8. Exercise and hypoxia

Exercise Affects Ventilation

With moderate physical activity, both oxygen consumption and carbon dioxide production increase. Minute ventilation can increase up to 25-fold. It would seem logical to guess that the increase in CO_2 production increases $PaCO_2$ which would, in turn, stimulate ventilation. However, measurements of $PaCO_2$ during moderate exercise show that it does not change appreciably. In fact, neither does PaO_2 nor pH. However, at near maximal exercise, arterial H+ concentration does rise and $PaCO_2$ falls. Why does the H+ concentration increase while $PaCO_2$ decreases?

The mediators that cause increased ventilation in response to moderate exercise are likely to include increased body temperature, increased epinephrine level, reflex input from the mechanoreceptors of the joints and muscles, and conditioned behavior (feed forward).

The deep breathing after exercise removes the “Oxygen Debt” restoring oxygen storing molecule (myoglobin) and energy storing (creatine phosphate) in the muscles, as well as removing lactic acid and H+.

HYPOXIA (LOW PO_2) & VENTILATORY CONTROL

Hypoxia is defined as a deficiency of oxygen at the tissue level. There are many causes of hypoxia but they can be grouped into four classes.

1. ***hypoxia- hypoxia*** in which arterial PO_2 is reduced.
2. ***anemic hypoxia*** in which arterial PO_2 is normal but the content of O_2 is reduced because of inadequate numbers of red blood cells or incompetent Hb or competition of carbon monoxide for Hb.
3. ***ischemic hypoxia*** in which blood flow to the tissues is too low.
4. ***histotoxic hypoxia*** in which the O_2 content in the tissue is normal but the cell is unable to utilize it because a toxic agent (such as cyanide) interferes with oxidative metabolism.

Individuals who reside at high altitudes (where O_2 tension is reduced) or who have sleep apnea syndrome (that is, they stop breathing for prolonged periods during sleep) may have diminished hypoxia drive to breathe. This is due to the “resetting” of their chemoreceptors set point.

The effects of O_2 deprivation vary from individual to individual but most people who ascend rapidly to altitudes above 10,000 ft experience some degree of ***altitude sicknesss***. The symptoms of altitude sickness are headache, nausea, vomiting, fatigue and possible mental confusion. In severe cases, life threatening pulmonary edema can occur due to pulmonary hypertension. Over a course of a several days these symptoms will disappear due to acclimatization which includes increased hematocrit (more red blood cells), increased 2,3, DPG and a shift in the O_2 dissociation curve to the right to facilitate unloading in the tissues.

During sleep, breathing frequency and inspiratory flow rate are reduced and minute ventilation decreases. This is accompanied by a relaxation of the skeletal muscle tone throughout the body including those muscles associated with the larynx, pharynx and tongue. Relaxation of muscle tone in these areas can cause partial obstruction of the upper airways and **snoring**. However, in some individuals the airways are completely occluded which can lead to **sleep apnea**. In sleep apnea, respiration stops for long periods (30-60 sec) and $PaCO_2$ rises; the respiratory center is stimulated. The individual reacts by gasping and often awakens.