Lesson 3 addresses the "cardio half" of cardiorespiratory exercise physiology.

**Learning Objectives**

After completion of Lesson 2, the student should be able to:

1. Define the following terms, and be able to use each term appropriately in discussions; for terms that describe quantitative variables, include "textbook" normal values: cardiac output, stroke volume, heart rate, ejection fraction, cardiac cycle, end-diastolic volume, end-systolic volume, diastolic blood pressure, systolic blood pressure, mean arterial pressure.

2. Describe the general anatomical structure of the heart, and the organization of the heart and circulatory system in terms of blood flow throughout the body.

3. State the relationship among blood flow, driving pressure, and resistance. Describe changes in these variables (in semi-quantitative terms) in response to different types of exercise.

4. Discuss preload, afterload, and contractility, including answers to the following questions: What do these refer to in the intact body? How do these affect cardiac output? How do these affect the heart’s work rate?

5. Briefly discuss the permissive vs. causative role of heart rate in increases in cardiac output.

6. Given pertinent data, accurately do calculations using the following: heart rate, stroke volume, end-diastolic volume, end-systolic volume, cardiac output.

7. List the functional categories of blood vessels. Categorize the body’s major groups of blood vessels by function.

8. Discuss the arteriolar resistance vessels, including answers to the following: Why are they called “resistance vessels”? Why are arterioles well-suited for this function? What is the physiological significance of regulation of arterial resistance by arterioles, especially during exercise?
9. Discuss the major hemodynamic differences between volume-overload exercise and pressure-overload exercise.

10. Describe the difference between volume-overload training and pressure-overload training in terms of hypertrophy and functional capacity of the left ventricle.

11. Describe the typical responses of the following variables to increasing VO$_2$ during progressive exercise: cardiac output, heart rate, stroke volume, diastolic blood pressure, systolic blood pressure, mean arterial pressure, total peripheral resistance, arterial-mixed venous oxygen difference, arterial blood oxygen content, mixed venous blood oxygen content.
Introduction to Unit 4 - Lesson 3 (cont.)

Outline of Content

III. Lesson 3 – Cardiovascular Physiology

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Cardiovascular physiology divides naturally into cardiac physiology (study of the function of the heart) and vascular physiology (study of the functions of blood vessels). The overall function of the cardiovascular system, of course, is to circulate blood throughout the body. The heart is the pump that generates the pressure to drive flow. The vascular system is the series of tubes in which blood flows. This division of the overall topic is useful for study, but it must not distract from the essential point that the heart and the vascular system at all times must work together in coordinated fashion. There may be a change in cardiac function (e.g., change in heart rate or stroke volume) or a change in certain blood vessels (e.g., dilatation of arterioles in contracting skeletal muscles), and each can be examined separately. But no such change occurs in isolation; it affects overall blood flow in the circulation in some way.

In intense, dynamic exercise using large muscles, total body blood flow (i.e., cardiac output) has to increase a lot (perhaps 500%) to meet tissue demands. A fundamental point is that changes have to occur in both the heart’s function and in the vascular system to bring about this large increase in blood flow. The changes in either one without the other cause very small increases in blood flow. Only when they change simultaneously can the demand for blood flow be met. So, we will study the heart and the vascular system separately at times, but they are in no way separated functionally.

Following is a list of some of the essential “substances” that are transported from one site in the body to another by the blood.

- gases (e.g., oxygen and carbon dioxide)
- fuels for metabolism (e.g., glucose, fatty acids, triglycerides, amino acids)
- hormones (e.g., insulin, epinephrine, growth hormone)
- water
- electrolytes (e.g., sodium, chloride, potassium)
- substances related to immune responses (e.g., white blood cells, antibodies)
- metabolic end-products or waste-products (e.g., lactic acid, urea, heat)
### Cardiac Physiology

Hemodynamics is the study of the movement of the blood and the forces that affect that movement. A fundamental law that governs flow of any liquid or gas through a tube is Poiseuille’s Law:

- Flow rate is proportional to \( \frac{\text{driving pressure}}{\text{resistance}} \).

