At altitudes approximately 5,000 feet above sea level, few problems are encountered with the administration of anesthesia. The slight effects that are seen are primarily in patients who have cardiorespiratory deficiencies which may produce hypoxic states. In Denver, Colorado, 5,280 feet (one-mile) above sea level, residents who are free of cardiorespiratory disease present few anesthesia problems with the use of nitrous oxide.

At altitudes above 10,000 feet, compensatory mechanisms (to be described later) are used to offset the decreased oxygen in the air. This is an important area to examine given that an estimated ten million people live at this altitude.

At 15,000 feet and above, however, oxygen must be added to the inhalation mixture to avoid hypoxia. Altitudes of 40,000 to 47,000 feet are man's limit when breathing 100% oxygen. As we reach altitudes above this, the ambient oxygen pressure must be raised.

Barometric pressure decreases as one rises to progressively higher and higher altitudes. This decrease in pressure and the resultant decrease in oxygen causes hypoxia in high altitudes. The pressure of alveolar oxygen falls disproportionately compared with carbon dioxide and water vapor. The sum of the partial pressures of agents and gases in the alveoli equals the atmospheric pressure. (By comparison, the effect of oxygen tension is reversed in sea diving. Oxygen tension increases as the pressure increases one atmosphere or 760 mmHg for every 33 feet of sea water.)

Physiologic changes

As the altitude rises, the partial pressure of the oxygen in the alveoli declines. Therefore, the arterial saturation level of the blood is lower.

Physiological changes and effects that may be noted in a patient suffering from hypoxia include: excitation, drowsiness, headaches, mental fatigue, lassitude, euphoria, and an increase in ventilation. The latter, which is most often seen as one of the body's compensatory mechanisms, begins at altitudes above 8,000 feet; the other mentioned physiological changes begin at 12,000 feet. Approximately 98% arterial saturation or less is sufficient to stimulate the chemoreceptors to increase ventilation.

At altitudes between 16,000 and 20,000 feet, ventilation has reached a maximum effectiveness. At altitudes above this, the chemoreceptors are not stimulated to increase ventilation any further.

When there is an increase in pulmonary ventilation, a reduction in carbon dioxide results. This
alkalemia is compensated by changes in renal function. The kidneys excrete more base; therefore, the urine becomes less acid.1

Severinghaus has suggested that cerebrospinal fluid pH has a role in regulating ventilation at high altitudes. When hypoxia is relieved as a result of breathing increased oxygen concentrations, ventilation decreases by small amounts. The PCO₂ elevates cerebrospinal fluid H⁺, making up most of the respiratory drive, so ventilation is only slightly depressed.6,7

High altitude residents

The naturally acclimatized, high altitude native has physical attributes which have increased the efficiency of the cardiorespiratory oxygen transport and utilization system. The native generally is lighter in weight and has a smaller body surface area with more muscle and less adipose tissue. He has a barrel chest with an increased total lung capacity and residual volume. Together, these qualities give him a high ratio of ventilatory capacity to body mass.4,5

The naturally acclimatized individual may also appear somewhat cyanotic and phethoric; and in some cases may have clubbed fingers.8 The heart, particularly the right heart, provides increased pulmonary arterial pressure to pump blood through greatly expanded pulmonary capillary beds.1

Tissues of the naturally acclimatized high altitude native can utilize oxygen with greater efficiency than the unacclimatized new resident. This is accomplished by a shift of the oxyhemoglobin dissociation curve to the right which allows the hemoglobin to release oxygen more readily. By virtue of the acclimatization process, the high altitude native acquires to some extent the appearance and biologic characteristics of an athlete.4,5

Acclimatization and maximal overall adaptation for the newcomer occur approximately after six months of residence. Upon returning to sea level, the individual loses these characteristics within 2-16 weeks. This time difference depends upon the length of time spent and how he acclimatized to the higher altitude.9 The new resident will never acquire the degree of acclimatization that the native has.

In general, acclimatization occurs by increasing the following: pulmonary ventilation, blood values, cardiovascular performance, the diffusing capacity of the lungs, the vascularity of the tissues, and the ability of the cells to utilize oxygen.

