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G.R. ZUBIETA-CALLEJA[1, 2], P-E. PAULEV[1, 2], L. ZUBIETA-CALLEJA[1],
N. ZUBIETA-CALLEJA[1], G. ZUBIETA-CASTILLO[1]

HYPOVENTILATION IN CHRONIC MOUNTAIN SICKNESS: A MECHANISM TO PRESERVE ENERGY

[1]High Altitude Pathology Institute, La Paz, Bolivia; [2]Panum Institute,
Medical Physiology Institute, University of Copenhagen, Copenhagen, Denmark

Chronic Mountain Sickness (CMS) patients have repeatedly been found to hypoventilate. Low saturation in CMS is attributed to hypoventilation. Although this observation seems logical, a further understanding of the exact mechanism of hypoxia is mandatory. An exercise study using the Bruce Protocol in CMS (n = 13) compared to normals N (n = 17), measuring ventilation (VE), pulse (P), and saturation by pulse oximetry (SaO_2) was performed. Ventilation at rest while standing, prior to exercise in a treadmill was indeed lower in CMS (8.37 l/min compared with 9.54 l/min in N). However, during exercise, stage one through four, ventilation and cardiac frequency both remained higher than in N. In spite of this, SaO_2 gradually decreased. Although CMS subjects increased ventilation and heart rate more than N, saturation was not sustained, suggesting respiratory insufficiency. The degree of veno-arterial shunting of blood is obviously higher in the CMS patients both at rest and during exercise as judged from the SaO_2 values. The higher shunt fraction is due probably to a larger degree of trapped air in the lungs with uneven ventilation of the CMS patients. One can infer that hypoventilation at rest is an energy saving mechanism of the pneumo-dynamic and hemo-dynamic pumps. Increased ventilation would achieve an unnecessary high SaO_2 at rest (low metabolism). This is particularly true during sleep.

Key words: *arterial oxygen saturation, chronic mountain sickness, heart rate, ventilation*

INTRODUCTION

High altitude residence with low barometric pressure gives rise to adaptation to a different environment as compared with sea level. Acute exposure can produce acute mountain sickness in about 25 % of those going to the altitude of

3510 m. However, after around 2 days at altitude, most people gradually adapt and feel as well as at sea level and are able to carry on a normal life. The normal sea level hematocrit of 45% (in young males) gradually increases upon arrival to high altitude to around 50% and from the sea level point of view this condition is classified as polycythemia. This physiologic polycythemia is actually part of the normal adaptation process. However, for the high altitude physician, the 50% value is considered a normal hematocrit. Some long term residents at altitude have been observed to suffer what is known as chronic mountain sickness (CMS) (1). They present a higher hematocrit than normal residents and for 3510 m altitude the threshold is considered to be 58% (2). High altitude physicians call this "polycythemia", whereas sea level colleagues call it "increased polycythemia". Relativity, as described by Einstein, is also applicable to altitude differences.

CMS patients are cyanotic and have a typical physiognomy that is easily recognizable by the experienced physician. When examined, these patients not only present a high hematocrit, but also low oxyhemoglobin saturation (SaO_2), as measured by pulse oximetry or through arterial blood gases. Whereas sea level residents have a SaO_2 of 98%, normal residents at 3510 m present 91% and CMS patients below 85% (3). This is clearly a low saturation that results from hypoxemia. It can even reach very low levels at around 60% when CMS patients are suffering acute diseases such as an intense flu or pneumonia. These three levels of hypoxia (hypobaric hypoxia + CMS hypoxia + acute lung disease) have been named by us as the triple hypoxia syndrome (4). The third hypoxia is reversible by hyperoxic therapy and treatment of the underlying cause.

Ventilation measured in CMS patients has repeatedly been found to be low as compared with normals (5-8). Hence some recent medical reviews have attributed CMS to hypoventilation (9). Normal, sedentary sea level residents present a gradual increase in ventilation and heart rate during incremental exercise (10-12). Saturation is sustained along with PaO_2, but may suffer an increase at the last stage. This sustained saturation is explained by recruitment of normally resting non-ventilating regions in the lower part of the lung, and the subsequent increase in tidal volume.

Normal high altitude sedentary residents gradually decrease slightly the SaO_2 during exercise (13). However, well trained athletes are able to sustain the SaO_2 at resting levels during the first 3 stages of exercise with a small decrease at the end of the test (14). In the present study, CMS patients performed a treadmill exercise test and the behavior of ventilation, SaO_2, and pulse was evaluated during rest prior to exercise and during exercise.

MATERIAL AND METHODS

The study was approved by an institutional Ethics Review Board. Thirteen CMS patients with increased polycythemia, called from now on polyerythrocythemia, as the authors deemed it to be the most convenient denomination, were compared with 17 normal young men in the military (N). Results are shown in *Table 1*.

