Immediate and Long Term Physiological Adjustment to Altitudes: A Review

***Bulus Yuyu Maiyanga, Bala Mohammed Shira & Bala Yunusa Tilde**
Aminu Saleh College of Education Azare,
Bauchi State,
Nigeria
*maiyangabulus@yahoo.com, balashira3425@gmail.com
balayunusatilde@gmail.com

Abstract
Physiological Adjustment to altitude takes place by a number of processes within the body. Although some of these adjustments takes place at biochemical level with immediate exposure while some after prolong exposure. The aim of this review is to provide a brief overview of immediate and long term physiological adjustment at altitudes to students, coaches and athletes. The review covers ventilation, body fluid alteration, hormonal changes, blood and bioenergetics, the immediate performance response to altitudes, acclimatization, pulmonary and vo_2 response to long term exposure to altitude. Some of the immediate and long term adjustment includes hyperventilation, increased heart rate, increase hemotocrit and decreased vo_2 max. The body has to make this adjustment to enable it meet the demand place on the body as a result of decreased in the air pressure and less oxygen availability in altitude.

Keywords: *immediate, long term, Adjustment, Altitude*

Introduction

Human subject themselves to the rigors of the mountain regions of the world for reasons other than pure survival. People travel to high altitudes for recreational pursuit such as skiing, mountaineering, camping and sightseeing while others for training of athletes and research purposes.

According to Rick (1995), altitude is defined on the following scale, high (8000 – 12000 feet (2438- 3658 meters) very high 12000 – 18000 feet (3658 – 5487 meters) and extremely high 18000+ feet (55oo + meters). Peter, Erik and Swenson (2013) opined that, traditionally 2500 meters has been used as the threshold for high altitude.

According to Fulco (1988) there has been large number of people participating either passively or actively on altitudes, it is important to understand the Physiological changes and problems encountered during treks into high mountainous regions. The immediate effects of lowered ambient pressure of oxygen ($P0_2$) pertaining to the human response and adaptations to prolong exposure are complex. Several system (i.e. cardiovascular, pulmonary, endocrine) react to the hypoxia associated with altitude exposure adding to the complexity, these systems rarely react in isolation but rather interact to allow the work of the individual to be accomplished in this type of environment (Wyatt, 2005).

Physiological adaption to high altitudes or acclimatization takes place by a number of processes within the body. Although some of these adaptations takes place at a biochemical level shortly after the initial exposure to altitudes, a number of response, including the hypoxic ventilator response and an increased in cardiac output are the most important during early acclimatization.

Physiological Adjustment to High Altitude

Immediately exposure to altitude places the body in an environment with reduced partial pressure of oxygen ($P0_2$). Because of this reduction ($P0_2$) as the body works there is a diminished supply of oxygen to the tissue thereby resulting in a condition of hypoxia. The hypoxic ventilatory response is an increase in depth and rate of inhabitation in response to an increased in the oxygen saturation of the blood. This is detected by the carotid body and the medulla effects to these changes. The increased level of ventilation, leads to an increase in blood PH, known as respiratory alkalosis and a reduction in the carbon dioxide content of the blood (Gumersall, 2012).

Hypoxia (low amount of oxygen) as it relate to acute exposure to altitude, the human body experiences a condition known as hypoxic hypoxia, which describes arterial blood oxygen (Klausen 1969). The response by the human body to reduce (PO_2) depends on the intensity of work of the individual, the altitude at which the individual is exposed and the fitness of the individual. Utilizing maximal oxygen capacity (Vo_2 max) as a bench mark for performance at altitude, evidence suggests that decrements upon acute exposure begin to occur above 700m. However, declines in VO_2 max up to 1500m (Robergs & Kateyian, 2003) added that, the variability up to 1500m max reveal a concept at acute exposure that is seen with acclimatization known as responders and non-responders. General changes upon acute exposure to altitude include increased resting and sub maximal heart rate, increased resting and sub maximal ventilation, increased blood pressure, increased catecholamine secretion and decreased VO_2 max. These changes results in vascular resistance lactate production and decreased work capacity (Brooks, Fahey, White & Baldwin 2000).

