CHAPTER 3

PHYSIOLOGIC RESPONSES AND LONG-TERM ADAPTATIONS TO EXERCISE

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CHAPTER 3

PHYSIOLOGIC RESPONSES AND LONG-TERM ADAPTATIONS TO EXERCISE

Introduction

When challenged with any physical task, the human body responds through a series of integrated changes in function that involve most, if not all, of its physiologic systems. Movement requires activation and control of the musculoskeletal system; the cardiovascular and respiratory systems provide the ability to sustain this movement over extended periods. When the body engages in exercise training several times a week or more frequently, each of these physiologic systems undergoes specific adaptations that increase the body's efficiency and capacity. The magnitude of these changes depends largely on the intensity and duration of the training sessions, the force or load used in training, and the body's initial level of fitness. Removal of the training stimulus, however, will result in loss of the efficiency and capacity that was gained through these training-induced adaptations; this loss is a process called detraining.

This chapter provides an overview of how the body responds to an episode of exercise and adapts to exercise training and detraining. The discussion focuses on aerobic or cardiorespiratory endurance exercise (e.g., walking, jogging, running, cycling, swimming, dancing, and in-line skating) and resistance exercise (e.g., strength-developing exercises). It does not address training for speed, agility, and flexibility. In discussing the multiple effects of exercise, this overview will orient the reader to the physiologic basis for the relationship of physical activity and health. Physiologic information pertinent to specific diseases is presented in the next chapter. For additional information, the reader is referred to the selected textbooks shown in the sidebar.

Selected Textbooks on Exercise Physiology

Åstrand PO, Rodahl K. *Textbook of work physiology*. 3rd edition. New York: McGraw-Hill Book Company, 1986.

Brooks GA, Fahey TD, White TP. *Exercise physiology: human bioenergetics and its applications*. 2nd edition. Mountain View, CA: Mayfield Publishing Company, 1996.

Fox E, Bowers R, Foss M. *The physiological basis for exercise and sport*. 5th edition. Madison, WI: Brown and Benchmark, 1993.

McArdle WD, Katch FI, Katch VL. *Essentials of exercise physiology*. Philadelphia, PA: Lea and Febiger, 1994.

Powers SK, Howley ET. *Exercise physiology: theory and application to fitness and performance*. Dubuque, IA: William C. Brown, 1990.

Wilmore JH, Costill DL. *Physiology of sport and exercise*. Champaign, IL: Human Kinetics, 1994.

Physiologic Responses to Episodes of Exercise

The body's physiologic responses to episodes of aerobic and resistance exercise occur in the musculoskeletal, cardiovascular, respiratory, endocrine, and immune systems. These responses have been studied in controlled laboratory settings, where exercise stress can be precisely regulated and physiologic responses carefully observed.

Cardiovascular and Respiratory Systems

The primary functions of the cardiovascular and respiratory systems are to provide the body with

oxygen (O_2) and nutrients, to rid the body of carbon dioxide (CO_2) and metabolic waste products, to maintain body temperature and acid-base balance, and to transport hormones from the endocrine glands to their target organs (Wilmore and Costill 1994). To be effective and efficient, the cardiovascular system should be able to respond to increased skeletal muscle activity. Low rates of work, such as walking at 4 kilometers per hour (2.5 miles per hour), place relatively small demands on the cardiovascular and respiratory systems. However, as the rate of muscular work increases, these two systems will eventually reach their maximum capacities and will no longer be able to meet the body's demands.

Cardiovascular Responses to Exercise

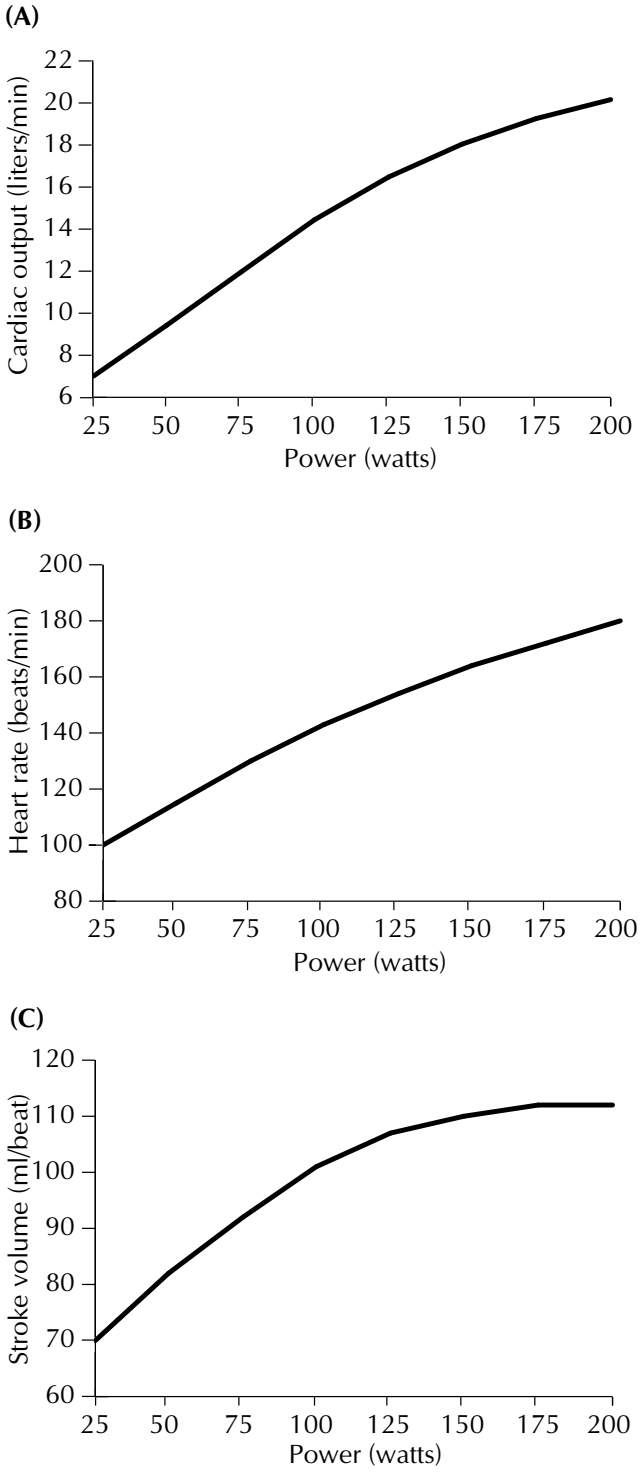
The cardiovascular system, composed of the heart, blood vessels, and blood, responds predictably to the increased demands of exercise. With few exceptions, the cardiovascular response to exercise is directly proportional to the skeletal muscle oxygen demands for any given rate of work, and oxygen uptake ($\dot{V}O_2$) increases linearly with increasing rates of work.

Cardiac Output

Cardiac output ($\dot{Q}$) is the total volume of blood pumped by the left ventricle of the heart per minute. It is the product of heart rate (HR, number of beats per minute) and stroke volume (SV, volume of blood pumped per beat). The arterial-mixed venous oxygen ($A\text{-}\bar{v}O_2$) difference is the difference between the oxygen content of the arterial and mixed venous blood. A person's maximum oxygen uptake ($\dot{V}O_2$ max) is a function of cardiac output ($\dot{Q}$) multiplied by the $A\text{-}\bar{v}O_2$ difference. Cardiac output thus plays an important role in meeting the oxygen demands for work. As the rate of work increases, the cardiac output increases in a nearly linear manner to meet the increasing oxygen demand, but only up to the point where it reaches its maximal capacity ($\dot{Q}$ max).

To visualize how cardiac output, heart rate, and stroke volume change with increasing rates of work, consider a person exercising on a cycle ergometer, starting at 50 watts and increasing 50 watts every 2 minutes up to a maximal rate of work (Figure 3-1 A, B, and C). In this scenario, cardiac output and heart rate increase over the entire range of work, whereas stroke volume only increases up to approximately 40

Figure 3-1. Changes in cardiac output (A), heart rate (B), and stroke volume (C) with increasing rates of work on the cycle ergometer



to 60 percent of the person's maximal oxygen uptake ($\dot{V}O_2$ max), after which it reaches a plateau. Recent studies have suggested that stroke volume in highly trained persons can continue to increase up to near maximal rates of work (Scruggs et al. 1991; Gledhill, Cox, Jamnik 1994).

