Effect Of Aerobic Capacity On The Incidence And Severity Of Acute Mountain Sickness After Passive Ascent To 3450 m

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1. Project description

Ascent to high altitude is a known hypoxic stressor requiring rapid adaptive responses in order to maintain cellular integrity. The earliest and most consistent responses are i) increase in pulmonary ventilation (V̇e) mediated primarily by hypoxic stimulation of the peripheral chemoreceptors (1); ii) increase in cardiac output mediated by sympatho-adrenal activation (2); and iii) reduction in blood volume by inhibition of the renin-angiotensin-aldosterone axis in order to improve oxygen transport capacity (3, 4). In case these responses fall short of mitigating the reduction in convective oxygen delivery, the risk of suffering high-altitude associated diseases increases. Among these, acute mountain sickness (AMS) is the most common one and occurs in ~30% of non-acclimatized subjects ascending too fast to altitudes ~3500 m (5). AMS is a syndrome consisting of non-specific symptoms - i.e. headache, nausea, lightheadedness, fatigue, and poor sleep - of which the pathophysiology is incompletely understood (6). The leading theories are increased cerebral blood flow caused by cerebral vasodilation secondary to reduced hypoxic ventilatory drive, exaggerated sympatho-adrenergic response and increased blood volume.

Based on anecdotal reports (7) and our own observations from previous studies, individuals with a high aerobic capacity or maximal oxygen uptake (V̇O2max) upon spiroergometry, as endurance athletes, might be even at particular risk for developing AMS. This, at first glance paradox appearing hypothesis, might make more sense when comparing the above mentioned pathophysiological mechanisms contributing to AMS with physiological characteristics of endurance trained athletes, namely reduced hypoxic ventilator drive (8, 9), increased sympatho-adrenal activity upon stress (10) and increased blood volume (11). Surprisingly, current literature does not support this theory and states that physical fitness appears to have no association with susceptibility to AMS (12). Still, studies assessing the association of physical fitness and AMS have defined fitness heterogeneously using questionnaires or routine ergometry only.

Accurate prediction of AMS susceptibility is of importance to enable enhanced prevention measures through increased observation, behavioral changes (e.g. slower ascent profile), and/or prophylactic medication. Preliminary risk assessment is even more important in an athletic population that is prone to higher risk activities and is driven to reach the highest and technically-demanding peaks at a fast pace. In addition, investigating how individuals with high aerobic capacity respond to hypoxia holds promise to shed more light on the obscure pathophysiology of AMS.

To this end, this prospective, controlled study is the first investigating whether endurance athletes with exceptional high aerobic capacity have higher incidence and severity of AMS than sedentary individuals after rapid (< 2 h) and passive ascent from 750 to 3450 m.

All listed study investigations were performed between 3 and 46 h after arrival at 3450 m:

AMS-questionnaires: Assessment of incidence and severity of acute mountain sickness by use of two internationally standardized and well established questionnaires. As in our previous studies AMS was assessed using the Lake Louise scoring system and the cerebral symptoms of AMS (AMS-C) score of the Environmental Symptoms Questionnaire (13, 14).

Resting metabolic rate: The tests were performed under quiet conditions with a room temperature of 20°C-25°C. Energy expenditure was recorded using a metabolic analyzer (indirect calorimetry).

Hypoxic ventilatory response: Subjects breathed through a low-resistance spirometric device in order to determine minute ventilation (V̇E) during normoxia and hypoxia. Simultaneously, arterial oxygen saturation (SpO2) was recorded continuously using a pulse oximeter.

Carbomonoxide (CO)-Rebreathing: In order to assess blood volume a body size-related dose of CO was introduced into a low-volume closed-circuit rebreathing system. Carboxyhaemoglobin saturation for the estimation of blood volume was assessed by venous blood analysis before and after rebreathing maneuver.

Inert Gas Rebreathing: In order to assess stroke volume and cardiac output the inert gas rebreathing technique applied by the Innocor-device was used

Heart rate variability: Subject's heart rate and heart rate variability was assessed with a heart rate monitor using two chest electrodes.

Transthoracic echocardiography: Cardiac function and pulmonary artery systolic pressure was assessed using a portable ultrasound system.

Laboratory investigations: Samples of venous and capillary blood were drawn. In addition, 24 hours urine was collected for further analyses.

The study was performed in December 2018 and the data are currently under analyses.

References

1. Dempsey JA, Forster HV. Mediation of Ventilatory Adaptations. Physiol Rev. 1982; 62 (1): 262-346.
2. Naeije R. Physiological adaptation of the cardiovascular system to high altitude. Prog Cardiovasc Dis. 2010; 52 (6): 456-66.
3. Siebenmann C, Robach P, Lundby C. Regulation of blood volume in lowlanders exposed to high altitude. J Appl Physiol (1985). 2017; 123 (4): 957-66.
4. Siebenmann C, Cathomen A, Hug M, Keiser S, Lundby AK, Hilty MP, et al. Hemoglobin mass and intravascular volume kinetics during and after exposure to 3,454-m altitude. J Appl Physiol (1985). 2015; 119 (10): 1194-201.
5. Berger MM, Macholz F, Mairbäurl H, Bärtsch P. Remote ischemic preconditioning for prevention of high-altitude diseases: fact or fiction? J Appl Physiol (1985). 2015; 119 (10): 1143-51.
6. Hackett PH, Roach RC. High-altitude illness. N Engl J Med. 2001; 345 (2): 107-14.
7. Milledge JS, Beeley JM, Broome J, Luff N, Pelling M, Smith D. Acute mountain sickness susceptibility, fitness and hypoxic ventilatory response. Eur Respir J. 1991; 4 (8): 1000-3.
8. Byrne-Quinn E, Weil JV, Sodal IE, Filley GF, Grover RF. Ventilatory control in the athlete. J Appl Physiol. 1971; 30 (1): 91-8.
9. Chapman RF, Stray-Gundersen J, Levine BD. Epo production at altitude in elite endurance athletes is not associated with the sea level hypoxic ventilatory response. J Sci Med Sport. 2010; 13 (6): 624-9.
10. Kjaer M, Secher NH, Galbo H. Physical stress and catecholamine release. Baillieres Clin Endocrinol Metab. 1987; 1 (2): 279-98.
11. Heinicke K, Wolfarth B, Winchenbach P, Biermann B, Schmid A, Huber G, et al. Blood volume and hemoglobin mass in elite athletes of different disciplines. Int J Sports Med. 2001; 22 (7): 504-12.
12. Bärtsch P, Swenson ER. Acute high-altitude illnesses. N Engl J Med. 2013; 369 (17): 1666-7.
13. Berger MM, Macholz F, Lehmann L, Dankl D, Hochreiter M, Bacher B, et al. Remote ischemic preconditioning does not prevent acute mountain sickness after rapid ascent to 3,450 m. J Appl Physiol (1985). 2017; 123 (5): 1228-34.
14. Berger MM, Macholz F, Sareban M, Schmidt P, Fried S, Dankl D, et al. Inhaled budesonide does not prevent acute mountain sickness after rapid ascent to 4559 m. Eur Respir J. 2017; 50 (3).

Internet data bases

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