ADAPTATION TO EXERCISE

INTRODUCTION

- Exercise training is an adaptive process. The body will adapt to the stress of exercise with increased fitness if the stress is above minimum threshold intensity. To achieve maximum effectiveness, we must consider factors involved in the adaptation of muscle to stress and deconditioning. These factors include overload, specificity, reversibility, and individual differences.

METABOLISM AND ENERGY

- **Metabolism** is the sum total of all chemical reactions in maintaining the living state of the cells, and thus the organism. In general metabolism may be divided into two categories, i.e. catabolism and anabolism.

OXYGEN CONSUMPTION IN NORMAL TRAINED MEN AND WOMEN

- Jo Smith and Lars Mc Naughton investigated the effects of intensity of exercise on excess post exercise oxygen consumption (EPOC) in eight trained men and eight trained women. Three exercise intensities were employed i.e. 40%, 50% and 70% of the predetermined maximal oxygen consumption (*V*O2max). The men had significantly higher resting *V*O2 values being 0.31 l/min than did the women, 0.26 l/min.

- The results indicated that, there were highly significant EPOC for both the men and the women during the 3 hours post exercise and these were dependent upon the exercise intensity employed. The duration of EPOC differed between the men and women but increased with exercise intensity: for the men 40%-31.2 min; 50%-42.1 min and 70%-47.6 min and for the women, 40%-26.9 min; 50%-35.6 min and 70%-39.1 min. The highest EPOC in terms of both time and energy utilized was at 70% *V*O2max.

- These findings indicated that men and women can exercised at the same percentages of their *V*O2 max to achieve a maximal benefits in terms of energy expenditure. However, it also shows that a significant EPOC can be achieved at moderate to low exercise intensities.

Measuring *V*O2 (Oxygen Consumption)

- Oxygen consumption (*V*O2) can be measured during a graded exercise test in which exercise intensity is progressively increased while measuring ventilation, oxygen and carbon dioxide of the inhaled and exhaled air.

- *V*O2 max is reached when oxygen consumption remains at steady state despite an increase in workload. *V*O2 max varies considerably in the population with sex being a primary determining factor in this variability.
- The average young untrained male will have a *V*O2 max of approximately 3.5 l/min and 45 ml/kg/min. The average young untrained female will score a *V*O2 max of approximately 2.0 l/min and 38 ml/kg/min.

ATP Ultimate source of energy

- Several energy sources are available which can be use to power the production of ATP. Among which include; Creatine phosphate, fatty acid and carbohydrate.

Respiratory Exchange Ratio (RER)

- The ratio of the volume of carbon dioxide eliminated from the lungs per minute to the volume of oxygen taken into the lungs during the same time.
- RER=VCO2/VO2.
- During rest, the respiratory exchange ratio is usually equals the respiratory quotient (RQ), but during exercise an unstable state arises due to expired air containing carbon dioxide and oxygen derive from stores within the body and the RER value is not the same as the RQ value.

- In one breath, you normally breathe in more molecules of oxygen than you breathe out molecules of CO2. Measuring this ratio can be used for estimating the RQ, an indicator of which fuel (carbohydrate or fat) is being metabolized to supply the body with energy.

CHANGES IN MUSCLES FUNCTION

- **Fast and slow twitch muscle fibers**
- Muscle fiber types can be broken down into two main types: **slow twitch (type I)** muscle fibers and **fast twitch (type II)** muscle fibers. Fast twitch muscle fibers can further be categories into **type IIa** and **type IIb** fibers.

Lactate Accumulation and Exercise

- At rest the normal range of blood lactate is 0.5-2.2 mmol/L. It is thought that complete exhaustion occurs some where in the range of 20-25 mmol/L for most individuals although values greater than 30 mmoles per litre have been recorded.

- Blood lactate concentrations peak about 5mins after the cessation of intense exercise (assuming cessation is due to exhaustion from acidosis). The delay is attributed to the time required to buffer and transport lactic acid from the tissue to the blood. A return to pre-exercise levels of blood lactate usually occurs within an hour (light activity during the post exercise period has been shown to accelerate this clearance. Training can also increase the rate of lactate clearance in both aerobic and anaerobically trained athletes compared to untrained individuals.

Muscle Pain and Muscle Soreness after Exercise

- Delayed onset muscle soreness (DOMS) describe a phenomenon of muscle pain, muscle soreness or muscle stiffness that is felt 12-48hrs after exercise, particularly at the beginning of a new exercise program after a change in sports activities or after a dramatic increase in the duration or intensity of exercise.

- This muscle pain is a normal response to unusual exertion and is part of an adaptation process that leads to greater stamina and strength as the muscles recover and build (hypertrophy). The delayed soreness of DOMS is generally at its worst within the first two days following the activity and subsides over the next few days.

CIRCULATORY ADJUSTMENTS

- During exercise, blood flow to skeletal muscle may increase by as much as 20 times, and the transfer of oxygen from blood to muscle may increase three fold resulting in a 60-fold increase in oxygen utilization by the muscle. Active hyperemia is primarily responsible for increasing blood flow to muscle; the resulting decrease in peripheral resistance leads to an increase in cardiac output (CO) mediated by sympathetic nerves.

- At the same time there is a reduction in flow to the gut, kidney, and at high levels of exercise, the skin . Cardiac output can increase up to 10 times above the resting level owing to large increases in heart rate (HR) and small changes in stroke volume (SV). Much of the increase in cardiac output can be accounted for by a decrease in peripheral resistance to about 50% of the resting value and by an increase in venous return to the heart due to both the pumping action of skeletal muscles on veins and the increase in breathing rate associated with exercise.