Blood Flow

The pattern of blood flow changes dramatically when a person goes from resting to exercising. At rest, the skin and skeletal muscles receive about 20 percent of the cardiac output. During exercise, more blood is sent to the active skeletal muscles, and, as body temperature increases, more blood is sent to the skin. This process is accomplished both by the increase in cardiac output and by the redistribution of blood flow away from areas of low demand, such as the splanchnic organs. This process allows about 80 percent of the cardiac output to go to active skeletal muscles and skin at maximal rates of work (Rowell 1986). With exercise of longer duration, particularly in a hot and humid environment, progressively more of the cardiac output will be redistributed to the skin to counter the increasing body temperature, thus limiting both the amount going to skeletal muscle and the exercise endurance (Rowell 1986).

Blood Pressure

Mean arterial blood pressure increases in response to dynamic exercise, largely owing to an increase in systolic blood pressure, because diastolic blood pressure remains at near-resting levels. Systolic blood pressure increases linearly with increasing rates of work, reaching peak values of between 200 and 240 millimeters of mercury in normotensive persons. Because mean arterial pressure is equal to cardiac output times total peripheral resistance, the observed increase in mean arterial pressure results from an increase in cardiac output that outweighs a concomitant decrease in total peripheral resistance. This increase in mean arterial pressure is a normal and desirable response, the result of a resetting of the arterial baroreflex to a higher pressure. Without such a resetting, the body would experience severe arterial hypotension during intense activity (Rowell 1993). Hypertensive patients typically reach much higher systolic blood pressures for a given rate of work, and they can also experience increases in diastolic blood pressure. Thus, mean arterial pressure is generally much higher in these patients, likely owing to a lesser reduction in total peripheral resistance.

For the first 2 to 3 hours following exercise, blood pressure drops below preexercise resting levels, a phenomenon referred to as postexercise hypotension (Isea et al. 1994). The specific mechanisms underlying this response have not been established. The acute changes in blood pressure after an episode of exercise may be an important aspect of the role of physical activity in helping control blood pressure in hypertensive patients.

Oxygen Extraction

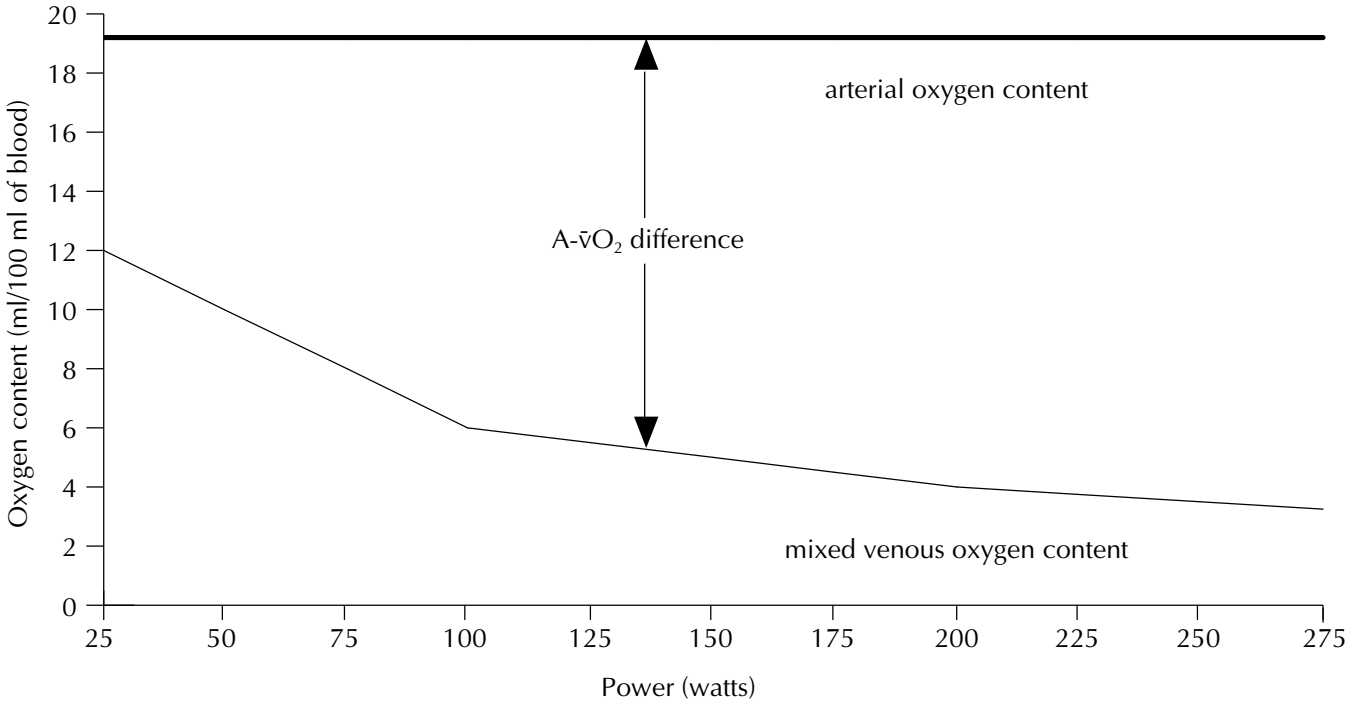
The A-$\bar{v}O_2$ difference increases with increasing rates of work (Figure 3-2) and results from increased oxygen extraction from arterial blood as it passes through exercising muscle. At rest, the A-$\bar{v}O_2$ difference is approximately 4 to 5 ml of O_2 for every 100 ml of blood (ml/100 ml); as the rate of work approaches maximal levels, the A-$\bar{v}O_2$ difference reaches 15 to 16 ml/100 ml of blood.

Coronary Circulation

The coronary arteries supply the myocardium with blood and nutrients. The right and left coronary arteries curve around the external surface of the heart, then branch and penetrate the myocardial muscle bed, dividing and subdividing like branches of a tree to form a dense vascular and capillary network to supply each myocardial muscle fiber. Generally one capillary supplies each myocardial fiber in adult humans and animals; however, evidence suggests that the capillary density of the ventricular myocardium can be increased by endurance exercise training.

At rest and during exercise, myocardial oxygen demand and coronary blood flow are closely linked. This coupling is necessary because the myocardium depends almost completely on aerobic metabolism and therefore requires a constant oxygen supply. Even at rest, the myocardium's oxygen use is high relative to the blood flow. About 70 to 80 percent of the oxygen is extracted from each unit of blood crossing the myocardial capillaries; by comparison, only about 25 percent is extracted from each unit crossing skeletal muscle at rest. In the healthy heart, a linear relationship exists between myocardial oxygen demands, consumption, and coronary blood flow, and adjustments are made on a beat-to-beat

Figure 3-2. Changes in arterial and mixed venous oxygen content with increasing rates of work on the cycle ergometer



basis. The three major determinants of myocardial oxygen consumption are heart rate, myocardial contractility, and wall stress (Marcus 1983; Jorgensen et al. 1977). Acute increases in arterial pressure increase left ventricular pressure and wall stress. As a result, the rate of myocardial metabolism increases, necessitating an increased coronary blood flow. A very high correlation exists between both myocardial oxygen consumption and coronary blood flow and the product of heart rate and systolic blood pressure (SBP) (Jorgensen et al. 1977). This so-called double product (HR • SBP) is generally used to estimate myocardial oxygen and coronary blood flow requirements. During vigorous exercise, all three major determinants of myocardial oxygen requirements increase above their resting levels.

The increase in coronary blood flow during exercise results from an increase in perfusion pressure of the coronary artery and from coronary vasodilation. Most important, an increase in sympathetic nervous system stimulation leads to an increase in circulating catecholamines. This response triggers metabolic processes that increase both perfusion pressure of the coronary artery and coronary vasodilation to meet the increased need for blood flow required by the increase in myocardial oxygen use.