Pulmonary Adjustment

The human initial response to high altitude is an increase in ventilation. The number of breaths taken each minute, as well as the depth of each breath is increase, this increased ventilation causes the air deep within the lungs to contain a higher pressure of oxygen than it otherwise would have by increasing the pressure of oxygen within the lungs, more oxygen can defuse (filter) into the blood for transport to the tissues (Fulco, 1988).

There is an increase in resting and sub maximal ventilation upon arrival to altitude. This is accomplished through both an increase in rate and volume of breaths. Ascending to altitude stimulates ventilation as a result of carotid and aortic body sensitivity to reduced PO_2 in arterial blood ($Pa0_2$). This hyperventilation to increased PO_2 in the alveoli, ($Pa0_2$) and at the same time reduces the partial pressure of carbon dioxide ($PC0_2$) with an increase in ventilator drive, a reduced $PC0_2$ allows for reduced co_2 in the blood and thus a lowered hydrogen concentration in the blood (Fulco, 1988). Furthermore, in compensation, bicarbonate ($Hc0_3$) is gradually reduced through excretion from the kidneys (renal dieresis) during the first few days of exposure. This excretion is associated with a decrease in plasma volume and subsequent effects on the cardiovascular system. The increased ventilator drive also reduces total body water through lost of water vapor during respiration. This coupled with the above renal dieresis and increased evaporative cooling can lead to rapid dehydration upon immediate exposure to altitude (Robergs & Keteyian 2003).

Fulco (1988) stated that, increased in ventilation causes the air deep within the lungs to contain a higher pressure of oxygen than it otherwise would have. The increased pressure of oxygen within the lungs causes more oxygen to diffuse into the blood for transport to the tissue. The increase in ventilation also causes carbon dioxide to be blown off making the blood less acidic. Ascending to altitude stimulates ventilation as a result of carotid and aortic body sensitivity to reduced $P0_2$ in arterial blood ($Pa0_2$) and at the same time reduce the partial pressure of carbon dioxide ($Pa0_2$) with an increase in ventilator drive, a reduces $Pa0_2$ allows for reduced CO_2 in the blood and thus H+ concentration in the blood. In compensation,

bicarbonate (Hco_3^o) is gradually reduced through excretion from the kidney (Wyatt, 2005, Gumersall, 2012).

Body Fluid Alteration

At sea level under resting condition the volume of fluid inside the cells, between the cells, and in the blood vessels (plasma volume) remains relatively constant. However exposure to altitude quickly alters the normal distribution of body fluids. There is marked movement of fluids into cells from out-of the blood vessels and the spaces between the cells. The reduction in plasma volume occurs almost immediately upon exposure to altitudes above 3,000 meters (Gumersall, 2012). Wyatt (2005) is of the view that the increased ventilator drive also reduces the total body water through loss of water vapor during respiration. This coupled with renal dieresis increased evaporative cooling and can lead to rapid dehydration upon acute exposure to altitudes.

The change in fluid status especially the rapid flux that occurs within the first days of exposure to high altitude is thought to play a major role in the cause of some altitude induced medical problems as high altitudes cerebral edema and high altitude pulmonary edema. At altitudes greater than 4,300 meters, plasma volume may be reduced by 20 percent to 30 percent; the total volume of body fluid may or may not be altered, with long altitude exposure there is some reversion of fluid compartment to sea level values (Fulco, 1988).

Hormonal Changes

Changes in fluids status at altitudes can be caused by alteration in the concentration of hormones involved in altering a normal fluid and electrolyte (e.g. sodium & potassium) balance in the body like aldosterone and arginine vasopressin have an effect on the kidneys to reabsorb water to help limit catecholamine release upon acute exposure to reduce $P0_2$ Norepinephrine increase progressively during rest as exercise peaking inside of a week of exposure develop more (Wyatt, 2005).