I want to emphasize the simple, logical concepts that this law describes:

- Flow occurs only when there is pressure driving it.
- Rate of flow is directly related to driving pressure; the greater the driving pressure, the greater the rate of flow (unless increased resistance offsets the increase in pressure).
- Rate of flow is inversely related to resistance; the greater the resistance, the lower the rate of flow (unless increased pressure offsets the increase in resistance). Indeed, the definition of resistance says that it impedes flow.

### Cardiac Physiology (cont)

The heart is the muscular pump that generates the driving pressure for blood flow. The heart is really two pumps that are in series with each other in the cardiovascular system. The left side of the heart receives the newly oxygenated, arterialized blood from the lungs (via the left atrium) and generates the pressure to pump the blood through the systemic circulation (via the left ventricle). The right side of the heart receives the partially deoxygenated, venous blood from the body’s tissues (via the right atrium) and generates the pressure to pump the blood through the pulmonary circulation (via the right ventricle). The left ventricle must generate much higher pressures to drive blood flow through the systemic circulation than the right ventricle must generate to drive blood flow through the pulmonary circulation. These two pumps could do their respective jobs totally separated from each other, but their being joined as part of a single organ is a more effective design. You should review the basic structure of the heart, as well as the basic physiology of the cardiac cycle.
Three important factors that determine the effectiveness of the ventricles in pumping blood are:

- **Preload.** This term refers to the degree of filling of the ventricle before systole begins. The end-diastolic volume is one indicator of preload. In brief, the greater the preload (i.e., the more the ventricle is filled during diastole, just prior to contraction), the more effective the pumping.

- **Afterload.** The arterial pressure the ventricle must overcome to eject blood out of the heart and into the circulation. This is best indicated by the arterial diastolic blood pressure. In brief, the greater the afterload (i.e., the greater the arterial diastolic pressure), the less effective the pumping. (Note: Individuals who have diastolic hypertension, or high blood pressure, force the left ventricle to continually work harder to overcome this increased afterload.)

- **Contractility.** Recall that with skeletal muscle, contractility means “active development of force.” Contractility means more than that when referring to the heart. Myocardial contractility refers to an inherent, variable capacity of individual fibers for generating force, which is not inherent in skeletal muscle. Increased contractility means that the myocardium can generate greater force and pump more effectively; decreased contractility means that it can generate less force, and it pumps less effectively. Myocardial contractility is not only variable but also subject to regulation. It is affected by different variables. For example, increased sympathetic nervous stimulation and increased heart rate, which both occur during exercise, increase contractility.
Cardiac Physiology (cont.)

During rhythmic, dynamic exercise, involving large muscle groups (e.g., running, cycling), there is usually an increase in preload (better return of venous blood to the heart resulting in greater filling of the ventricle prior to systole) and an increase in myocardial contractility, with little change or even a decrease in afterload. The more-forceful contraction of the ventricle results in a greater fraction of the end-diastolic blood volume being ejected during systole. This is called the ejection fraction. The overall result is an increase in stroke volume, a very important adaptation to the exercise.

**EXAMPLE:**

At rest in a person of average size and fitness, there is about 120 mL of blood in the left ventricle at the end of diastole (end-diastolic volume), and the ventricle ejects about 60% of this volume during systole (ejection fraction); $SV = 120 \text{ mL} \times 0.60 = 72 \text{ mL}$.

If **contractility alone increased**, a greater fraction (e.g., 70%) of the starting volume would be ejected and the stroke volume would increase: $SV = 120 \text{ mL} \times 0.70 = 84 \text{ mL}$.

If there were better venous return of blood to the heart so that the **end-diastolic volume alone increased** (e.g., to 140 mL), the stroke volume would increase: $SV = 140 \text{ mL} \times 0.60 = 84 \text{ mL}$.

