PHYSIOLOGIC RESPONSES AND ADAPTATIONS OF CARDIOVASCULAR SYSTEM TO EXERCISE

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.

The body's physiologic responses to episodes of exercises occur in the musculoskeletal, cardiovascular, respiratory, endocrine, and immune systems.

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. 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 (VO2) increases linearly with increasing rates of work.

Cardiac Output

Cardiac output (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-vO2) difference is the difference between the oxygen content of the arterial and mixed venous blood. 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.

The relationship between cardiac output, oxygen uptake, and difference between the oxygen content of arterial and mixed-venous blood embodies the principle discovered by German physiologist Adolph Fick (1829–1901) in 1870.

Cardiac output, mL/min [VO2, mL/min ÷ a–vO2 diff, mL]*100

RESTING CARDIAC OUTPUT: UNTRAINED VERSUS TRAINED

Each minute, the left ventricle ejects the entire 5-L blood volume of an average sized man. This value pertains to most individuals, but stroke volume and heart rate vary considerably depending on cardiovascular fitness status. A heart rate of about 70 b/min sustains the average adult's 5-L (5000 mL) resting cardiac output. Substituting this heart rate value in the cardiac output equation (Cardiac output = Stroke volume *Heart rate; Stroke volume = Cardiac output ÷ Heart rate) yields a calculated stroke volume of 71 mL/b.

The resting heart rate for an endurance athlete averages close to 50 b/min. The athlete's resting cardiac output also averages 5 L/min as blood circulates with a proportionately larger stroke volume of 100 mL per beat (5000 mL ÷ 50 b/min). Stroke volumes for women usually average 25% below values for men with equivalent training. The smaller body size of the typical woman chiefly accounts for this "gender difference."

	Cardiac Output Rate ($mL \cdot min^{-1}$)	Heart Stroke ($b \cdot min^{-1}$)	Volume ($mL \cdot b^{-1}$)
Untrained	5000	70	71
Trained	5000	50	100

The underlying mechanisms for the heart rate and stroke volume differences between trained and untrained individuals remain unclear. The following two factors probably interact as aerobic fitness improves

1. Increased vagal tone slows the heart, allowing more time for ventricular filling.

2. Enlarged ventricular volume and a more powerful myocardium eject a larger volume of blood with each systole.

EXERCISE CARDIAC OUTPUT: UNTRAINED VERSUS TRAINED

Blood flow from the heart increases in direct proportion to exercise intensity for both trained and untrained individuals. From rest to steady-rate exercise, cardiac output increases rapidly, followed by a more gradual increase until it plateaus as blood flow matches exercise metaboli requirements.

In sedentary, college-age men, cardiac output in maximal aerobic exercise increases about four times the resting level to an average maximum of 22 L of blood per minute. Maximum heart rate for these young adults averages 195b/min. Consequently, stroke volume averages 113 mL of blood per beat during exercise (22,000mL÷195b/min). In contrast, world-class endurance athletes generate maximum cardiac outputs of 35L/min, with a similar or slightly lower maximum heart rate than untrained counterparts. The difference between maximum cardiac output of both individuals relates solely to differences in stroke volume.

	Cardiac Output Rate ($mL \cdot min^{-1}$)	Heart Stroke ($b \cdot min^{-1}$)	Volume ($mL \cdot b^{-1}$)
Untrained	22,000	195	113
Trained	35,000	195	179

STROKE VOLUME

Stroke volume, at rest or during exercise, is regulated by three factors: (1) the end-diastolic volume (EDV), which is the volume of blood in the ventricles at the end of diastole; (2) the average aortic blood pressure; and (3) the strength of ventricular contraction. EDV is often referred to as “preload,” and it influences stroke volume in the following way. Two physiologists, Frank and Starling, demonstrated that the strength of ventricular contraction increased with an

enlargement of EDV (i.e., stretch of the ventricles). This relationship has become known as the Frank Starling law of the heart. The increase in EDV results in a lengthening of cardiac fibers, which improves the force of contraction in a manner similar to that seen in skeletal muscle. The mechanism to explain the influence of fiber length on cardiac contractility is that an increase in the length of cardiac fibers increases the number of myosin crossbridge interactions with actin, resulting in increased force production. A rise in cardiac contractility results in an increase in the amount of blood pumped per beat. The principal variable that influences EDV is the rate of venous return to the heart. An increase in venous return results in a rise in EDV and, therefore, an increase in stroke volume. Increased venous return and the resulting increase in EDV play a key role in the increase in stroke volume observed during upright exercise.

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.

Blood Pressure

During upright exercise, the normal blood pressure response is to observe a progressive increase in systolic blood pressure with no change or even a slight decrease in diastolic blood pressure. The slight decrease in diastolic blood pressure is due primarily to the vasodilation of the arteries from the exercise bout. Thus, the expansion in artery size may lower blood pressure during the diastolic phase. A failure of the systolic blood pressure to rise with an increase in intensity (called exertional hypotension) is considered abnormal, and may occur in patients with a number of cardiovascular problems. Contrariwise, an increase in diastolic blood pressure of more than 10 mmHg during or after exercise represents an unstable form of hypertension, and may be associated with coronary artery disease. Following exercise, systolic blood pressure progressively declines during an active recovery. With a passive (such as seated) recovery, systolic blood pressure may drop abruptly due to the pooling of blood in the peripheral areas of the body. There may also be a drop in diastolic blood pressure, during the recovery phase of exercise due to the vasodilation. Persons on medications will have variable responses to exercise. Therefore it is prudent to contact their health practitioners to be aware of what would be considered normative under these circumstances.

Arterial-Mixed Venous O2 Content during Exercise

The a− vO2 difference represents the amount of O2 that is taken up from 100 ml of blood by the tissues during one trip around the systemic circuit. An increase in the a − vO2 difference during exercise is due to an increase in the amount of O2 taken up and used for the oxidative production of ATP by skeletal muscle. The relationship between cardiac output ($\dot{Q}$), a − vO2 diff, and oxygen uptake is given by the Fick equation: VO2 = Q × (a – vO2 diff) Simply stated, the Fick equation says that VO2 is equal to the product of cardiac output and the

a −vO2 diff. This means that an increase in either cardiac output or a − vO2 difference would elevate VO2. So that, during exercise, oxygen consumption increases due to both an increase in cardiac output and an increase in the a−vO2 difference.

REFERENCES

- ✓ Katch, and Victor L. Katch. Exercise Physiology: Energy, Nutrition, and Human Performance. Philadelphia: Lippincott Williams & Wilkins, 2001.
- ✓ Wilmore, J. H., Costill, D. L., & Kenney, W. L. (2008). Physiology of sport and exercise. Champaign, IL: Human Kinetics.