Cardiovascular Adjustment

Increase in catecholamine release upon acute exposure to reduce $P0_2$ morepinephrine increases progressively during rest and exercise peaking inside of a week of exposure, the response is associated with increases seen with heart rate and blood pressure, catecholamine activity regulates stroke volume, peripheral vascular resistance and affects substrate utilization with exposure (Wyatt, 2005). Added that, Cardiac output is the amount of blood pumped by the heart and the volume of blood circulated within the body each minute. During the first couple of days at high altitude, cardiac output is increased at rest at any level of oxygen uptake, except during maximal exercise. After the first couple of days there is a progressive reduction in cardiac output. By the tenth day of continued exposure, cardiac output is at rest and at any level of oxygen uptake is actually less than that found at sea level. The reduction in cardiac output seems primarily to be due to a reduction ion, stroke volume, which in turn is due to the reduction in plasma volume for the rest of the altitude sojourn, the reduced level of cardiac output is stabilized (Fulco 1988).

Resting and sub maximal cardiac output increase upon immediate exposure to altitude. As compensation for reduced $P0_2$ and tissue hypoxia, an increase in cardiac output is provided primarily by an increase in heart rate (bpm). Blood flow increase at altitude seen to be in response to arterial deactivation (Mc Ardle, Katch, Kate, 2001). Furthermore, an increase in heart rate (HR) is also in response to an increased peripheral resistance allowing for a decrease in stroke volume, this is to maintain a prescribed heart rate increases, in addition, the increase catecholamine response also further increase HR. Wyatt (2005) added that, Utilizing hypobaric chambers with simulated altitudes between 4000m and 800m indicated an increase

in heart rate despite reduced blood volume and reduced ventricular filling, the increase in heart rate seems to confirm the heart rate is responsible for the adjustment and the increase in heart rate is as a result of sympathetic activity resulting from increased blood norepinephrine concentration. Hahn and Gore (2001) says that, Increase in heart rate as a result of increase in altitude exposure has coincided with increased level of norepinephrine within a few days of exposure declines. This is partially due to increased parasympathetic influence and decrease responsiveness to catecholamine with prolong exposure. The reduced stroke volume could be due to reduced plasma volume but there are reports of diminished myocardial contractility occurring 2 to 8 days after exposure. One possible explanation for reduced heart rate after exposure is the increase in arteriovenous oxygen difference (a-vo_2 diff). Young, Andrew, Reeves and John (2002) added that, the percentage of saturation of hemoglobin with oxygen determines the content of oxygen in our blood, after 2100 meters above sea level, the saturation of oxyhaemoglobin begins to decrease. Upon exposure to lowered $P0_2$ and subsequent tissue hypoxia, there are many hemoglobin (Hb) concentration and hematocrit which have been shown to increase within 24 hours of exposure to attitude. The stimulation of red blood cell (RBC) production occurs; $P0_2$ sensitive cells within the kidney stimulate the release of erythropoietin (Wyatt 2005). With initial exposure to high altitude the hematocrit and hemoglobin concentrates are increased due to the reduction in red blood cell production, this result to increases in the hematocrit capacity of the blood (Fulco, 1988). Frisancho (1979) opined that, exposure to high altitude increased production of red blood cells that exceeds the destruction, hemoglobin concentration increase, increased in red blood volume and decrease in plasma volume. The total blood volume is increased minimally, subsequently, the hematocrit increases. Wyatt (2005) also reported that, altitude increases levels of red blood cells, promote oxygen unloading at the muscle and theoretically increased oxygen utilization.

Bioenergetics Adjustment

Brooks, et al, (2000) indicated that hypoglycemia and reduced liver glycogen content are common with acute altitude exposure. Hahn and George, (2001) in their study of trained cyclists exercising at 2300m there was a reduction in activity of phosphor fructokinase, a glycotic rate-limiting enzyme. This indicates that those training at altitude may have reduced glycotic reliance over time. However, in this same study the reduced muscle-glycogen depletion over time was associated with a concomitant increase in blood glucose dependence for fuel. The increase in glucose metabolic clearance rate is related to increase arterial norepinephrine levels and activation of the sympathetic nervous system.

