Benha University
Faculty of Science
Department of Zoology

Adaptation Physiology (318 Z)

Semester: second (2014/2015)
Level: third level
Sepc: Zoology & Chemistry
Date: 27/5/2015
Exam time: 1:00 hours

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Group (B): (1 hour, 24 marks)

1. Compare between the followings: (12 marks)

a) Skeletal muscle in feeding and fasting.

Skeletal muscle in feeding

A. Carbohydrate metabolism

1. Increased glucose transport: The transient increase in plasma glucose and insulin after a carbohydrate-rich meal leads to an increase in glucose transport into muscle cells by GLUT-4. Glucose is phosphorylated to glucose 6-phosphate by hexokinase, and metabolized to provide the energy needs of the cells.
2. Increased glycogen synthesis: The increased insulin to glucagon ratio and the availability of glucose 6-phosphate favor glycogensyn-thesis, particularly if glycogen stores have been depleted as a result of exercise.

B. Fat metabolism

Fatty acids are released from chylomicrons and VLDL by the action of lipoprotein lipase. However, fatty acids are of secondary importance as a fuel for muscle during the fed state, in which glucose is the primary source of energy.

C. Amino acid metabolism

1. Increased protein synthesis: A spurt in amino acid uptake and protein synthesis occurs in the absorptive period after ingestion of a meal containing protein. This synthesis replaces protein degraded since the previous meal.
2. Increased uptake of branched-chain amino acids: Muscle is the principal site for degradation of branched-chain amino acids because it contains the required transaminase. The branched-chain amino acids, leucine, isoleucine, and valine, escape metabolism by the liver, and are taken up by muscle, where they are used for protein and as sources of energy.

Skeletal muscle in fasting

A. Carbohydrate metabolism

Glucose transport into skeletal muscle cells via insulin-sensitive GLUT-4 proteins in the plasma membrane and subsequent glucose metabolism are depressed because of low levels of circulating insulin.

B. Lipid metabolism

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During the first 2 weeks of fasting, muscle uses fatty acids from adipose tissue and ketone bodies from the liver as fuels. After about 3 weeks of fasting, muscle decreases its use of ketone bodies and oxidizes fatty acids almost exclusively. This leads to a further increase in the already elevated level of circulating ketone bodies. [Note: The increased use of ketone bodies by the brain as a result of their increased concentration in the blood is correlated with the decreased use of these compounds by the muscle.]

C. Protein metabolism

During the first few days of fasting, there is a rapid breakdown of muscle protein, providing amino acids that are used by the liver gluconeogenesis. Because muscle does not have glucagon receptors, muscle proteolysis likely is initiated by the fall in insulin and sustained by the rise in glucocorticoids. [Note: Alanine and glutamine are quantitatively the most important gluconeogenic amino acids released from muscle. They are produced by the catabolism of branched-chain amino acids.] By several weeks of fasting, the rate of muscle proteolysis decreases paralleling a decline in the need for glucose as a fuel for the brain, which has begun using ketone bodies as a source of energy.

b) Acute and chronic mountain sickness.

Acute mountain sickness:

A small percentage of people who ascend rapidly to high altitudes become acutely sick and can die if not given oxygen or removed to a low altitude. Tness begins from a few hours up to about 2 days after ascent. Two events frequently occur:

1. Acute cerebral edema. This is believed to result from local vasodilation of the cerebral blood vessels, caused by the hypoxia. Dilation of the arterioles increases blood flow into the capillariesthus increasing capillary pressure, which in turn causes fluid to leak into the cerebral tissues.
2. Acute pulmonary edema. The severe hypoxia causes the pulmonary arterioles to constrict potently, but the constriction is much greater in some parts of the lungs than in other parts, so that more and more of the pulmonary blood flow is forced through fewer and fewer still unconstricted pulmonary vessels. The postulated result is that the capillary pressure in these areas of the lungs becomes especially high and local edema occurs.

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Chronic mountain sickness

Occasionally, a person who remains at high altitude too long develops chronic mountain sickness, in which the following effects occur:
(1) The red cell mass and hematocrit become exceptionally high.
(2) The pulmonary arterial pressure becomes elevated even more than the normal elevation that occurs during acclimatization.
(3) The right side of the heart becomes greatly enlarged.
(4) The peripheral arterial pressure begins to fall.
(5) Congestive heart failure ensues.
(6) Death often follows unless the person is removed to a lower altitude.

c) Using helium and nitrogen during very deep dives.