Pulmonary ventilation will improve with hypoxic stimulation of the chemoreceptors. If one remains at high altitudes for several days, ventilation will increase. With this increased ventilation, carbon dioxide is given up, reducing PCO₂ and increasing the pH of the body fluid. This tends to reduce the respiratory center stimulation. In several days, however, this inhibition fades and ventilation increases again.

The cause of this fading inhibition is unknown. There is evidence that it results from reduced cerebrospinal fluid bicarbonate ion, and this ion in turn decreases the pH.5,6,7

Blood values are also increased during acclimatization. Hypoxia is said to be the stimulus for increasing red blood cell production. The average increase in hemoglobin is approximately 22 gm%. The hematocrit may rise to an average of 60-65%.5

Blood volume increases as well. It may be as much as 20-30% above normal, which could cause the circulating hemoglobin to rise as much as 50-90% above normal. This increased blood volume increases the vascular bed which raises the number and size of capillaries. Therefore, blood comes into much closer contact with cells and is utilized to a greater extent.4,5 These altered blood values require months to develop.

As the blood volume rises during acclimatization, so does the diffusing capacity. A rise in pulmonary capillary blood and lung volume increases the pulmonary arterial pressure and decreases systemic blood pressure with chronic hypoxia.5

Diffusing capacity may have a three-fold increase at high altitudes. These altered pressure relationships may account for a high incidence of patent ductus arteriosus in babies born under high altitude conditions. Statistics show the malfunction is more apt to occur at high altitudes than at sea level.4,6

Circulatory and cardiac changes which occur in acclimatization include a 20-30% rapid increase in cardiac output, followed by a gradual decline to normal. The decline usually takes several days. Blood flow through the skin and kidneys decreases. At the same time, there is an increase in circulation in the skeletal muscle, heart and brain.8

Along with increased cardiac output with acclimatization, there is a tachycardia. With adaptation, there is a bradycardia, mild hypotension, and a slight increase in venous pressure.8,4

An increase in the vascularity of the tissues and the increased ability of the cells to utilize oxygen, is explained by Guyton.

As mentioned earlier, hypoxic tissue increases the number and size of capillaries, which in turn causes the blood to come into much closer contact with the cells. Based on a study of animals, it has been shown that the cells of certain oxidative
enzyme systems and the mitochondria are increased at higher altitudes.

**Anesthetic management**

Anesthetic considerations for preoperative preparations in high altitudes must include the depressant effects of premedicating drugs. If heavy sedation with hypnotics, narcotics or other drugs is necessary, oxygen should be started at the time the preoperative medication is given. To increase oxygen levels, nasal oxygen at 3-4 L/min gives an approximate oxygen concentration of 24-27%. Mask oxygen at 5-6 L/min gives an approximate oxygen concentration of 40-50%.

When opiates are used, cyanosis and tachycardia have been observed. Opiates suppress pulmonary ventilation, which is the most important compensation for the low oxygen tension. Post-anesthetic headache and nausea are common whenever pulmonary ventilation is suppressed.

The anesthetist may observe an increase in bleeding for patients living at increased altitudes. Hemorrhage is a common complication. This is most likely due to the polycythemia and longer coagulation times. The increased blood volume, venous pressure vasodilation, and number of capillaries may explain why there is an increase in bleeding and longer coagulation times. More study is needed in this area of increased oozing.

It has been recommended that preoperative blood-letting (phlebotomy) be done on patients with hemoglobin values above those considered normal at a particular altitude. These values are not firmly established in the current literature.

Prolonged circulation times occur at high altitudes, which may slow drug action. Animal studies not only illustrate decreased drug action, they also show an increase in liver metabolism. This decrease in drug action and liver metabolism is thought to be caused by stress produced by exposure to altitude and hypoxia which may change blood pH and so change metabolism. A survey of the literature records evidence that altitude produces changes in body fluid compartments which may change the distribution of a drug.

With increased altitudes, anesthetic agents, gases, vapors, and oxygen should be given in higher concentrations to maintain arterial partial pressures for anesthesia. Increased oxygen concentrations should also be provided with intravenous barbiturate anesthesia. It has been suggested by Mitten, that patients be pre-oxygenated for 3-5 min with 100% oxygen before induction, in high altitude conditions.