Immediate Performance Response to Altitude

The consensus is that upon acute exposure to altitude performance decline with endurance performance, the standard measure to determine this decline has been $V0_2$ max. For most, oxygen consumption begins to decline at approximately 1500m with a subsequent rate of decline of 3% per 300m (Brooks, et al, 2000). Wyatt (2005) indicated that, short power output is not compromised at altitude with more prolonged high intensity work there is increased reliance on glycolysis and increased formation of lactate, there is also an increased blood acidosis resulting from diminished blood bicarbonate,

this allows for earlier onset of fatigue upon immediate exposure, the general trend in performance with altitude steady decline with increased distance. McAdrle, et al, 2001, opined that, in less than two minutes in length, the performance difference between sea levels and altitude is negligible, as from 2 to 5 minutes performance time increase at altitude up to 155% of sea level time at 400m. Performance from 20 to 30 minutes in length shows a near linear increase at altitude of 100% of sea level at 100m to over 155% of sea level performance at 4000m, the time increase is even more dramatic with performance over 2hr in

length, at 4000m the increase in time compared to sea level is around 125%. Yet at least under 300m of this performance time is increased to over 125% at a comparative sea level time overall, the threshold for performance decline seems to begin at approximately 1600m for events of 2 to 5 minutes and at 600-700 with events over 20 minutes (Brooks, et al, 2000).

Acclimatization at High Altitude

Full acclimatization and the time for this to occur is a controversial area. While some reports with "responders" indicate a 12-14 day period up at an altitude of 2300m, others note this process within the process in acclimatization several systems indicate tolerance and work ability are enhance (Wyatt 2005).The human body can adapt to high altitude through immediate and long term acclimatization. At high altitudes, in short term, lack of oxygen is sensed by carotid bodies which cause an increase in the breathing rate, which in turn causes adverse effect of respiratory alkalosis inhibiting the respiratory centre in ability to increase breathing caused by inadequate carotid body response, (Young, et al,2002).

Full acclimatization, however requires days or even weeks, gradually the body compensates for respiratory alkalosis by renal excretion of bicarbonate, allowing adequate respiration to provide oxygen without risking alkalosis can be enhanced by drugs such as acetazolamide. Eventually his body has lower lactate production, decreased plasma volume, increased hematocrit, increased Red Blood Cell (RBC) mass, higher concentration of capillaries in skeletal muscle tissue, more use of myoglobin, increased mitochondria, increased aerobic enzyme concentration, increased hypoxic pulmonary artery vasoconstriction and right ventricular hypertrophy, pulmonary artery pressure increases in an effort to oxygenate more blood (Young, et al, 2002).

Cardiovascular Response to Long Term Exposure to Altitude

With acclimatization there is a reduction in resting and sub maximal heart rate indicating a return to normal homeostasis within systems, cardiac contractility does not seem to be affected, yet stroke volume will diminish because of reduced cardiac filling pressure (Wyatt, 2005). The ratio pressure product (heart rate & systolic blood pressure) has been shown to increase to nearly 100% of that shown at sea level with acclimatization, mean arterial pressure increases due to systematic vascular resistance, increase catecholamine secretion of given work load and increased blood viscosity resulting from increased hematocrit (Young, et al, 2002). Furthermore, the myocardial response during vigorous work at altitude is brought on by a combination of decreased platform volume, increased total peripheral resistance and an increase in parasympathetic tone decreasing maximal heart rate. While it seems that maximal heart rate and maximal output decreases along with muscle blood flows with long altitude exposure at 4300 meter, VO_2max values increase after acclimatization.

