

www.biopac.com

Biopac Student Lab[®] Lesson 15 AEROBIC EXERCISE PHYSIOLOGY Introduction

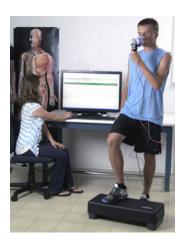
Rev. 01152013

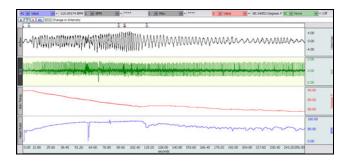
Richard Pflanzer, Ph.D.

Associate Professor Emeritus Indiana University School of Medicine Purdue University School of Science

William McMullen

Vice President, BIOPAC Systems, Inc.





I. INTRODUCTION

The ability to exercise depends on providing increased energy to the skeletal muscles for muscle contraction. Skeletal muscle contraction and relaxation requires chemical energy derived from adenosine triphosphate (ATP,) an energy-rich compound formed within the skeletal muscle fibers from the metabolism of foodstuff. Chemical energy is present in all kinds of food such as protein, carbohydrate, and fat; however, this energy cannot be directly used for contraction and relaxation. Instead, the muscle fiber must metabolize basic nutrient molecules and use some of the energy released during the metabolism to form ATP. The ATP molecules are then used as an energy source for contraction and relaxation. The process is summarized in Fig. 15.1.

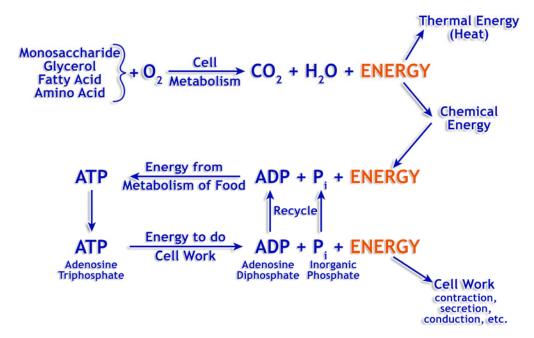


Fig. 15.1 ATP metabolism process

Exercise increases the demands for ATP. Skeletal muscle fibers store very little ATP so immediate and continued replenishment of ATP must occur if exercise is to continue. ATP can quickly be generated from muscle stores of creatine phosphate, another high-energy phosphate (Fig. 15.2). Although the energy in creatine phosphate cannot directly be used for contraction, it can be transferred with phosphate to ADP thereby replenishing ATP.

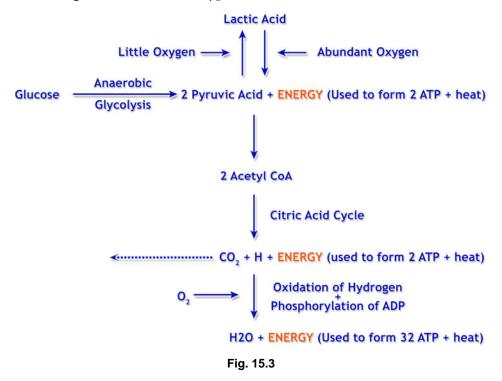
Page I-1 ©BIOPAC Systems, Inc.

$$\begin{array}{c} \text{Cr} \leftarrow \text{P}_{i} \longrightarrow \text{Cr} + \text{P}_{i} + \text{ENERGY} \\ \text{Creatine Phosphate} & \downarrow \\ \text{P}_{i} + \text{ENERGY} + \text{ADP} \longrightarrow \text{ATP} \\ \text{CH}_{2}\text{O} + \text{O}_{2} \xrightarrow{\text{Metabolism}} \text{CO}_{2} + \text{H}_{2}\text{O} + \text{ENERGY} \end{array}$$

Fig. 15.2

Dietary creatine supplementation can slightly increase the ability to perform short-term high intensity exercise; however, creatine phosphate levels normally are sufficient by themselves only for very brief periods of acute exercise (e.g., the first five seconds of running a 100-meter sprint). After the first few seconds, the remaining energy requirement for contraction and relaxation is provided by ATP generated during glycolysis (some) and oxidative phosphorylation (most).

