Arterial blood gases and oxygen content in climbers on the mount everest article ...

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The pressure of atmospheric oxygen decreases gradually as barometric pressure drops with increasing altitude. The ability to perform work, such as walking and climbing belittles with the decreased availability of oxygen in the atmosphere for aerobic respiration. The Mount Everest is the highest peak on the earth with an altitude of 8848 meters. At such a higher altitude, the partial pressure of inspired oxygen or PIO2 reaches the limit at which humans can maintain body functions, such as cognition and ambulation. In 1953, Hillary and Tenzing used supplemental oxygen while climbing the Everest. It was only 25 years later that Messner and Habeler climbed the Everest without using any supplemental oxygen. Only 4 percent of people who climb the Everest do not use supplemental oxygen. Two studies, namely, Operation Everest II and Operation Everest III published the measurements of PaO2 or partial pressure of oxygen in arterial blood after stimulating an ascent of the Mount Everest by positioning subjects in a hypobaric chamber.

The results obtained in the two studies for the mean (±SD) resting at a barometric pressure of 253. 0 mm Hg or 33. 73 kPa, equivalent to the summit of the Mount Everest were PaO2 of 30. 3±2. 1 mm Hg (4. 04±0. 28 kPa) and 30. 6±1. 4 mm Hg (4. 08±0. 19 kPa) respectively. Such a condition of having abnormally little oxygen in the blood was tolerable because the subjects had been gradually habituated to the stimulated altitude for a period of 37-40 days. In the year 1981, researchers measured the partial pressures of oxygen and carbon dioxide at end expiration of a person who was able to withstand the higher altitude without supplemental oxygen for ten minutes. The estimated Bohr integration of the chamber with the use of

Bohr integration was 28 mm Hg (3. 73 kPa). The subjects in the study were 9 healthy men and a woman in the age group of 22 to 48 years, who wrote written consent to ascend the Mount Everest as a part of the medical research expedition.

After ascending an altitude of 6800 meters, there was no evidence of any sort of ill effects from higher altitude in the subjects. Subjects ascending altitudes higher than 7950 meters previously ascended altitudes higher than 7950 meters with no incident. The researchers collected blood samples from the subjects at various altitudes of 5300 m, 6400 m, 7100 m and 8400 m using radial arterial cannulae. They collected blood sample from the right femoral artery identified by digital palpation after confirming the intra-arterial placement of the needle by filling heparinized 2-ml oiled glass syringe and sealed the syringes with an airtight cap in an insulated vacuum flask. The researchers allowed the subjects to use supplemental oxygen at altitudes higher than 7100 m at the rate of 2 to 3 liters per minute while climbing and 0. 5 liter per minute while sleeping. At altitudes of 7950 m, the researchers took arterial blood samples after allowing the subjects to breathe atmospheric air for four hours.

At the balcony or 8400 m, researchers collected the samples after allowing the subjects to breathe ambient air for 20 minutes to make sure that there is no left over of supplemental oxygen. After analyzing the arterial blood samples using RapidLab 348 blood gas analyzer, researchers measured the levels of PaO2, PaCO2 and pH. After calculating the values for the bicarbonate concentration, oxygen saturation (SaO2) and blood base excess using standard formulas, researchers arrived at certain conclusions. The

measurement of arterial blood gases and hemoglobin levels in ten subjects on the Mount Everest provided information of the pattern and limits of changes in human blood gases in response to hypobaric hypoxia at the highest altitude of the earth. The values of PaO2 and SaO2 reported during the research were the lowest values ever documented in humans. The decrease in the level of PaO2 is proportional to the fall in the barometric pressure at an increasing altitude. However, there was no major difference in the SaO2 values, which explains the characteristics of oxygen–hemoglobin dissociation curve and the decreased levels of PaCO2.

The increase in the levels of hemoglobin concentration counterbalance the fall in SaO2 levels so that the level of CaO2 remains constant until the subject reaches an altitude of 7100 m. At the highest altitude the subjects successfully acclimatized to prolonged abnormal availability of atmospheric oxygen. The subjects had clear cognition as recorded effectively by radio communication. Such absence of cognitive abnormalities suggests the risk of long-term cognitive deficit and structural neurologic damage due to exposure to extreme altitudes. The research also suggests the inactivity of anaerobic metabolism in producing energy at an extreme altitude when the subject is at rest. The research relates the role of supplemental oxygen in influencing the PaO2 and PaCO2 levels. The removal of supplemental oxygen in a hypoxic environment triggers hypoxic ventilatory response, which leads to hyperventilation within 10-30 minutes of exposure to atmospheric oxygen.

Hypoxia associated with an increased alveolar–arterial oxygen difference contributes to a ventilation–perfusion mismatch and a limitation in

pulmonary diffusion due to the disequilibrium in pulmonary alveolar–end-capillary diffusion. Increased basal atelectasis or central fluid shifts in the arterial blood samples prove dangerous to pulmonary gas exchange due to the inverse relation between the PaO2 levels and alveolar–arterial oxygen difference. Tissue hypoxia as a result of arterial hypoxemia is a universal phenomenon among critically ill persons. Also, hypoxia leads to several adaptive and maladaptive systemic responses of the body that do not have accurate answers. To conclude, the measurements of partial pressure of oxygen and carbon dioxide, hemoglobin, pH and lactate concentrations in the arterial blood of humans at extreme altitudes explain the functional limitation in pulmonary diffusion or subclinical pulmonary edema.