Respiratory Responses to Exercise

The respiratory system also responds when challenged with the stress of exercise. Pulmonary ventilation increases almost immediately, largely through stimulation of the respiratory centers in the brain stem from the motor cortex and through feedback from the proprioceptors in the muscles and joints of the active limbs. During prolonged exercise, or at higher rates of work, increases in CO_2 production, hydrogen ions (H^+), and body and blood temperatures stimulate further increases in pulmonary ventilation. At low work intensities, the increase in ventilation is mostly the result of increases in tidal volume. At higher intensities, the respiratory rate also increases. In normal-sized, untrained adults, pulmonary ventilation rates can vary from about 10 liters per minute at rest to more than 100 liters per minute at maximal rates of work; in large, highly trained male athletes, pulmonary

ventilation rates can reach more than 200 liters per minute at maximal rates of work.

Resistance Exercise

The cardiovascular and respiratory responses to episodes of resistance exercise are mostly similar to those associated with endurance exercise. One notable exception is the exaggerated blood pressure response that occurs during resistance exercise. Part of this response can be explained by the fact that resistance exercise usually involves muscle mass that develops considerable force. Such high, isolated force leads to compression of the smaller arteries and results in substantial increases in total peripheral resistance (Coyle 1991). Although high-intensity resistance training poses a potential risk to hypertensive patients and to those with cardiovascular disease, research data suggest that the risk is relatively low (Gordon et al. 1995) and that hypertensive persons may benefit from resistance training (Tipton 1991; American College of Sports Medicine 1993).

Skeletal Muscle

The primary purpose of the musculoskeletal system is to define and move the body. To provide efficient and effective force, muscle adapts to demands. In response to demand, it changes its ability to extract oxygen, choose energy sources, and rid itself of waste products. The body contains three types of muscle tissue: skeletal (voluntary) muscle, cardiac muscle or myocardium, and smooth (autonomic) muscle. This section focuses solely on skeletal muscle.

Skeletal muscle is composed of two basic types of muscle fibers distinguished by their speed of contraction—slow-twitch and fast-twitch—a characteristic that is largely dictated by different forms of the enzyme myosin adenosinetriphosphatase (ATPase). Slow-twitch fibers, which have relatively slow contractile speed, have high oxidative capacity and fatigue resistance, low glycolytic capacity, relatively high blood flow capacity, high capillary density, and high mitochondrial content (Terjung 1995). Fast-twitch muscle fibers have fast contractile speed and are classified into two subtypes, fast-twitch type "a" (FT_a) and fast-twitch type "b" (FT_b). FT_a fibers have moderately high oxidative capacity, are relatively fatigue resistant, and have high glycolytic capacity, relatively high blood flow capacity, high capillary density, and high mitochondrial content (Terjung 1995). FT_b fibers have low oxidative capacity, low fatigue resistance, high glycolytic capacity, and fast contractile speed. Further, they have relatively low blood flow capacity, capillary density, and mitochondrial content (Terjung 1995).

There is a direct relationship between predominant fiber type and performance in certain sports. For example, in most marathon runners, slow-twitch fibers account for up to or more than 90 percent of the total fibers in the leg muscles. On the other hand, the leg muscles in sprinters are often more than 80 percent composed of fast-twitch fibers. Although the issue is not totally resolved, muscle fiber type appears to be genetically determined; researchers have shown that several years of either high-intensity sprint training or high-intensity endurance training do not significantly alter the percentage of the two major types of fibers (Jolesz and Sreter 1981).

Skeletal Muscle Energy Metabolism

Metabolic processes are responsible for generating adenosine triphosphate (ATP), the body's energy source for all muscle action. ATP is generated by three basic energy systems: the ATP-phosphocreatine (ATP-PCr) system, the glycolytic system, and the oxidative system. Each system contributes to energy production in nearly every type of exercise. The relative contribution of each will depend on factors such as the intensity of work rate at the onset of exercise and the availability of oxygen in the muscle.

Energy Systems

The ATP-PCr system provides energy from the ATP stored in all of the body's cells. PCr, also found in all cells, is a high-energy phosphate molecule that stores energy. As ATP concentrations in the cell are reduced by the breakdown of ATP to adenosine diphosphate (ADP) to release energy for muscle contraction, PCr is broken down to release both energy and a phosphate to allow reconstitution of ATP from ADP. This process describes the primary energy system for short, high-intensity exercise, such as a 40- to 200-meter sprint; during such exercise, the system can produce energy at very high rates, and ATP and PCr stores, which are depleted in 10–20 seconds, will last just long enough to complete the exercise.

At high rates of work, the active muscle cell's oxygen demand exceeds its supply. The cell must then rely on the glycolytic energy system to produce ATP in the absence of oxygen (i.e., anaerobically). This system can only use glucose, available in the blood plasma and stored in both muscle and the liver as glycogen. The glycolytic energy system is the primary energy system for all-out bouts of exercise lasting from 30 seconds to 2 minutes, such as an 800-meter run. The major limitation of this energy system is that it produces lactate, which lowers the pH of both the muscle and blood. Once the pH drops below a value of 6.4 to 6.6, enzymes critical for producing energy are no longer able to function, and ATP production stops (Wilmore and Costill 1994).

The oxidative energy system uses oxygen to produce ATP within the mitochondria, which are special cell organelles within muscle. This process cannot generate ATP at a high enough rate to sustain an all-out sprint, but it is highly effective at lower rates of work (e.g., long distance running). ATP can also be produced from fat and protein metabolism through the oxidative energy system. Typically, carbohydrate and fat provide most of the ATP; under most conditions, protein contributes only 5 to 10 percent at rest and during exercise.

Metabolic Rate

The rate at which the body uses energy is known as the metabolic rate. When measured while a person is at rest, the resulting value represents the lowest (i.e., basal) rate of energy expenditure necessary to maintain basic body functions. Resting metabolic rate is measured under highly controlled resting conditions following a 12-hour fast and a good night's sleep (Turley, McBride, Wilmore 1993). To quantify the rate of energy expenditure during exercise, the metabolic rate at rest is defined as 1 metabolic equivalent (MET); a 4 MET activity thus represents an activity that requires four times the resting metabolic rate. The use of METs to quantify physical activity intensity is the basis of the absolute intensity scale. (See Chapter 2 for further information.)

Maximal Oxygen Uptake

During exercise, $\dot{V}O_2$ increases in direct proportion to the rate of work. The point at which a person's $\dot{V}O_2$ is no longer able to increase is defined as the maximal oxygen uptake ($\dot{V}O_2$max) (Figure 3-3). A person's $\dot{V}O_2$max is in part genetically determined; it can be increased through training until the point that the genetically possible maximum is reached. $\dot{V}O_2$max is considered the best estimate of a person's cardiorespiratory fitness or aerobic power (Jorgensen et al. 1977).