Anaerobic glycolysis, a process that does not require oxygen, generates a small amount of ATP and hydrogen as glucose is metabolized to pyruvic acid (Fig. 15.3). In the presence of adequate oxygen, pyruvic acid is converted to acetyl CoA, which enters the citric acid cycle. Hydrogen produced before and during the conversion and during the citric acid cycle is oxidized to water, a process known as oxidative phosphorylation because in addition to requiring oxygen, ADP is phosphorylated resulting in the formation of a large amount of ATP. If oxygen is not adequately supplied to the exercising muscle, pyruvic acid is converted to lactic acid, a metabolite which enters the extracellular fluid increasing its acidity (lactic acidosis). Immediately after exercise, lactic acid is taken up by the muscle, converted back to pyruvic acid, and metabolized to form ATP through oxidative pathways. The additional amount of oxygen, above the amount required by resting muscle, to process the lactic acid produced during exercise is called the *oxygen debt*.



Glucose from intramuscular glycogen, a polymer of glucose, and circulating free fatty acids are the major exercise energy substrates during mild or moderate work; amino acid usage is very low at any work intensity. During mild or moderate exercise, fat is the primary energy source for contracting muscle. Increased sympathetic stimulation of adipose tissue during exercise accelerates the breakdown of fat, increasing circulating levels of glycerol and fatty acids. Fatty acid metabolism yields approximately twice as much ATP as does the metabolism of an equivalent quantity of monosaccharide or amino acid, and body fat stores are very large relative to the energy demands of even the most prolonged mild exercise. Consequently, mild exercise is not limited by depletion of energy substrate and can be continued almost indefinitely, with oxidative (aerobic) metabolism generating nearly all of the required ATP. Small amounts of ATP are also generated anaerobically from the conversion of glucose to pyruvate (glycolysis) and from the dephosphorylation of creatine phosphate.

During moderate to heavy dynamic exercise (work rate above 50% of the peak oxygen consumption,) adequate maintenance of ATP production to sustain the work rate also depends on the breakdown of glycogen (glycogenolysis) and metabolism of the glycogen-derived glucose. When intramuscular glycogen stores are depleted, exhaustion occurs. Thus, the capacity for sustained work at moderate to heavy intensities depends on the amount of stored glycogen, which varies from person to person, and which can be altered by prior activity and diet.

Sustained exercise at any level of intensity is oxygen dependent. The primary dependence on oxidative metabolism for ATP production allows steady-state exercise intensity to be measured in terms of oxygen consumption. During dynamic exercise, oxygen consumption increases with increasing intensity of the exercise until a plateau, called *peak oxygen consumption*, is reached. During rest. A normal adult consumes oxygen at a rate of about 250 mL per minute. During heavy exercise, an untrained person's oxygen consumption may increase twelve-fold to a peak of 3000 mL per minute. It is not uncommon for a highly trained athlete to have a peak oxygen consumption of 5000 mL per minute. Peak oxygen consumption during exercise is limited by the ability of the respiratory and the cardiovascular systems to deliver oxygen to skeletal muscle, and by the ability of the working skeletal muscle to utilize the provided oxygen.

Maximal oxygen uptake from the atmosphere to consumption by the muscle is limited by the maximum value of one or more of the following factors:

- 1. Pulmonary ventilation
- 2. Pulmonary diffusion
- 3. Cardiac output
- 4. Muscle blood flow
- 5. Oxygen use in the muscle fiber

Some of these factors, such as maximal cardiac output and maximal oxygen use in the muscle fiber can be increased through appropriate training.