Higher concentrations of oxygen are necessary because anesthesia deprives compensatory hyperventilation of the native resident. Patients who have a lowered arterial P02 may develop hypoxia more rapidly with airway complications; residents at increased altitudes poorly compensate metabolic acidosis following hypotension or hemorrhage because buffer capacity is reduced.

Increasing the inspired oxygen concentration with nitrous oxide decreases the amnestic and anesthetic effects of the anesthetic. In this situation, nitrous oxide is unable to achieve anesthetic partial pressures without lowering the oxygen tension to a dangerous hypoxic level. Because of decreased barometric pressures, low partial pressures interfere with nitrous oxide uptake.

In Denver (5,280 feet), it is recommended that nitrous oxide be supplemented using a balanced anesthetic technique. The anesthetist must remember that increased concentrations of this less potent anesthetic agent may decrease oxygen tensions to hypoxic levels. Therefore, the technique of oxygen and nitrous oxide anesthesia should be avoided in higher altitudes.

Halothane and cyclopropane are satisfactory agents for use in higher altitudes. They permit the use of higher concentrations of oxygen and provide rapid induction with a rapid recovery. Assisted or controlled ventilation is required even with these agents to prevent muscle fatigue and avoid hypoxia.

The disadvantages of halothane are possible bradycardia and hypotension. Cyclopropane has the disadvantage of being an explosive agent and it also increases bleeding because of vasodilatation. Ethrane may now be replacing cyclopropane in higher altitudes; however, more documentation is needed to establish its use.

Spinal anesthesia is avoided whenever possible at the higher altitudes. The incidence of postspinal headache is very high, but it is not known why this condition exists. Possibly, it may result from a decrease in alveolar ventilation and hypoxia, or from poor hydration. The use of spinal anesthesia and its resulting side effects must be weighed against the possible benefits.

It has been observed also that local anesthetics have a shorter duration in the higher altitudes. The reasons for this pharmacologic phenomenon are also unknown.

With the use of muscle relaxants, deep levels of anesthesia are avoided, therefore, the risk of hypoxia and acidosis is lessened. Long-acting muscle relaxants should be used with caution to avoid prolonged muscular weakness and because specific information is not available on the use of reversal agents at higher altitudes.
Altitude may also affect anesthetic equipment. Flow meters, calibrated at sea level, deliver a higher flow than indicated due to the reduced density of gases. The error for all anesthetic gases is calculated at approximately 1%/1,000 feet up to 50,000 feet. In Denver (5,280 feet), vaporizers deliver approximately 10% more agent than the flow-meter indicates. Vaporizers function more efficiently at lower barometric pressures because the boiling point of volatile anesthetics is decreased; the vapor pressure constitutes a higher percentage of the barometric pressure at the same temperature. Therefore, vaporization is easier and concentrations will be greater.

Environmental conditions may be such that volatile agents may boil inside vaporizers in a warm room. At 15,000 feet, the boiling point of halothane is 95°F. This in-circuit vapor, in the Vernitrol vaporizer or copper kettle, may be delivered to the patient even if the oxygen flow-meter through the vaporizer is turned off. Halothane is vaporized by the fact of its boiling at 95°F at 15,000 feet without oxygen vaporization. It is carried by the inflow of fresh gases by the flow-meter.

Postoperatively, it is recommended that oxygen be given for a minimum of one hour. All patients in increased altitudes require pre- and post-operative oxygen therapy. If inadequate ventilation is noted during the postoperative period, controlled or assisted ventilation should be continued until the patient is alert and muscle power is recovered.

Conclusion

In summary, the anesthetist working at increased altitudes must consider the physical and physiological effects of low barometric pressures, decreased oxygen tensions, physiological changes of the acclimatized and unacclimatized resident. Also to be considered are the changes that occur with volatile agents, adjunct drugs and anesthetic equipment.

The anesthetist should avoid drugs which have a prolonged depressing effect on the respiratory center. A smooth, rapid induction with tracheal intubation is recommended. Agents such as halothane, enflurane and cyclopropane are used primarily because they allow increased oxygen concentrations. Cyclopropane is not used as freely as it once was because of its explosive properties.

Short acting muscle relaxants are used to control alveolar ventilation, and thus avoid hypoxia. Spinal anesthesia is thought to decrease alveolar ventilation, resulting in hypoxia. This decrease is thought to increase the incidence of postspinal headache, but this has not been proven.

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