Lactate Threshold

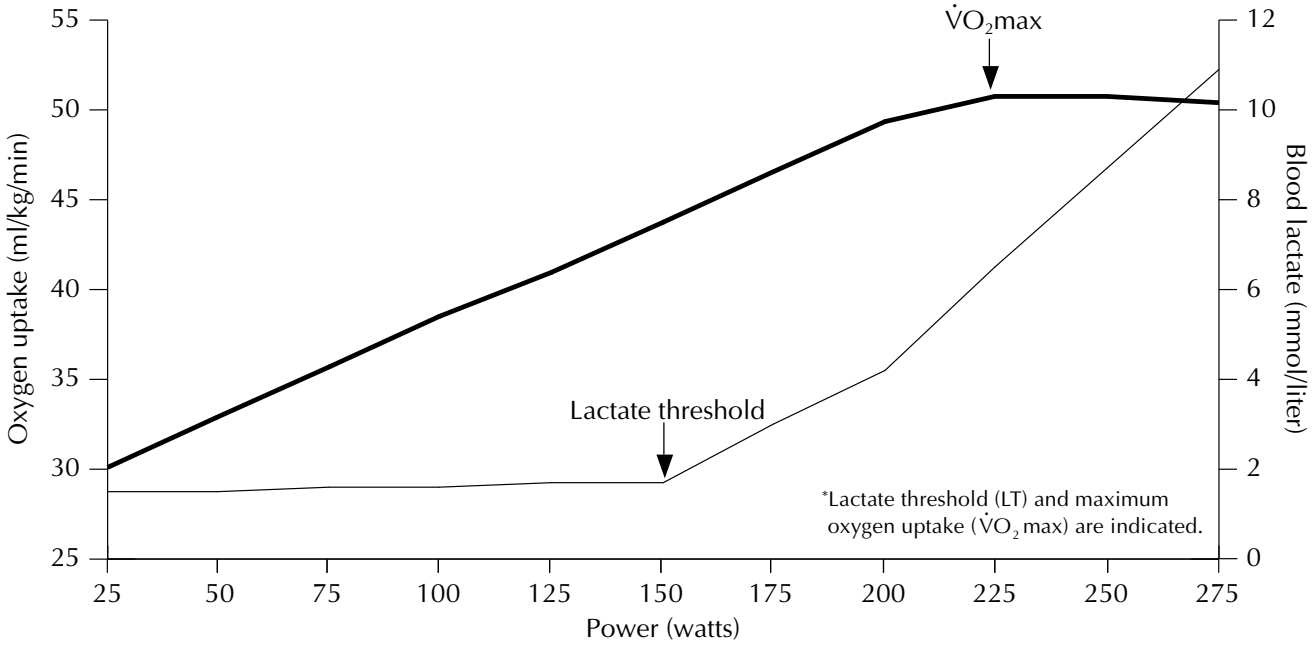
Lactate is the primary by-product of the anaerobic glycolytic energy system. At lower exercise intensities, when the cardiorespiratory system can meet the oxygen demands of active muscles, blood lactate levels remain close to those observed at rest, because some lactate is used aerobically by muscle and is removed as fast as it enters the blood from the muscle. As the intensity of exercise is increased, however, the rate of lactate entry into the blood from muscle eventually exceeds its rate of removal from the blood, and blood lactate concentrations increase above resting levels. From this point on, lactate levels continue to increase as the rate of work increases, until the point of exhaustion. The point at which the concentration of lactate in the blood begins to increase above resting levels is referred to as the lactate threshold (Figure 3-3).

Lactate threshold is an important marker for endurance performance, because distance runners set their race pace at or slightly above the lactate threshold (Farrell et al. 1979). Further, the lactate thresholds of highly trained endurance athletes occur at a much higher percentage of their $\dot{V}O_2$max, and thus at higher relative workloads, than do the thresholds of untrained persons. This key difference is what allows endurance athletes to perform at a faster pace.

Hormonal Responses to Exercise

The endocrine system, like the nervous system, integrates physiologic responses and plays an important role in maintaining homeostatic conditions at rest and during exercise. This system controls the release of hormones from specialized glands throughout the body, and these hormones exert their actions on targeted organs and cells. In response to an episode of exercise, many hormones, such as catecholamines, are secreted at an increased rate, though insulin is secreted at a decreased rate (Table 3-1). The actions of some of these hormones, as well as

Figure 3-3. Changes in oxygen uptake and blood lactate concentrations with increasing rates of work on the cycle ergometer*



their specific responses to exercise, are discussed in more detail in Chapter 4.

Immune Responses to Exercise

The immune system is a complex adaptive system that provides surveillance against foreign proteins, viruses, and bacteria by using the unique functions of cells produced by the bone marrow and the thymus gland. By interacting with neural and endocrine factors, the immune system influences the body's overall response to exercise (Reichlin 1992). A growing body of literature indicates that the incidence of some infections may be influenced by the exercise history of the individual (Nieman 1994; Hoffman-Goetz and Pedersen 1994).

Moderate exercise has been shown to bolster the function of certain components of the human immune system—such as natural killer cells, circulating T- and B-lymphocytes, and cells of the monocyte-macrophage system—thereby possibly decreasing the incidence of some infections (Keast, Cameron, Morton 1988; Pedersen and Ullum 1994; Woods and Davis 1994) and perhaps of certain types of cancer (Shephard and Shek 1995).

Exercise of high intensity and long duration or exercise that involves excessive training may have adverse effects on immune function. In general, a high-intensity, single episode of exercise results in a marked decline in the functioning of all major cells of the immune system (Newsholme and Parry-Billings 1994; Shephard and Shek 1995). In addition, overtraining may reduce the response of T-lymphocytes to mutagenic stimulation, decrease antibody synthesis and plasma level of immunoglobins and complement, and impair macrophage phagocytosis. The reduced plasma glutamine levels that occur with high-intensity exercise or excessive training are postulated to contribute to these adverse effects on the immune system (Newsholme and Parry-Billings 1994).

Long-Term Adaptations to Exercise Training

Adaptations of Skeletal Muscle and Bone

Skeletal muscle adapts to endurance training chiefly through a small increase in the cross-sectional area of slow-twitch fibers, because low- to moderate-

Table 3-1. A summary of hormonal changes during an episode of exercise

Hormone	Exercise response	Special relationships	Probable importance
Catecholamines	Increases	Greater increase with intense exercise; norepinephrine > epinephrine; increases less after training	Increased blood glucose; increased skeletal muscle and liver glycogenolysis; increased lipolysis
Growth hormone (GH)	Increases	Increases more in untrained persons; declines faster in trained persons	Unknown
Adrenocorticotropic hormone (ACTH)-cortisol	Increases	Greater increase with intense exercise; increases less after training with submaximal exercise	Increased gluconeogenesis in liver; increased mobilization of fatty acids
Thyroid-stimulating hormone (TSH)-thyroxine	Increases	Increased thyroxine turnover with training but no toxic effects are evident	Unknown
Luteinizing hormone (LH)	No change	None	None
Testosterone	Increases	None	Unknown
Estradiol-progesterone	Increases	Increases during luteal phase of the menstrual cycle	Unknown
Insulin	Decreases	Decreases less after training	Decreased stimulus to use blood glucose
Glucagon	Increases	Increases less after training	Increased blood glucose via glycogenolysis and gluconeogenesis
Renin-angiotensin-aldosterone	Increases	Same increase after training in rats	Sodium retention to maintain plasma volume
Antidiuretic hormone (ADH)	Expected increase	None	Water retention to maintain plasma volume
Parathormone (PTH)-calcitonin	Unknown	None	Needed to establish proper bone development
Erythropoietin	Unknown	None	Would be important to increase erythropoiesis
Prostaglandins	May increase	May increase in response to sustained isometric contractions; may need ischemic stress	May be local vasodilators

Adapted from Wilmore JH, Costill DL. *Physiology of sport and exercise*. Champaign, IL: Human Kinetics, 1994, p. 136.

intensity aerobic activity primarily recruits these fibers (Abernethy, Thayer, Taylor 1990). Prolonged endurance training (i.e., months to years) can lead to a transition of FT_b fibers to FT_a fibers, which have a higher oxidative capacity (Abernethy, Thayer, Taylor 1990). No substantive evidence indicates that fast-twitch fibers will convert to slow-twitch fibers under normal training conditions (Jolesz and Sreter 1981). Endurance training also increases the number of capillaries in trained skeletal muscle, thereby allowing a greater capacity for blood flow in the active muscle (Terjung 1995).

Resistance-trained skeletal muscle exerts considerably more force because of both increased muscle size (hypertrophy) and increased muscle fiber recruitment. Fiber hypertrophy is the result of increases in both the size and number of myofibrils in both fast-twitch and slow-twitch muscle fibers (Kannus et al. 1992). Hyperplasia, or increased fiber number, has been reported in animal studies, where the number of individual muscle fibers can be counted (Gonyea et al. 1986), and has been indirectly demonstrated during autopsies on humans by using direct fiber counts to compare dominant and nondominant paired muscles (Sjöström et al. 1991).

During both aerobic and resistance exercise, active muscles can undergo changes that lead to muscle soreness. Some soreness is felt immediately after exercise, and some can even occur during exercise. This muscle soreness is generally not physically limiting and dissipates rapidly. A more limiting soreness, however, may occur 24 to 48 hours following exercise. This delayed-onset muscle soreness is primarily associated with eccentric-type muscle action, during which the muscle exerts force while lengthening, as can happen when a person runs down a steep hill or lowers a weight from a fully flexed to a fully extended position (e.g., the two-arm curl). Delayed-onset muscle soreness is the result of structural damage to the muscle; in its most severe form, this damage may include rupture of the cell membrane and disruption of the contractile elements of individual muscle fibers (Armstrong, Warren, Warren 1991). Such damage appears to result in an inflammatory response (MacIntyre, Reid, McKenzie 1995).