Table 1. Physiological data of the two groups studied.

	n	Age (yr)	Weight (kg)	Ht (%)	SaO_2 (%)
Normal	17	19.7 ±1.7	65.1 ±5.9	50.0 ±2.1	90.4 ±1.7
CMS	13	54.8 ±11.7	73.6 ±13.1	72.1 ±5.3	87.2 ±3.0

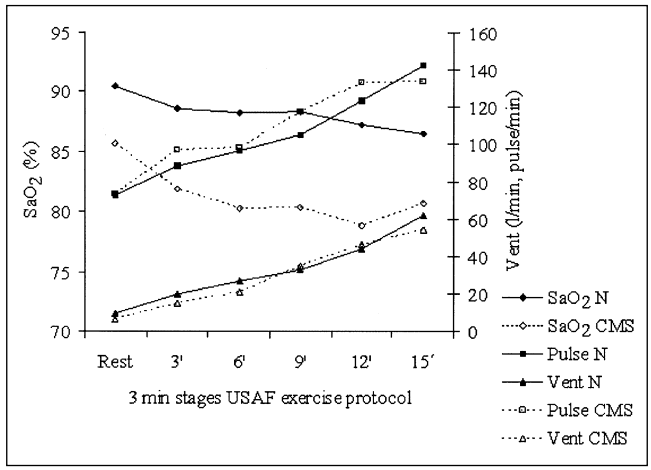

Fig. 1. SaO_2, pulse, and ventilation in normals (n=17) and CMS patients (n=13) during standardized cardio-pulmonary exercise testing using the USAF exercise protocol at 3510 m above sea level.

Both groups performed a USAF modified treadmill exercise protocol, similar to the Bruce protocol, with incremental gradient/mph 0/0, 0/2, 0/3, 5/3, 10/3, 10/4 during 3 min each. The measured variables were: ECG, ventilation (BTPS), ETO_2, $ETCO_2$, PEO_2, $PECO_2$, and pulse oximetry, and the calculated ones included: VO_2, VCO_2, and RQ. Resting ventilation was initially measured with the subjects standing up prior to exercise with a face mask, after 10 min of rest and habituation to the mask and with previous training for the treadmill exercise maneuver. Statistical analysis was performed using Student's *t*-test.

RESULTS

The mean minute ventilation at rest (standing position) in BTPS l/min in N and in CMS was 9.54 ±1.85 l/min and 8.73 ±2.33 l/min, respectively. Although the difference did not assume statistical significance, ventilation clearly tended to be lower in the CMS patients, as previously reported. The exercise results are shown in *Fig 1*.

DISCUSSION

Malnourished patients with chronic obstructive pulmonary disease (COPD) are characterized by a relative increase in resting energy requirements and, specifically, increased energy requirements for augmenting ventilation (15). On

the other hand, other authors affirm that hypoventilation causes the most important gas exchange alteration in COPD patients leading to hypercarbia and hypoxemia (16). This concept has been generalized and inadequately used to explain hypoventilation in CMS. In children, Ondine's curse constitutes an example of primary hypoventilation of genetic origin, which is a different entity of severe alteration (17) and should not be confused with hypoventilation in CMS.

Upon arrival to high altitude, hyperventilation and tachycardia are the immediate biological compensating strategies for hypobaric hypoxia. A respiratory quotient (RQ) of 0.8 is typical at sea level but at the high altitude of La Paz, it is around 0.9 (18). This is due presumably to hyperventilation. Prior to the exercise test, it is quite difficult to acquire a resting RQ of 0.9, since the subjects are in the standing position in the treadmill. This would imply additional VO_2 from the use of the orthostatic muscles and to some degree a tense wait for the exercise test to begin.

Basal metabolic rate (BMR) is equal to the oxygen consumption of the whole body at rest. This includes the resting global cellular oxygen consumption plus the two pumps, the heart (hemodynamic pump) and the respiratory muscles (pneumodynamic pump). These two organs constitute the driving systems for oxygenation and hence their energy expense can be reduced if some other system in the body compensates in order to make oxygen transport to the cells efficient. The heart muscle consumes around 24 ml O_2/min. The respiratory muscles consume 5% of the total resting VO_2 (19). Assuming a VO_2 of 250 ml/min, this would amount to around 12 ml O_2/min. Both systems together consume 36 ml O_2/min. This is roughly 15% of the total energetic cost. If a reduction of 1% is achieved (2.5 ml O_2/min), it may not seem too much, but when reported in 24 h it amounts to 3600 ml. Recall that this calculation assumes a permanent resting condition.

The exercise tests show a low initial SaO_2 in CMS. Undoubtedly, this is due to pulmonary insufficiency of some sort as reported before (3). If the organism would try to compensate the respiratory insufficiency through hyperventilation, the energy cost would be too high, making the biologic system completely inefficient and hence tending toward a progressive deterioration. Therefore, an increase of the hematocrit allows for the least energy expense. Poon (20) has previously mentioned an optimization of ventilation, but this refers to the ventilatory response during exercise, where ventilatory output (VE) is set by the respiratory center to minimize a net operating cost. The present paper presents the resting ventilation in CMS patients at high altitude, as the energy saving mechanism in the presence of lung disease.

During exercise, CMS patients also have a significant decrease of SaO_2, although their ventilation and cardiac frequency are higher in the first 4 stages of exercise compared with normals. This observation confirms that these patients have an abnormal cardio-respiratory system, since the increase of the pulse and

ventilation should (if the low saturation were due solely to centrally induced hypoventilation) sustain the SaO_2 or increase it to normal high altitude levels.

In conclusion, the low SaO_2 during exercise shows that even though the pneumo-dynamic and hemo-dynamic pumps are working well above that of the normal control group, there is a deficiency in the pneumo-dynamic pump, which is due to pulmonary insufficiency (veno-arterial shunts and uneven ventilation). Hence it is inferred that hypoventilation with low arterial oxygen saturation at rest is an energy saving mechanism. This is possible thanks to an increase in the number of red blood cells that allows the involved cardio-respiratory muscles to consume the least amount of oxygen required.

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Authors address: G. R. Zubieta-Calleja, High Altitude Pathology Institute (IPPA), P.O. Box 2852, La Paz, Bolivia; phone: 591 2 2245394, e-mail: gzubietajr@altitudeclinic.com, www.altitudeclinic.com