If **both of these changes occurred**, as often does during exercise, there would be an even greater increase in stroke volume compared to the resting state: $SV = 140 \text{ mL} \times 0.70 = 98 \text{ mL}$.
The increases in stroke volume that normally occur during dynamic exercise are also normally coupled with increased heart rate to give increased cardiac output.

**EXAMPLE:**

Resting –

- heart rate = 70 bpm; stroke volume = 72 mL
- C.O. = HR x SV = 70 bpm x 72 mL/beat
  - C.O. = 5,040 mL/min = 5.04 L/min

Exercise –

- heart rate = 150 bpm; stroke volume = 98 mL
- C.O. = 150 bpm x 98 mL/beat
  - C.O. = 14,700 mL/min = 14.70 L/min

This is a good place to make a peripheral but important point about the role of increased heart rate in increasing cardiac output. Refer to the relationship: C.O. = HR x SV. This suggests that there is a strong, direct correlation between heart rate and cardiac output, and in general there is, especially during exercise. This correlation and the mathematical relationship suggest a causal relationship, that increasing heart rate will cause an increase in cardiac output. This is not true, however, over a wide range of values. **How can this be?** The mathematical relationship gives the answer: If heart rate changes, stroke volume can change in the opposite direction, offsetting the effect of heart rate on cardiac output. But, just as certainly, the mathematical relationship shows that the largest increases in cardiac output can occur only if both heart rate and stroke volume increase. Thus, the increase in heart rate plays a permissive role rather than a causative role. In other words, increased heart rate is necessary for a large increase in cardiac output, but by itself an increase in heart rate does not cause an increase in cardiac output.

Increases in preload, afterload and contractility come at a price. An increase in any one of these increases the work of the heart and its rate of energy turnover. Cardiac muscle is very dependent on aerobic metabolism. Therefore, increases in preload, afterload and contractility increase the myocardial demand for oxygen, and blood flow through the coronary arteries to the myocardium must increase. Otherwise, myocardial ischemia occurs.

We will study other changes in cardiac function in response to exercise later in this lesson, after studying key concepts of vascular physiology.
Vascular Physiology

Blood vessels may be categorized in different ways. A common categorization is:

- **arteries** of all sizes, from the largest artery (the aorta) to the smallest arterioles. These conduct blood **away from** the heart.

- **veins** of all sizes, from the largest veins (the superior and inferior vena cavae) to the smallest venule. These conduct blood **toward** the heart.

- **capillaries**. The tiniest of blood vessels, with walls only a single cell thick. These function to exchange substances between the blood and the extravascular compartment and tissues.

Blood vessels may also be categorized on the basis of function:

- **Conduction**. All blood vessels have this function, simply conducting blood from one part of the body to another. But this is essentially the only function of the large arteries and veins.

- **Exchange**. Capillaries are the only vessels that have this function, exchanging gases, nutrients and other substances across their walls.

- **Capacitance**. Capacitance is the relationship between change in volume in blood vessels and change in pressure. All blood vessels have this characteristic, but the particular “capacitance function” belongs to the small veins and venules (the “capacitance vessels”). The structure of these vessels gives them the ability to change in volume with little change in pressure. The net result of this is that they make good storage vessels. When a high rate of arterial blood flow to tissues is not needed (e.g., at rest), a relatively large portion of the total blood volume is in these blood vessels; they expand to hold this “extra volume,” and pressure inside them does not increase much. When a high rate of arterial flow is needed (e.g., during exercise), these vessels constrict, reducing the volume of blood in them, again with little change in the pressure inside. This has the beneficial effect of shifting more of the total blood volume to the arterial circulation, enhancing delivery of blood to tissues when they need the increased flow.

- **Resistance**. Resistance refers to the forces or factors that impede blood flow. All blood vessels create some
impede blood flow. All blood vessels create some resistance to blood flow, but some vessels have the specific function of providing variable resistance. These are the arterioles (which are referred to as the “resistance vessels”) and the medium-sized veins.