Total inactivity results in muscle atrophy and loss of bone mineral and mass. Persons who are sedentary generally have less bone mass than those who exercise, but the increases in bone mineral and mass that result from either endurance or resistance training are relatively small (Chesnut 1993). The role of resistance training in increasing or maintaining bone mass is not well characterized. Endurance training has little demonstrated positive effect on bone mineral and mass. Nonetheless, even small increases in bone mass gained from endurance or resistance training can help prevent or delay the process of osteoporosis (Drinkwater 1994). (See Chapter 4 for further information on the effects of exercise on bone.)

The musculoskeletal system cannot function without connective tissue linking bones to bones (ligaments) and muscles to bones (tendons). Extensive animal studies indicate that ligaments and tendons become stronger with prolonged and high-intensity exercise. This effect is the result of an increase in the strength of insertion sites between ligaments, tendons, and bones, as well as an increase in the cross-sectional areas of ligaments and tendons. These structures also become weaker and smaller with several weeks of immobilization (Tipton and Vailas 1990), which can have important implications for musculoskeletal performance and risk of injury.

Metabolic Adaptations

Significant metabolic adaptations occur in skeletal muscle in response to endurance training. First, both the size and number of mitochondria increase substantially, as does the activity of oxidative enzymes. Myoglobin content in the muscle can also be augmented, increasing the amount of oxygen stored in individual muscle fibers (Hickson 1981), but this effect is variable (Svedenhag, Henriksson, Sylvén 1983). Such adaptations, combined with the increase in capillaries and muscle blood flow in the trained muscles (noted in a previous section), greatly enhance the oxidative capacity of the endurance-trained muscle.

Endurance training also increases the capacity of skeletal muscle to store glycogen (Kiens et al. 1993). The ability of trained muscles to use fat as an energy source is also improved, and this greater reliance on fat spares glycogen stores (Kiens et al. 1993). The increased capacity to use fat following endurance training results from an enhanced ability to mobilize free-fatty acids from fat depots and an improved capacity to oxidize fat consequent to the increase in the muscle enzymes responsible for fat oxidation (Wilmore and Costill 1994).

These changes in muscle and in cardiorespiratory function are responsible for increases in both $\dot{V}O_2$max and lactate threshold. The endurance-trained person can thus perform at considerably higher rates of work than the untrained person. Increases in $\dot{V}O_2$max generally range from 15 to 20 percent following a 6-month training period (Wilmore and Costill 1994). However, individual variations in this response are considerable. In one study of 60- to 71-year-old men and women who endurance trained for 9 to 12 months, the improvement in $\dot{V}O_2$max varied from 0 to 43 percent; the mean increase was 24 percent (Kohrt et al. 1991). This variation in response may be due in part to genetic factors and to initial levels of fitness. To illustrate the changes that can be expected with endurance training, a hypothetical sedentary man's pretraining values have been compared with his values after a 6-month period of endurance training and with the values of a typical elite endurance runner (Table 3-2).

Responses to endurance training are similar for men and women. At all ages, women and men show similar gains in strength from resistance training (Rogers and Evans 1993; Holloway and Baechle 1990)

Table 3-2. A hypothetical example of alterations in selected physiological variables consequent to a 6-month endurance training program in a previously sedentary man compared with those of a typical elite endurance runner

	Sedentary man		
Variable	**Pretraining**	**Posttraining**	**Runner**
Cardiovascular			
HR at rest (beats • min^{-1})	71	59	36
HR max (beats • min^{-1})	185	183	174
SV rest (ml)	65	80	125
SV max (ml)	120	140	200
$\dot{Q}$ rest (L • min^{-1})	4.6	4.7	4.5
$\dot{Q}$ max (L • min^{-1})	22.2	25.6	32.5
Heart volume (ml)	750	820	1,200
Blood volume (L)	4.7	5.1	6.0
Systolic BP rest (mmHg)	135	130	120
Systolic BP max (mmHg)	210	205	210
Diastolic BP rest (mmHg)	78	76	65
Diastolic BP max (mmHg)	82	80	65
Respiratory			
$\dot{V}_E$ rest (L • min^{-1})	7	6	6
$\dot{V}_E$ rest (L • min^{-1})	110	135	195
TV rest (L)	0.5	0.5	0.5
TV max (L)	2.75	3.0	3.9
RR rest (breaths • min^{-1})	14	12	12
RR max (breaths • min^{-1})	40	45	50
Metabolic			
A-$\bar{v}O_2$ diff rest (ml • 100 ml^{-1})	6.0	6.0	6.0
A-$\bar{v}O_2$ diff max (ml • 100 ml^{-1})	14.5	15.0	16.0
$\dot{V}O_2$ rest (ml • kg^{-1} • min^{-1})	3.5	3.5	3.5
$\dot{V}O_2$ max (ml • kg^{-1} • min^{-1})	40.5	49.8	76.5
Blood lactate rest (mmol • L^{-1})	1.0	1.0	1.0
Blood lactate max (mmol • L^{-1})	7.5	8.5	9.0

Adapted from Wilmore JH, Costill DL. *Physiology of sport and exercise*. Champaign, IL: Human Kinetics, 1994, p. 230.

HR = heart rate; max = maximal; SV = stroke volume; $\dot{Q}$ = cardiac output; BP = blood pressure; $\dot{V}_E$ = ventilatory volume; TV = tidal volume; RR = respiration rate; A-$\bar{v}O_2$ diff = arterial-mixed venous oxygen difference; $\dot{V}O_2$ = oxygen consumption.

and similar gains in $\dot{V}O_2$max from aerobic endurance training (Kohrt et al. 1991; Mitchell et al. 1992).

Cardiovascular and Respiratory Adaptations

Endurance training leads to significant cardiovascular and respiratory changes at rest and during steady-state exercise at both submaximal and maximal rates of work. The magnitude of these adaptations largely depends on the person's initial fitness level; on mode, intensity, duration, and frequency of exercise; and on the length of training (e.g., weeks, months, years).

Long-Term Cardiovascular Adaptations

Cardiac output at rest and during submaximal exercise is essentially unchanged following an endurance training program. At or near maximal rates of work, however, cardiac output is increased substantially, up to 30 percent or more (Saltin and Rowell 1980). There are important differences in the responses of stroke volume and heart rate to training. After training, stroke volume is increased at rest, during submaximal exercise, and during maximal exercise; conversely, posttraining heart rate is decreased at rest and during submaximal exercise and is usually unchanged at maximal rates of work. The increase in stroke volume appears to be the dominant change and explains most of the changes observed in cardiac output.

Several factors contribute to the increase in stroke volume from endurance training. Endurance training increases plasma volume by approximately the same percentage that it increases stroke volume (Green, Jones, Painter 1990). An increased plasma volume increases the volume of blood available to return to the right heart and, subsequently, to the left ventricle. There is also an increase in the end-diastolic volume (the volume of blood in the heart at the end of the diastolic filling period) because of increased amount of blood and increased return of blood to the ventricle during exercise (Seals et al. 1994). This acute increase in the left ventricle's end-diastolic volume stretches its walls, resulting in a more elastic recoil.

Endurance training also results in long-term changes in the structure of the heart that augment stroke volume. Short-term adaptive responses include ventricular dilatation; this increase in the volume of the ventricles allows end-diastolic volume to increase without excessive stress on the ventricular walls. Long-term adaptive responses include hypertrophy of the cardiac muscle fibers (i.e., increases in the size of each fiber). This hypertrophy increases the muscle mass of the ventricles, permitting greater force to be exerted with each beat of the heart. Increases in the thickness of the posterior and septal walls of the left ventricle can lead to a more forceful contraction of the left ventricle, thus emptying more of the blood from the left ventricle (George, Wolfe, Burggraf 1991).

Endurance training increases the number of capillaries in trained skeletal muscle, thereby allowing a greater capacity for blood flow in the active muscle (Terjung 1995). This enhanced capacity for blood flow is associated with a reduction in total peripheral resistance; thus, the left ventricle can exert a more forceful contraction against a lower resistance to flow out of the ventricle (Blomqvist and Saltin 1983).

