Respiratory Adjustments During Exercise and at High Altitudes

Effects of Exercise

Respiratory adjustments during exercise are geared to both the intensity and duration of the exercise. Working muscles consume tremendous amounts of oxygen and evolve large amounts of carbon dioxide; thus, ventilation can increase 10- to 20-fold during vigorous exercise. Breathing becomes deeper and more vigorous, but the respiratory rate may not be significantly changed. This breathing pattern is called **hyperpnea** (hi"perp-ne'ah) to distinguish it from the deep and often rapid pattern of hyperventilation. Also, the respiratory changes seen in hyperpnea match metabolic demands and so do not lead to significant changes in the O_2 or CO_2 content of the blood. By contrast, hyperventilation may provoke excessive ventilation, resulting in a low P_{CO_2} and alkalosis.

This exercise-enhanced ventilation does *not* appear to be prompted by rising P_{CO_2} blood levels and declining P_{O_2} and pH for two reasons. First, ventilation increases abruptly at or just prior to the onset of exercise, followed by a more gradual increase, and finally a steady state of ventilation. Similarly, ventilation declines suddenly when exercise is stopped, followed by a more gradual decrease to pre-exercise values. Second, while venous levels change, arterial P_{CO_2} and P_{O_2} levels remain surprisingly constant during exercise. In fact, P_{CO_2} levels may decline below normal arterial levels, and P_{O_2} levels may rise very slightly because of the efficiency of the respiratory adjustments. Our present understanding of the mechanisms that produce these observations is sketchy, but the most accepted explanation is as follows.

The abrupt increase in ventilation that occurs as exercise is initiated reflects the interaction of the following neural factors: (1) psychic stimuli (that is, our conscious anticipation of exercise), (2) simultaneous cortical motor activation of the skeletal muscles and of the respiratory centers, and (3) proprioceptors in moving muscles, tendons, and joints, which send excitatory impulses to the respiratory centers. The subsequent gradual increase and then plateauing of respiration is probably due to the central stimulatory effects of rising body temperature, and of epinephrine secreted by the adrenal medulla during sympathetic nervous system activation.

The abrupt decrease in ventilation that occurs as exercise ends reflects the "shutting off" of the neural control mechanisms. The subsequent gradual decline in ventilation to baseline levels occurs as the oxygen debt is being repaid and respiration continues to be stimulated by low arterial pH due to lactic acid accumulation during rigorous exercise. This rise in lactic acid levels is *not* a result of inadequate respiratory function, because alveolar ventilation and pulmonary perfusion are as well matched during exercise as during rest. Rather, it reflects cardiac output limitations or inability of the skeletal muscles to further increase their oxygen consumption (see Chapter 9). In light of this fact, the practice of inhaling pure oxygen by mask, commonly used by football players to replenish their "oxygen-starved" bodies as quickly as possible, is useless. The panting athlete *does* have an oxygen deficit. But extra oxygen will not help, because the shortage is in the muscles—not in the lungs. ■

Effects of High Altitude

Most Americans live between sea level and an altitude of approximately 2400 meters (8000 feet). The differences in barometric pressure within this range are not great enough to cause healthy people any problems when they spend brief periods in the higher-altitude areas. However, when you move (on a long-term basis) from a sea level region to the mountains, where air density and P_{O_2} are lower, your body must make several respiratory and hemopoietic adjustments. This multistep adaptive response of the body is called **acclimatization.**

As already explained, decreases in arterial P_{O_2} cause the central chemoreceptors to become more responsive to increases in P_{CO_2}, and a substantial decline in P_{O_2} directly stimulates the peripheral chemoreceptors. As a result, increased ventilation occurs as the brain attempts to restore gas exchange to previous levels. Within a few days, the minute respiratory volume becomes stabilized at a level 2 to 3 L/min higher than that seen at sea level. Since increased ventilation also reduces arterial carbon dioxide levels, the P_{CO_2} of individuals living at high altitudes is typically below 40 mm Hg (its value at sea level).

Because less oxygen is available to be loaded, high-altitude conditions always result in lower than normal hemoglobin saturation levels. For example, at about 6000 m (19,000 feet) above sea level, the oxygen saturation of arterial blood is only 67% (compared to nearly 98% at sea level). But hemoglobin unloads only 20% to 25% of its oxygen at sea level pressures; thus, even at these reduced hemoglobin saturations, oxygen needs of the tissues are still met adequately under resting conditions. As partial compensation for declining hemoglobin saturations at high altitudes, hemoglobin's affinity for oxygen is also reduced, and more oxygen is unloaded to the tissues during each circulatory round.

Although the tissues receive adequate oxygen under normal conditions, problems arise when all-out efforts are demanded of the cardiovascular and respiratory systems (as discovered by U.S. athletes competing in Olympic events on the high mesa of Mexico City). Unless one is fully acclimatized, such conditions almost guarantee that body tissues will become severely hypoxic. ■

When blood oxygen tension declines, the kidneys intervene by accelerating the production of erythropoietin, which stimulates bone marrow production of red blood cells (see Chapter 18). This phase of acclimatization, which occurs slowly, provides long-term compensation for living in high-altitude areas.