In very deep dives, especially during saturation diving, helium is usually used in the gas mixture instead of nitrogen for three reasons:
(1) It has only about one fifth the narcotic effect of nitrogen.
(2) Only about one half as much volume of helium dissolves in the body tissues as nitrogen, and the volume that does dissolve diffuses out of the tissues during decompression several times as rapidly as does nitrogen, thus reducing the problem of decompression sickness.
(3) The low density of helium (one seventh the density of nitrogen) keeps the airway resistance for breathing at a minimum, which is very important because highly compressed nitrogen is so dense that airway resistance can become extreme, sometimes making the work of breathing beyond endurance.

2. Explain the following: **(12 marks)**

a) Physiological problems of weightlessness.

Most of the problems that do occur are related to three effects of the weightlessness:
(1) Motion sickness during the first few days of travel, (2) translocation of fluids within the body because of failure of gravity to cause normal hydrostatic pressures, and (3) diminished physical activity

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because no strength of muscle contraction is required to oppose the force of gravity. Almost 50 per cent of astronauts experience motion sickness, with nausea and sometimes vomiting, during the first 2 to 5 days of space travel. This probably results from an unfamiliar pattern of motion signals arriving in the equilibrium centers of the brain, and at the same time lack of gravitational signals. The observed effects of prolonged stay in space are the following: (1) decrease in blood volume, (2) decrease in red blood cell mass, (3) decrease in muscle strength and work capacity, (4) decrease in maximum cardiac output, and (5) loss of calcium and phosphate from the bones, as well as loss of bone mass. Most of these same effects also occur in people who lie in bed for an extended period of time. For this reason, exercise programs are carried out by astronauts during prolonged space missions.

b) Natural acclimatization of native human being living at high altitudes.

Many of natives are born at these altitudes and live there all their lives. In all aspects of acclimatization, the natives are superior to even the best acclimatized lowlanders, even though the lowlanders might also have lived at high altitudes for 10 or more years. Acclimatization of the natives begins in infancy. The chest size, especially, is greatly increased, whereas the body size is somewhat decreased, giving a high ratio of ventilatory capacity to body mass. In addition, their hearts, which from birth onward pump extra amount of cardiac output, are considerably larger than the hearts of lowlanders. Delivery of oxygen by the blood to the tissues is also highly facilitated in these natives. The arterial oxygen Po2 in the natives at high altitude is only 40 mm Hg, but because of the greater quantity of hemoglobin, the quantity of oxygen in their arterial blood is greater than that in the blood of the natives at the lower altitude. Note also that the venous Po_2 in the high altitude natives is only 15 mm Hg less than the venous Po_2 for the lowlanders, despite the very low arterial Po_2, indicating that oxygen transport to the tissues is exceedingly effective in the naturally acclimatized high-altitude natives.

c) Increased pulmonary ventilation at high altitudes.

Immediate exposure to low Po_2 stimulates the arterial chemoreceptors, and this increases alveolar ventilation to a maximum of about 1.65 times normal.Therefore, compensation occurs within seconds for the

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high altitude, and it alone allows the person to rise several thousand feet higher than would be possible without the increased ventilation. Then, if the person remains at very high altitude for several days, the chemoreceptors increase ventilation still more, up to about five times normal.

The immediate increase in pulmonary ventilation on rising to a high altitude blows off large quantities of carbon dioxide, reducing the Pco_2 and increasing the pH of the body fluids. These changes inhibit the brain stem respiratory center and thereby oppose the effect of low PO_2 to stimulate respiration by way of the peripheral arterial chemoreceptors in the carotid and aortic bodies. But during the ensuing 2 to 5 days, this inhibition fades away, allowing the respiratory center to respond with full force to the peripheral chemoreceptor stimulus from hypoxia, and ventilation increases to about five times normal. The cause of this fading inhibition is believed to be mainly a reduction of bicarbonate ion concentration in the cerebrospinal fluid as well as in the brain tissues. This in turn decreases the pH in the fluids surrounding the chemosensitive neurons of the respiratory center, thus increasing the respiratory stimulatory activity of the center. An important mechanism for the gradual decrease in bicarbonate concentration is compensation by the kidneys for the respiratory alkalosis. The kidneys respond to decreased Pco_2 by reducing hydrogen ion secretion and increasing bicarbonate excretion. This metabolic compensation for the respiratory alkalosis gradually reduces plasma and cerebrospinal fluid bicarbonate concentration and pH toward normal and removes part of the inhibitory effect on respiration of low hydrogen ion concentration. Thus, the respiratory centers are much more responsive to the peripheral chemoreceptor stimulus caused by the hypoxia after the kidneys compensate for the alkalosis.

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