Arterial blood pressure at rest, blood pressure during submaximal exercise, and peak blood pressure all show a slight decline as a result of endurance training in normotensive individuals (Fagard and Tipton 1994). However, decreases are greater in persons with high blood pressure. After endurance training, resting blood pressure (systolic/diastolic) will decrease on average -3/-3 mmHg in persons with normal blood pressure; in borderline hypertensive persons, the decrease will be -6/-7 mmHg; and in hypertensive persons, the decrease will be -10/-8 mmHg (Fagard and Tipton 1994). (See Chapter 4 for further information.)

Respiratory Adaptations

The major changes in the respiratory system from endurance training are an increase in the maximal rate of pulmonary ventilation, which is the result of increases in both tidal volume and respiration rate, and an increase in pulmonary diffusion at maximal rates of work, primarily due to increases in pulmonary blood flow, particularly to the upper regions of the lung.

Maintenance, Detraining, and Prolonged Inactivity

Most adaptations that result from both endurance and resistance training will be reversed if a person stops or reduces training. The greatest deterioration

in physiologic function occurs during prolonged bed rest and immobilization by casts. A basic maintenance training program is necessary to prevent these losses in function.

Maintaining Fitness and Muscular Strength

Muscle strength and cardiorespiratory capacity are dependent on separate aspects of exercise. After a person has obtained gains in $\dot{V}O_2$max by performing cardiorespiratory exercise six times per week, two to four times per week is the optimal frequency of training to maintain those gains (Hickson and Rosenkoetter 1981). Further, a substantial part of the gain can be retained when the duration of each session is reduced by as much as two-thirds, but only if the intensity during these abbreviated sessions is maintained at ≥70 percent of $\dot{V}O_2$max (Hickson et al. 1985). If training intensity is reduced by as little as one-third, however, a substantial reduction in $\dot{V}O_2$max can be expected over the next 15 weeks (Hickson et al. 1985).

In previously untrained persons, gains in muscular strength can be sustained by as little as a single session per week of resistance training, but only if the intensity is not reduced (Graves et al. 1988).

Detraining

With complete cessation of exercise training, a significant reduction in $\dot{V}O_2$max and a decrease in plasma volume occur within 2 weeks; all prior functional gains are dissipated within 2 to 8 months, even if routine low- to moderate-intensity physical activity has taken the place of training (Shephard 1994). Muscular strength and power are reduced at a much slower rate than $\dot{V}O_2$max, particularly during the first few months after an athlete discontinues resistance training (Fleck and Kraemer 1987). In fact, no decrement in either strength or power may occur for the first 4 to 6 weeks after training ends (Neufer et al. 1987). After 12 months, almost half of the strength gained might still be retained if the athlete remains moderately active (Wilmore and Costill 1994).

Prolonged Inactivity

The effects of prolonged inactivity have been studied by placing healthy young male athletes and sedentary volunteers in bed for up to 3 weeks after a control period during which baseline measurements were made. The resulting detrimental changes in physiologic function and performance are similar to those resulting from reduced gravitational forces during space flight and are more dramatic than those resulting from detraining studies in which routine daily activities in the upright position (e.g., walking, stair climbing, lifting, and carrying) are not restricted.

Results of bed rest studies show numerous physiologic changes, such as profound decrements in cardiorespiratory function proportional to the duration of bed rest (Shephard 1994; Saltin et al. 1968). Metabolic disturbances evident within a few days of bed rest include reversible glucose intolerance and hyperinsulinemia in response to a standard glucose load, reflecting cell insulin resistance (Lipman et al. 1972); reduced total energy expenditure; negative nitrogen balance, reflecting loss of muscle protein; and negative calcium balance, reflecting loss of bone mass (Bloomfield and Coyle 1993). There is also a substantial decrease in plasma volume, which affects aerobic power.

From one study, a decline in $\dot{V}O_2$max of 15 percent was evident within 10 days of bed rest and progressed to 27 percent in 3 weeks; the rate of loss was approximately 0.8 percent per day of bed rest (Bloomfield and Coyle 1993). The decrement in $\dot{V}O_2$max from bed rest and reduced activity results from a decrease in maximal cardiac output, consequent to a reduced stroke volume. This, in turn, reflects the decrease in end-diastolic volume resulting from a reduction in total blood and plasma volume, and probably also from a decrease in myocardial contractility (Bloomfield and Coyle 1993). Maximal heart rate and A-$\bar{v}O_2$ difference remain unchanged (Bloomfield and Coyle 1993). Resting heart rate remains essentially unchanged or is slightly increased, whereas resting stroke volume and cardiac output remain unchanged or are slightly decreased. However, the heart rate for submaximal exertion is generally increased to compensate for the sizable reduction in stroke volume.

Physical inactivity associated with bed rest or prolonged weightlessness also results in a progressive decrement in skeletal muscle mass (disuse atrophy) and strength, as well as an associated reduction in bone mineral density that is approximately proportional to the duration of immobilization or weightlessness (Bloomfield and Coyle 1993). The loss of muscle mass is not as great as that which

occurs with immobilization of a limb by a plaster cast, but it exceeds that associated with cessation of resistance exercise training. The muscle groups most affected by prolonged immobilization are the antigravity postural muscles of the lower extremities (Bloomfield and Coyle 1993). The loss of normal mechanical strain patterns from contraction of these muscles results in a corresponding loss of density in the bones of the lower extremity, particularly the heel and the spine (Bloomfield and Coyle 1993). Muscles atrophy faster than bones lose their density. For example, 1 month of bed rest by healthy young men resulted in a 10 to 20 percent decrease in muscle fiber cross-sectional area and a 21 percent reduction in peak isokinetic torque of knee extensors (Bloomfield and Coyle 1993), whereas a similar period of bed rest resulted in a reduction in bone mineral density of only 0.3 to 3 percent for the lumbar spine and 1.5 percent for the heel.

Quantitative histologic examination of muscle biopsies of the vastus lateralis of the leg following immobilization shows reduced cross-sectional area for both slow-twitch and fast-twitch fibers, actual necrotic changes in affected fibers, loss of capillary density, and a decline in aerobic enzyme activity, creatinine phosphate, and glycogen stores (Bloomfield and Coyle 1993). On resuming normal activity, reversibility of these decrements in cardiorespiratory, metabolic, and muscle function is fairly rapid (within days to weeks) (Bloomfield and Coyle 1993). By contrast, the reversal of the decrement of bone mineral density requires weeks to months.

Special Considerations

The physiologic responses to exercise and physiologic adaptations to training and detraining, reviewed in the preceding sections, can be influenced by a number of factors, including physical disability, environmental conditions, age, and sex.

Disability

Although there is a paucity of information about physiologic responses to exercise among persons with disabilities, existing information supports the notion that the capacity of these persons to adapt to increased levels of physical activity is similar to that of persons without disabilities. Many of the acute effects of physical activity on the cardiovascular, respiratory, endocrine, and musculoskeletal systems have been demonstrated to be similar among persons with disabilities, depending on the specific nature of the disability. For example, physiologic responses to exercise have been studied among persons with paraplegia (Davis 1993), quadriplegia (Figoni 1993), mental retardation (Fernhall 1993), multiple sclerosis (Ponichtera-Mulcare 1993), and postpolio syndrome (Birk 1993).

Environmental Conditions

The basic physiologic responses to an episode of exercise vary considerably with changes in environmental conditions. As environmental temperature and humidity increase, the body is challenged to maintain its core temperature. Generally, as the body's core temperature increases during exercise, blood vessels in the skin begin to dilate, diverting more blood to the body's surface, where body heat can be passed on to the environment (unless environmental temperature exceeds body temperature). Evaporation of water from the skin's surface significantly aids in heat loss; however, as humidity increases, evaporation becomes limited.

These methods for cooling can compromise cardiovascular function during exercise. Increasing blood flow to the skin creates competition with the active muscles for a large percentage of the cardiac output. When a person is exercising for prolonged periods in the heat, stroke volume will generally decline over time consequent to dehydration and increased blood flow in the skin (Rowell 1993; Montain and Coyle 1992). Heart rate increases substantially to try to maintain cardiac output to compensate for the reduced stroke volume.