- Vascular Physiology (cont)

  The role of the resistance blood vessels is very important. At first thought, it may seem absurd to have blood vessels designed to resist blood flow. Why would you want to impede blood flow? The answer is: To provide control of blood flow. If all of the tissues in the body increased their demands for blood flow to maximums at the same time, the total blood volume would not be adequate to meet these simultaneous demands, and the cardiac pump couldn’t generate enough pressure to drive flow to all these tissues at the rates demanded. A version of this actually happens in shock. There is widespread dilatation of blood vessels throughout the body, but the heart can’t generate enough pressure to drive flow to all the tissues. If flow to the brain or other vital organs decreases too much, unconsciousness and even death can result. The inadequate driving pressure in shock is analogous to the situations that sometimes occur in communities in a dry period of the summer in Texas. If everybody tries to water outdoors with a hose at the same time when the water level in the storage tank is low, the rates of water flow through the hoses are low because the driving pressure isn’t enough to handle the demands.

  Arterioles are called the “resistance vessels.” They have the major responsibility for regulating resistance on the arterial side. First of all, arterioles have small diameters, which creates more resistance than the larger diameters of larger vessels. Second, arterioles can change their internal diameters a relatively large amount, thereby regulating their resistance to flow. By regulating resistance in various tissue beds, the arterioles shift arterial blood flow to different tissues in proportion to needs. At the same time, this regulates arterial blood pressure. For example, during running, arterioles will dilate in the active leg muscles, decreasing the resistance so flow can increase to those tissues. At the same time, arterioles will constrict in other tissues, such as the kidneys, the stomach, or even other muscles that are not active. This increases resistance to flow in these tissues, so flow to these tissues is reduced during the period of exercise. Figure 9-19 in the textbook nicely summarizes the changes in blood flow distribution during exercise compared to rest. This distribution is regulated by changes in diameter of the arteriolar resistance vessels. What is presented in Figure 9-19 is one typical example of exercise. Exact distributions differ, depending on the type of exercise, the muscle groups involved, the intensity and duration of exercise, whether there is environmental heat stress, and other factors.
The resistance to venous blood flow provided by the medium-sized veins is not nearly as important to total body hemodynamics as is the resistance of the arterioles. Nevertheless, these vessels provide some regulation of venous return of blood to the heart. During situations when cardiac output must increase greatly (e.g., exercise), it is obviously important that the volume of blood pumped out of the heart be returned to the heart at the same rate; venous return and cardiac must be equal. Venous return is facilitated in these situations by dilatation of the medium-sized veins, reducing the resistance to flow.

What changes take place in the vascular system in response to exercise? This depends very much on the type of exercise. When the exercise involves large muscle groups contracting dynamically (e.g., running, cycling), the major change in blood vessels is dilatation of arterioles in the active muscles, with constriction in some other tissues, as noted previously. In this type of exercise, there is much more dilatation than constriction overall, so the total peripheral resistance decreases greatly (to perhaps 25-30% of the resting value). This change in the vasculature is critical in terms of facilitating an increased cardiac output. Without this big decrease in total peripheral resistance, the cardiac pump would have to generate very high pressure to increase blood flow. The heart actually has to do this in other types of exercise.

In exercise involving contractions of only small muscle groups (e.g., submaximal weightlifting curls with only one arm, cycling with the arms on an ergometer), the arterioles dilate in the active muscles. But they won’t dilate and may even constrict in the inactive muscles, as well as in the GI tract, kidneys and other tissue beds. As a result, the total vasodilatation is relatively small, and there may actually be more constriction than dilatation. So, total peripheral resistance may decrease a little, remain unchanged, or may even increase. As a result, the heart must generate greater pressure for a given cardiac output in this type of exercise. Of course, the cardiac output doesn’t have to be as high in this type of exercise than in exercise with larger muscle groups, because there is less active muscle tissue demanding flow. This is fortunate; otherwise, blood pressure would be much higher in this exercise than in exercise with larger muscles.