Pulmonary ventilation increases linearly with work intensity during mild and moderate work, then more steeply in intense exercise. Pulmonary ventilation, or minute respiratory volume, is the volume of air moved into and out of the respiratory system in one minute. It is the product of tidal volume, which is the volume of air moved into and then back out of the respiratory system with each breath, and respiratory rate, which is the number of breaths per minute. During dynamic exercise, increases in both tidal volume and breathing frequency contribute to increasing ventilation. Increased ventilation maintains oxygen partial pressure and hemoglobin saturation unchanged in arterial blood in even the most intense exercise. In light to moderate dynamic exercise, increased ventilation also maintains a rate of carbon dioxide excretion that matches the increased rate of carbon dioxide production in active skeletal muscle, helping to keep blood pH within normal limits.

The cardiovascular responses to dynamic exercise include increased cardiac output, increased mean arterial pressure, increased skeletal muscle and coronary arterial blood flow, and decreased blood flow in the kidneys, skin, and abdominal viscera.

Dynamic exercise increases sympathetic neural activity and decreases parasympathetic neural activity. Increased sympathetic neural outflow increases heart rate, and increases cardiac contractility thereby increasing stroke volume. Cardiac output is the product of stroke volume, the volume of blood ejected per beat by the ventricle, and heart rate, the number of beats per minute. In dynamic exercise, an increase in stroke volume and an increase in heart rate increase cardiac output, promoting increased delivery of blood to active skeletal muscles.

Increased sympathetic neural outflow also vasoconstricts arterioles in skeletal muscle, kidneys, skin, and abdominal viscera thereby increasing mean arterial pressure, the prime force governing blood circulation. In active skeletal muscles, local chemical changes resulting from increased muscle metabolism override the sympathetic effects and cause vasodilation of the arterioles that supply the muscles. The vasodilation decreases local vascular resistance which, coupled with increased mean arterial pressure and peripheral vasoconstriction in other organs, permits enormous increases in skeletal muscle blood flow, facilitating oxygen delivery and carbon dioxide removal.

In moderate dynamic exercise oxygen delivery and removal of carbon dioxide and other metabolites by the blood is adequate to meet the metabolic demands of active skeletal muscle. However, as exercise intensity and duration increase above 50% peak oxygen consumption, active muscles consume more oxygen per minute than can be delivered to them by the blood. ADP and inorganic phosphate (P_i) concentrations in the muscle fibers increase as creatine phosphate levels fall and muscle fibers reach their limit of aerobically generating ATP. These conditions create an intense stimulation of glycolysis, the process of anaerobically generating ATP and pyruvate. When oxygen delivery to skeletal muscle is inadequate, pyruvate is converted to lactic acid and released into the blood. As the intensity of exercise increases, the amount of lactic acid released increases causing the blood pH to fall. This process, known as lactic acidosis, is common in intense exercise. At the end of a period of intense exercise and during the recovery period, muscle fibers remove lactate from the blood, convert it back into pyruvate, and oxidatively metabolize it generating and replenishing ATP. An additional amount of oxygen above and beyond the amount needed to maintain normal resting muscle metabolism is required to process the lactate-derived pyruvate.

The additional amount of oxygen referred to earlier as the oxygen debt accounts for the need to maintain elevated ventilation and cardiac output for a brief period after exercise.

Exercise increases the metabolic production of heat (Fig. 15.1) and the excess heat must be dissipated to the external environment. Core body temperature is controlled by balancing heat gain with heat loss. Heat loss mechanisms include convection, conduction, radiation, and evaporation. All involve varying amounts of heat loss by way of the skin surface or surfaces of the airways. The most effective way to remove heat is to use the heat to evaporate liquids into gas. Exercise increases sympathetic neural outflow thereby increasing sweat production. By excreting sweat, the heat produced during exercise and transported to the skin can be used to evaporate the sweat, removing heat, and cooling the blood.

In this lesson, we will observe and record changes in skin temperature associated with moderate levels of dynamic exercise. By recording changes in respiratory airflow and heart rate, we can gain an appreciation of some of the cardiovascular and respiratory adjustments that occur in moderate dynamic exercise.