Pulmonary and VO_2 Response to Long Term Exposure to Altitude

Long term exposure to altitude will increase pulmonary blood pressure allowing for improved pulmonary perfusion, ventilation elevate with acclimatization and increased chemoreceptor sensitively to get blood gas changes, dieresis and excretion of bicarbonate, hyperventilation allows for normalization of alveolar and arterial oxygen pressures and reduced pco_2 levels for acid base balance (Brooks, et al, 2000). Improvements in VO_2 max with acclimatization are due in part to combined hematological and muscular adaptations that allows for increase oxygen transport and utilization (Wyatt 2005).

Hematological and Muscular Adaptation to Long Term Exposure to Altitude

The increased in hemoglobin and red blood cell,with the increase in RBC, the oxygen dissociation curve shifts to the right indicating a facilitated release of oxygen to the working

tissues, facilitating the oxyhemoglobin dissociation shift is a decreased bicarbonate in the cerebrospinal fluid and excretion of bicarbonate by the kidney with acclimatization, there is also an increase in skeletal muscle vascularity and tissue myoglobin to provide improved oxygen transport and cellular oxygen transport (Wyatt, 2005).

Bioenergetics and Performance Adjustment to Long Term Exposure to Altitude

With prolonged exposure to altitude the reliance on muscular glycogen store is reduced with increase in blood glucose utilization during maximal with long exposure measures, appears to be associated with increased control of ATP & ADP ratios (Wyatt, 2005). It has been reported that with prolonged exposure, there is a drop in circulating blood lactate levels during period of exertion, current explanation indicated that after acclimatization, there is an increase in lactate uptake by active and inactive skeletal muscles, heart, kidney and liver (Brooks, et al, 2000).

Wyatt (2005) reported that there is weight loss and associated changed in body composition, both losses in lean tissues and body fat has reduced appetite experienced at increasing altitude as well as reported elevation of basal metabolic rates. For any given workload, the level of exertion is increased as partial pressure of oxygen is reduced this is reflected in the decline in maximal oxygen consumption (VO_2 max) seen with increasing altitude, this decline is in comparison to sea level performance, but when comparing immediate values to acclimatization values performance does improved. This is believed to be in response to increase in total red blood cells and VO_2 max (Wyatt, 2005).

Reduced ability of the central nervous system and cardiovascular system in hypoxic environments reduced the level of work the body can reach for extended periods of time; with prolonged exposure to altitude there is weight loss and associated changes in the body composition, both losses in lean tissue and body fat have been reported elevation of basal metabolic rates, fat catabolism may increase as well as glucaneogenesis if diet is inadequate (McArdle, et al, 2001). In addition, for any given workload, the level of exertion is increased as the partial pressure of oxygen is reduced, this is reflected in the decline in maximal oxygen consumption (VO_2 max) even with increasing altitude this decline may begin as low as 589m above sea level with a steady decline at a rate of 7% to 9% for each increase in elevation of 1000m beyond 6300m there seems to be curvilinear drop in Vo_2 max with average being one end half of sea level values at approximately 700m. Because of the decline in VO_2 max, performance based on oxygen utilization is reduced, the decline is in comparison to sea level performance, when comparing acute values to acclimatized values performance does improve, this is believed to be response to increase in total blood cells and Vo_2 max (Wyatt 2005).In general, even after acclimatization to each altitude the greater the distance to be covered in performance the greater the time to achieve that distance as altitude is increased.
The reduced plasma volume combined with increases in hemotocrit would increase blood viscosity and reduced oxygen transport capabilities, the reported decreases in bicarbonate (Hco_3) would result in decreased lactate efflux to the blood and subsequent decrease in muscle tissue pit. This of course leads to an earlier onset of fatigue (Brooks, et al, 2000). Lastly, the hyper ventilator response at altitude adversely affects performance in that the increased work of breathing could lead to an earlier on set of fatigue.

Conclusion

It is evident that the body positively responds to lowered oxygen pressures, but there are controversies over these responses in relation to the level of altitude, time of exposure and intensity of work.

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