- Increased sympathetic and decreased parasympathetic activity in nerves innervating the heart during exercise has the effect of increasing both HR and the force of contraction so as to maintain SV at a relatively constant level. In fact, despite the large increase in HR and the associated reduce time available for filling and emptying, SV still increases by 1.5 times during exercise in mammals.

- Exercise is associated with only small changes in average arterial blood pressure, pH and gas tension. The oscillation in Pco2 and Po2 with breathing are somewhat increase, as it is the arterial pressure pulse. The increase pressure pulse is damped to some extent by an increase in the elasticity of the arterial walls, which is due to a rise in circulating catecholamine.

RESPIRATORY ADJUSTMENTS

- Exercise increases O2 utilization, CO2 production, and metabolic acid production. CO increases to meet the higher demands of the tissues. Even though this increases the transit time for blood through the lung capillaries, O2 levels in blood living the lung remain in equilibrium with those in the alveolar gas because ventilation volume increases.

- This initial sudden increase in ventilation volume is followed by a more gradual rise until a steady state is obtained both for ventilation volume and O2 uptake.
- Several receptors system appears to be involved in the respiratory responses to exercise. Muscle contraction stimulates stretch, acceleration, and position mechanoreceptors in muscles joints and tendons.

- Activity in these receptors reflexly stimulates ventilation and this system probably causes the sudden changes in ventilation that occur at the beginning and end of a period of exercise. This increase in ventilation varies with the group of muscles being stimulated.
- In the absence of exercise, large changes in CO2 and O2 levels are required to produce equivalent changes in ventilation.

- The chemoreceptors in the aortic and carotid bodies and in the medulla are probably not directly involved in the respiratory response to exercise, because mean partial PO2 and PCO2 levels in arterial blood donot change much during exercise. However, the sensitivity of these receptors will increase during exercise so that relatively small changes in gas partial pressure can cause an increase in ventilation.

- In this regard , it is significant that catecholamines which are released in increased quantities during exercise increase the sensitivity of the medullary receptors to changes in CO_2. Threshold levels of CO_2 are required to drive ventilation during exercise as under resting conditions

THERMOREGULATORY ADJUSTMENTS

- Body temperature regulation is an important component of any exercise or training regimen. Human body temperature is closely regulated by a complex system of control mechanisms, keeping a fairly regular rhythm of temperatures ranging from a low of 36 degrees Celsius in the early morning to a high of 37.5 degrees Celsius in the late afternoon. During exercise, heat is being generated and is lost via body surfaces such as the skin and, to a lesser extent, the lungs.

- Heat is lost to the environment by four major methods: conduction, convection, radiation, and evaporation. When the body senses a rise in temperature, the thermal center of the hypothalamus triggers dilation of surface veins and increases sweating. Evaporation of sweat cools the skin, and dilating the surface blood vessels enhances the dissipation of heat. The external environment also plays a significant role in heat regulation; when ambient temperatures exceed body temperature, heat is absorbed into the body and vice-versa.

Hyperthermia

- Hyperthermia in its advanced state referred to as heat stroke or sunstroke**,** is an acute condition which occurs when the body produces or absorbs more heat than it can dissipate. It is usually caused by prolonged exposure to high temperatures. The heat-regulating mechanisms of the body eventually become overwhelmed and unable to effectively deal with the heat, causing the body temperature to rise uncontrollably.

- Hyperthermia is a medical emergency which requires immediate treatment. Hyperthermia can be created artificially by drugs or by medical devices. In these instances it may be used to treat cancer and other conditions. Malignant hyperthermia is a rare complication of some types of general anesthesia.

Benefits of hyperthermia

- The body protects its self from viruses, bacteria and other harmful substances through the use of numerous defense systems. One of this is fever. Fever raises the body's temperature above normal in an attempt to destroy invading organisms and sweat impurities out of the system. Fever is a highly effective and natural process of curing disease and restoring health, and has been recognized as such for thousands of years.

- Hyperthermia deliberately creates fever in the patient in order to utilize this natural healing process. Hyperthermia can be used in the treatment of upper and respiratory tract infections, bladder problems and urinary tract infections such as cystitis.
- Hyperthermia stimulates cellular activity and increases metabolism. Circulation, tissue rebuilding and healing are also increased. Injuries heal faster with heat. That is the reason why heating pad is used to help heal muscle aches and pains. In fact hyperthermia stimulates all body functions.

Chemotherapeutic benefits of hyperthermia

These benefits are;

a) Synergy with chemotherapeutic drugs and also with ionizing radiation.

b) Low host toxicity

c) Easy to control (heating precisions in the range of plus or minus 0.1 degree C and specific definable localized areas.

d) Low resistance (chemotherapeutic resistance and hyperthermia resistance)

- The potential for hyperthermia and chemotherapy in the treatment of specific human cancers, both disseminated and tumors are discussed with respect to current therapy and the possible benefits of hyperthermia treatment.

REFERENCES

- Bailey, D. and Davis, B. (1997). Physiological implications of altitudes training for endurance performance at sea level: a review. *British journal of sports medicine*, **31**:183-190
- Berglund, B. (1992). High altitude training: aspect of hematological adaptation. *International journal of sport medicine*, **14**:289-303.
- Boning, D. (1997). Altitude and hypoxia training. *International journal of sport medicine*, **18**:565-570
- Dick, F. (1992). Training at altitudes in practice. *International journal of sport medicine*, **13**:203-205.
- Howe, A. and Boden, B. (2007) Heat-Related Illness in Atheletes. *The American Journal of Sports Medicine*, 35(8): 1384-95.
- Epstein, Y., Moran, D.S., Shapiro, Y., Sohar, E. and Shemer, J. (i999) Exertional heat stroke: *a case series. Med,* **51**:240-243

THANKS FOR YOUR KIND ATTENTION