3300 m above sea level. Individual measurements were correlated (r=0.7626) using the software INSTAT2. (Linear regression; 95% confidence interval.)

Systolic/diastolic blood pressure

This was 100.1/62.13 mmHg at 2440m. It showed a tendency to decline with decreasing altitude (93.1/59.4–99.6/59.4 mmHg) and to rise with increasing altitude (101.46/62.8–102.5/63.1mmHg) as shown in Table 2.

DISCUSSION

Individuals moving from low to high altitude have varying abilities to tolerate hypoxia via physiological reserves and long-term acclimatization. It is also observed that humans moving to similar altitudes in different parts of the world develop physiological changes that are markedly different. This may be attributed to prevailing environmental conditions like ambient temperature and relative humidity and perhaps partly to differences in anthropometric values and individual susceptibility.

Previous study by Astrand (1960) showed that there was no correlation between O₂ uptake and FVC and FEV1 apparently reflecting that lung function values do not change with corresponding change in O₂ uptake. In the present study, most lung function indices measured at altitudes above or below the altitude of residence (2440 m) showed only little or no variations in eight young male sojourners (Table 1). Of the lung function indices, however, FEF_{200–1200} ml measured at 2550–3100 meters was found to be higher than the baseline value (Table 2). There was no change with descent. Care was taken to avoid artifacts of analysis and co-operation of subjects was ascertained. Therefore, it is possible to believe that the relative increase in FEF_{200–1200} ml probably reflects that large airways start to open up more widely perhaps concomitant with the enlargement of alveolar spaces at 2550 m and above to compensate for the drop in PaO₂. The results observed in SaO₂ %, VE and working capacity in the eight young sojourners acutely exposed to the higher levels of altitude appeared to show quite significant changes. SaO₂% was about 96% at 2550 m–490 m at rest and 94–95% during exercise. The corresponding values at 3100–3300m were about 92% at rest and 87–90 % during exercise. Exercise at 3300 m appears to be relatively more potent in inducing O₂ desaturation (Table 2). The low degree of saturation is apparently signified by the significant increase in minute ventilation at 3300m.

At the altitude of residence, VE was 5.62 L/min at rest and 25.72 L/min during exercise. At descent to 1370–490 m, the corresponding mean values did not differ significantly (Table 2). There was quite a progressive increase with ascent. VE increased to 7.78 L/min (38.43% increase) at rest and to 37.1L/min (44.25% increase) during exercise at 3300 m. It was also quite correlated to altitude ($r = 0.6698$) and to mean working capacity ($r = -0.6426$). It was shown in previous studies that VE was much higher in residents of one-mile altitude than in sea level residents both at rest and during exercise

(Anderson *et al.*, 1953) indicating that there is greater O₂ utilization even at an altitude of 1600 m above sea level.

In general, the moderate rise in VE, the decrease in SaO₂% and working capacity at 3100-3300 m of altitude apparently show that hypoxia begins to develop to affect one's physical ability for performance at altitudes higher than 3000m. At 3300 m, the pattern of change was more marked for working capacity, which showed a significant correlation with the concurrent decrease in SaO₂ % ($r = 0.7955$ for mean values at 490-3300 m and $r = 0.7626$, Figure 3, for individual measurements recorded only at 3300m). The decrease in working capacity at high altitude is even more significant when the stay is prolonged because hypoxia becomes chronic and more limiting to physical performance (Heath and Williams, 1981). The increase in the mean of total working capacity with descent from 2440 m to 490 m was only 9.42%; with ascent from 2440 m to 3300 m, its decrease was 20.14% thus showing again that O₂ desaturation with ascent apparently decreases physical performance. The overall mean working capacity was significantly negatively correlated with altitude levels ($r = -0.9101$, Table 2, Fig. 2).

Overall mean pulse rate was slightly and paradoxically decreased (from a mean of 82 beats/min to 72 beats/min) as sojourners ascended from 490 to 3300 m of altitude. This is probably a change in the circadian rhythm secondary to the progressive decrease in the ambient temperature and relative humidity (Table 2) occurring with the increase in altitude. The overall mean systolic/diastolic blood pressure increased from 93.1/59.4 mmHg at 490 m to 99.6/59.4 mmHg at 1370 m, to 102.5/63.1 mmHg at 2550 m, and to 101.6/62.5 mmHg at 3300 m (Table 2). This pattern of change is in agreement with the peculiarity of BP changes observed in highland and lowland residents in Ethiopia. In general, the arterial blood pressure seems to show a slight decline with descent and quite an increase with ascent. This apparently signifies that the ambient conditions in this range of altitude induce changes in the peripheral vascular resistance. The pressure variations can be attributed to apparent changes in vascular resistance secondary to changes in ambient temperature and relative humidity at different altitudes. The ambient temperature and relative humidity in Dubti (490 m) were so high (38° C and 70%, respectively) that sojourners were feeling sticking hot and sweated profusely at the time of study. This probably indicates that the peripheral vascular resistance was low enough to induce vasodilatation that apparently leads to a drop in arterial blood pressure and a concurrent increase in pulse rate. The reverse was apparently happening at high altitude where the ambient temperature and relative humidity were much lower (16°C and 30% at 3300 m). Since the exposure to hypoxia was acute, there would be no fall in total peripheral vascular resistance to induce a drop in arterial blood pressure at high altitude. Besides, cold weather at high altitude can induce vasoconstriction and perhaps an increase in peripheral vascular resistance. The decrease in hand blood flow observed at high altitude

(Durand and Martinaud, 1971) and the peripheral vascular resistance progressively increasing with ascent (Vogel *et al.*, 1974) are probably partly a reflection of the effect of the ambient conditions. The present results are based on short exposure of subjects to different altitudes and I don't know if the findings can be partly attributable to the Ethiopian peculiarity (high arterial blood pressure in highland Ethiopians and a lower one in the lowlanders as observed by Clegg and Harrison (1976)). For certain, the pattern of change in these medium altitude residents is at least partly related to the prevailing ambient conditions.

The reverse is true elsewhere in the world. With a progressive increase in ascent, arterial blood pressure decreases (Martecorena *et al.*, 1962; Lancet Annotations, 1975; Clegg and Harrison, 1976); so do cardiac output and stroke volume (Vogel *et al.*, 1974; Heath and Williams, 1981). With this in mind, the general belief is that the chance for the development of hypertension at high altitude is low. This could be unlikely for Ethiopians with a different pattern of cardiovascular adaptation.

The sojourners in this study, being residents of about 2440 m above sea level, were relatively less sensitive to acute exposure to the present ascent of altitude. Only one of them experienced generalized fatigue and weakness at 3300m. None of them experienced the common signs and symptoms of acute mountain sickness. Besides, the occurrence of alterations in many physiological variables in response to high altitude is subject to great individual and regional variability. It is, therefore, reasonable to suggest that even 3300 m cannot be taken as an altitude threshold point that can invariably induce marked physiological alterations and mountain sickness in Ethiopians residing at medium altitude.

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