High air temperature is not the only factor that stresses the body's ability to cool itself in the heat. High humidity, low air velocity, and the radiant heat from the sun and reflective surfaces also contribute to the total effect. For example, exercising under conditions of 32°C (90°F) air temperature, 20 percent relative humidity, 3.0 kilometers per hour (4.8 miles per hour) air velocity, and cloud cover is much more comfortable and less stressful to the body than the same exercise under conditions of 24°C (75°F) air temperature, 90 percent relative humidity, no air movement, and direct exposure to the sun.

Children respond differently to heat than adults do. Children have a higher body surface area to body mass ratio (surface area/mass), which facilitates heat loss when environmental temperatures are below skin temperature. When environmental temperature exceeds skin temperature, children are at an even greater disadvantage because these mechanisms then become avenues of heat gain. Children also have a lower rate of sweat production; even though they have more heat-activated sweat glands, each gland produces considerably less sweat than that of an adult (Bar-Or 1983).

The inability to maintain core temperature can lead to heat-related injuries. Heat cramps, characterized by severe cramping of the active skeletal muscles, is the least severe of three primary heat disorders. Heat exhaustion results when the demand for blood exceeds what is available, leading to competition for the body's limited blood supply. Heat exhaustion is accompanied by symptoms including extreme fatigue, breathlessness, dizziness, vomiting, fainting, cold and clammy or hot and dry skin, hypotension, and a weak, rapid pulse (Wilmore and Costill 1994). Heatstroke, the most extreme of the three heat disorders, is characterized by a core temperature of 40˚C (104˚F) or higher, cessation of sweating, hot and dry skin, rapid pulse and respiration, hypertension, and confusion or unconsciousness. If left untreated, heatstroke can lead to coma, then death. People experiencing symptoms of heat-related injury should be taken to a shady area, cooled with by whatever means available, and if conscious given nonalcoholic beverages to drink. Medical assistance should be sought. To reduce the risk of developing heat disorders, a person should drink enough fluid to try to match that which is lost through sweating, avoid extreme heat, and reduce the intensity of activity in hot weather. Because children are less resistant to the adverse effects of heat during exercise, special attention should be given to protect them when they exercise in the heat and to provide them with extra fluids to drink.

Stresses associated with exercising in the extreme cold are generally less severe. For most situations, the problems associated with cold stress can be eliminated by adequate clothing. Still, cold stress can induce a number of changes in the physiologic response to exercise (Doubt 1991; Jacobs, Martineau, Vallerand 1994; Shephard 1993). These include the increased generation of body heat by vigorous activity and shivering, increased production of catecholamines, vasoconstriction in both the cutaneous and nonactive skeletal muscle beds to provide insulation for the body's core, increased lactate production, and a higher oxygen uptake for the same work (Doubt 1991). For the same absolute temperature, exposure to cold water is substantially more stressful than exposure to cold air because the heat transfer in water is about 25 times greater than in air (Toner and McArdle 1988). Because the ratio of surface area to mass is higher in children than in adults, children lose heat at a faster rate when exposed to the same cold stress. The elderly tend to have a reduced response of generating body heat and are thus more susceptible to cold stress.

Altitude also affects the body's physiologic responses to exercise. As altitude increases, barometric pressure decreases, and the partial pressure of inhaled oxygen is decreased proportionally. A decreased partial pressure of oxygen reduces the driving force to unload oxygen from the air to the blood and from the blood to the muscle, thereby compromising oxygen delivery (Fulco and Cymerman 1988). $\dot{V}O_2max$ is significantly reduced at altitudes greater than 1,500 meters. This effect impairs the performance of endurance activities. The body makes both short-term and long-term adaptations to altitude exposure that enable it to at least partially adapt to this imposed stress. Because oxygen delivery is the primary concern, the initial adaptation that occurs within the first 24 hours of exposure to altitude is an increased cardiac output both at rest and during submaximal exercise. Ventilatory volumes are also increased. An ensuing reduction in plasma volume increases the concentration of red blood cells (hemoconcentration), thus providing more oxygen molecules per unit of blood (Grover, Weil, Reeves 1986). Over several weeks, the red blood cell mass is increased through stimulation of the bone marrow by the hormone erythropoietin.

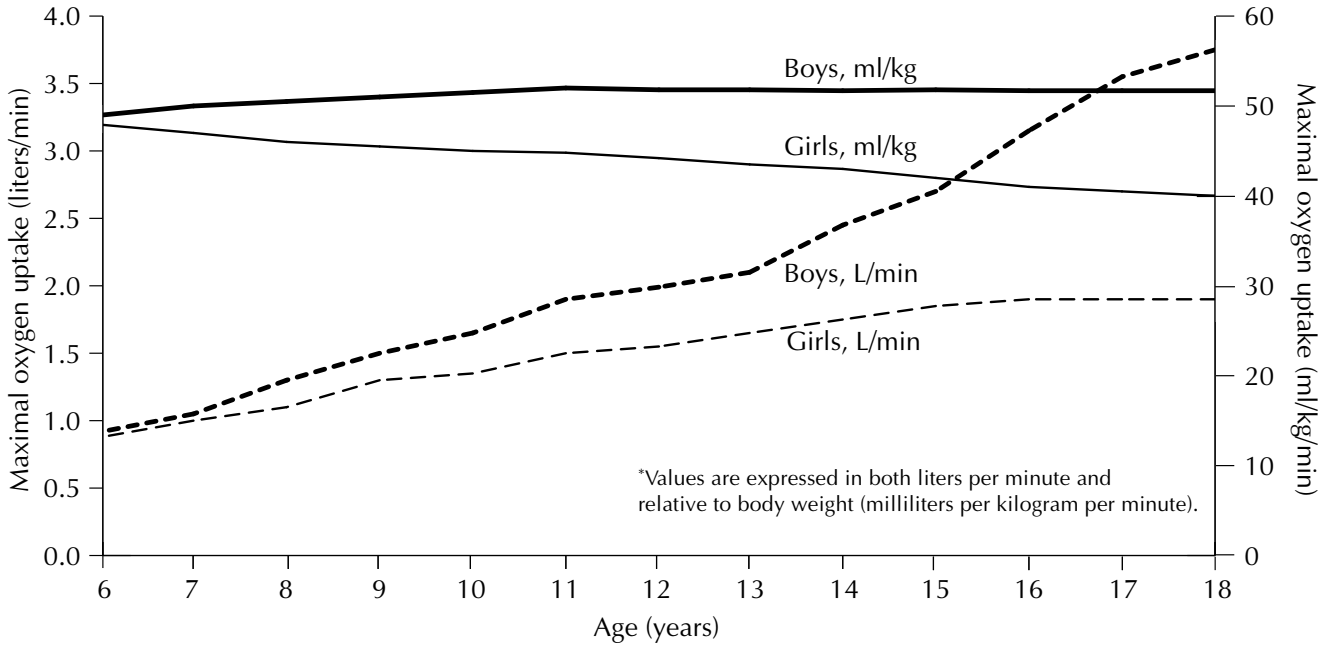
Exercising vigorously outdoors when air quality is poor can also produce adverse physiologic responses. In addition to decreased tolerance for exercise, direct respiratory effects include increased airway reactivity and potential exposure to harmful vapors and airborne dusts, toxins, and pollens (Wilmore and Costill 1994).

Effects of Age

When absolute values are scaled to account for differences in body size, most differences in physiologic function between children and adults disappear. The exceptions are notable. For the same absolute rate of work on a cycle ergometer, children will have approximately the same metabolic cost, or $\dot{V}O_2$ demands, but they meet those demands differently. Because children have smaller hearts, their stroke volume is lower than that for adults for the same rate of work. Heart rate is increased to compensate for the lower stroke volume; but because this increase is generally inadequate, cardiac output is slightly lower (Bar-Or 1983). The A-$\bar{v}O_2$ difference is therefore increased to compensate for the lower cardiac output to achieve the same $\dot{V}O_2$. The $\dot{V}O_2$max, expressed in liters per minute, increases during the ages of 6–18 years for boys and 6–14 years for girls (Figure 3-4) before it reaches a plateau (Krahenbuhl, Skinner, Kohrt 1985). When expressed relative to body weight (milliliters per kilogram per minute), $\dot{V}O_2$max remains fairly stable for boys from 6–18 years of age but decreases steadily for girls during those years (Figure 3-4) (Krahenbuhl, Skinner, Kohrt 1985). Most likely, different patterns of physical activity contribute to this variation because the difference in aerobic capacity between elite female endurance athletes and elite male endurance athletes is substantially less than the difference between boys and girls in general (e.g., 10 percent vs. 25 percent) (Wilmore and Costill 1994).

The deterioration of physiologic function with aging is almost identical to the change in function that generally accompanies inactivity. Maximal heart rate and maximal stroke volume are decreased in older adults; maximal cardiac output is thus decreased, which results in a $\dot{V}O_2$max lower than that of a young adult (Raven and Mitchell 1980). The decline in $\dot{V}O_2$max approximates 0.40 to 0.50 milliliters per kilogram per minute per year in men, according to data from cross-sectional studies; this rate of decline is less in women (Buskirk and Hodgson 1987). Through training, both older men and women can increase their $\dot{V}O_2$max values by approximately the same percentage as those seen

Figure 3-4. Changes in $\dot{V}O_2$max with increasing age from 6 to 18 years of age in boys and girls*



Data were taken from Krahenbuhl GS, Skinner JS, Kohrt WM 1985 and Bar-Or O 1983.

in younger adults (Kohrt et al. 1991). The interrelationships of age, $\dot{V}O_2$max, and training status are evident when the loss in $\dot{V}O_2$max with age is compared for active and sedentary individuals (Figure 3-5).

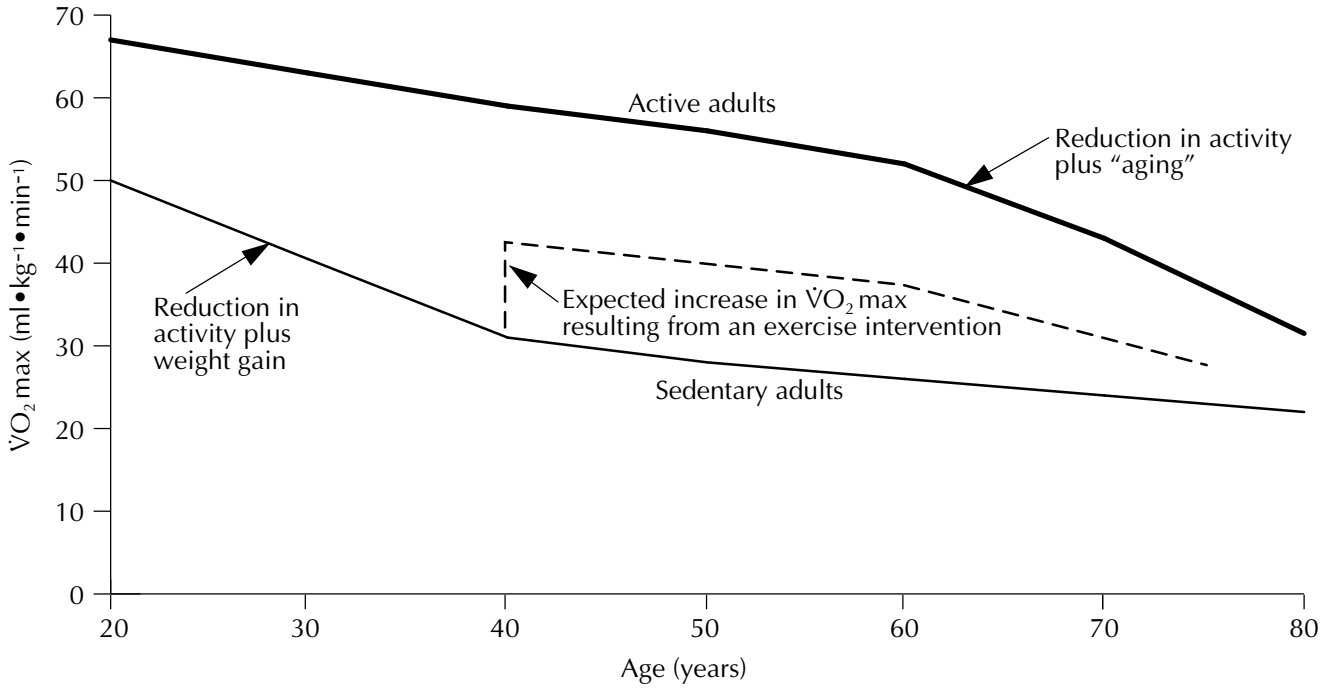
When the cardiorespiratory responses of an older adult are compared with those of a young or middle-aged adult at the same absolute submaximal rate of work, stroke volume for an older person is generally lower and heart rate is higher from the attempt to maintain cardiac output. Because this attempt is generally insufficient, the A-$\bar{v}O_2$ difference must increase to provide the same submaximal oxygen uptake (Raven and Mitchell 1980; Thompson and Dorsey 1986). Some researchers have shown, however, that cardiac output can be maintained at both submaximal and maximal rates of work through a higher stroke volume in older adults (Rodeheffer et al. 1984).

The deterioration in physiological function normally associated with aging is, in fact, caused by a combination of reduced physical activity and the aging process itself. By maintaining an active lifestyle, or by increasing levels of physical activity if previously sedentary, older persons can maintain relatively high levels of cardiovascular and metabolic function, including $\dot{V}O_2$max (Kohrt et al. 1991), and of skeletal muscle function (Rogers and Evans 1993). For example, Fiatarone and colleagues (1994) found an increase of 113 percent in the strength of elderly men and women (mean age of 87.1 years) following a 10-week training program of progressive resistance exercise. Cross-sectional thigh muscle area was increased, as was stair-climbing power, gait velocity, and level of spontaneous activity. Increasing endurance and strength in the elderly contributes to their ability to live independently.

Differences by Sex

For the most part, women and men who participate in exercise training have similar responses in cardiovascular, respiratory, and metabolic function (providing that size and activity level are normalized). Relative increases in $\dot{V}O_2$max are equivalent

Figure 3-5. Changes in $\dot{V}O_2$ max with aging, comparing an active population and sedentary population (the figure also illustrates the expected increase in $\dot{V}O_2$ max when a previously sedentary person begins an exercise program)



Adapted, by permission, from Buskirk ER, Hodgson JL. *Federation Proceedings* 1987.

for women and men (Kohrt et al. 1991; Mitchell et al. 1992). Some evidence suggests that older women accomplish this increase in $\dot{V}O_2$max mainly through an increase in the A-$\bar{v}O_2$ difference, whereas younger women and men have substantial increases in stroke volume, which increases maximal cardiac output (Spina et al. 1993). With resistance training, women experience equivalent increases in strength (Rogers and Evans 1993; Holloway and Baechle 1990), although they gain less fat-free mass due to less muscle hypertrophy.

Several sex differences have been noted in the acute response to exercise. At the same absolute rate of exercise, women have a higher heart rate response than men, primarily because of a lower stroke volume. This lower stroke volume is a function of smaller heart size and smaller blood volume. In addition, women have less potential to increase the A-$\bar{v}O_2$ difference because of lower hemoglobin content. Those differences, in addition to greater fat mass, result in a lower $\dot{V}O_2$max in women, even when normalized for size and level of training (Lewis, Kamon, Hodgson 1986).

Conclusions

1. Physical activity has numerous beneficial physiologic effects. Most widely appreciated are its effects on the cardiovascular and musculoskeletal systems, but benefits on the functioning of metabolic, endocrine, and immune systems are also considerable.
2. Many of the beneficial effects of exercise training—from both endurance and resistance activities—diminish within 2 weeks if physical activity is substantially reduced, and effects disappear within 2 to 8 months if physical activity is not resumed.
3. People of all ages, both male and female, undergo beneficial physiologic adaptations to physical activity.

Research Needs

1. Explore individual variations in response to exercise.
2. Better characterize mechanisms through which the musculoskeletal system responds differentially to endurance and resistance exercise.
3. Better characterize the mechanisms by which physical activity reduces the risk of cardiovascular disease, hypertension, and non–insulin-dependent diabetes mellitus.
4. Determine the minimal and optimal amount of exercise for disease prevention.
5. Better characterize beneficial activity profiles for people with disabilities.

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