There is a category of exercise in which blood pressures are greatly elevated: Exercise with static or nearly static contractions of large muscles (e.g., weightlifting with very heavy weights by large muscle groups; bodybuilding poses). During forceful contractions of skeletal muscles, intramuscular pressure increases greatly. This pressure pushes against the outside of blood vessels and closes many. This creates infinite resistance to flow in these contracting muscles. And, since a large proportion...
Now in these contracting muscles! And, since a large proportion of the total muscle mass is involved, the total peripheral resistance increases greatly. To overcome this resistance and increase blood flow, the heart must generate huge pressures. Systolic blood pressure values of about 500 mm Hg have been measured in highly trained weightlifters during the straining of maximal lifts! This pressure would rupture arteries and probably kill most people. But these athletes have adapted to this over years of training. This provides a great example of the big effect vascular changes can have on overall hemodynamics, even though these changes during weightlifting are imposed on the blood vessels, rather than being initiated by the blood vessels.

Summary of Cardiovascular Responses to Exercise

This section will indeed be a summary. An entire semester of advanced study could be spent just on the cardiovascular responses to exercise. As I have tried to do throughout this course, I will try to address the topics that I think are most important. I will divide my presentation as follows.

For simplicity, I will divide all exercise into two extreme types based on the cardiovascular stress:

- **Volume overload.** Exercise in which the primary challenge to the cardiovascular system is to increase total blood flow (i.e., cardiac output) for more than just a brief period of time.

- **Pressure overload.** Exercise in which the primary challenge to the cardiovascular system is to increase arterial blood pressure.

In all exercise, the primary challenge to the cardiovascular system is to support the metabolic needs of the active skeletal muscles. Therefore, the responses of different variables are similar in all exercise, at least in direction if not in magnitude. For example, heart rate typically increases in response to all exercise, though much more in some than in other exercise. Nevertheless, there are important differences in cardiovascular stresses in volume-overload exercise compared with pressure-overload exercise. This is true with acute exercise, as well as chronic exercise. Also, of course, every type of exercise isn’t just one type or the other. A lot of exercise involves some of both stresses.
### Summary of Cardiovascular Responses to Exercise (cont)

I will summarize responses to acute exercise first, and then I will summarize adaptations to chronic exercise (i.e., training). I will present the typical or average responses of healthy persons. Other responses would be abnormal, but not necessarily pathological. Furthermore, many other influences often interact with exercise to alter the typical response. Such influences include: amount of active skeletal muscle mass, body position, disease (especially cardiovascular and pulmonary diseases), drugs, environmental stress, exercise equipment, mode of exercise, psychological stress, and training status. With all of the possible interacting influences, the cardiovascular response to a given bout of exercise can be complex indeed. I will try to simplify here.

I urge you to refer to the Fick Equation in its various forms as you study the following. Most of the cardiovascular responses to exercise are logical, and most fit with what is expected based on the mathematical relationships expressed in the Fick Equation.

### Cardiovascular Responses to Acute Volume-Overload Exercise

In volume-overload exercise, skeletal muscles contract dynamically and repeatedly throughout the exercise, which is continued for more than a brief period, and aerobic metabolism contributes significantly to the power input during the exercise. Performance depends on continual transport of oxygen to the contracting muscles. VO2 is elevated for a relatively long period of time. Examples of volume overload exercise include walking, jogging, running and cycling for relatively prolonged period. (Swimming also fits this category, but some of the cardiovascular responses differ a little in swimming because of the horizontal body position.)

**Cardiovascular responses:**

**Cardiac output** (C.O.) increases in proportion to increases in VO2. There is a strong, rectilinear (straight-line) relationship between C.O. and VO2. VO2max coincides with maximal C.O.
Heart rate (HR) increases in proportion to increases in VO\textsubscript{2}. HR has a strong, rectilinear relationship with VO\textsubscript{2}, and generally maximal HR coincides with VO\textsubscript{2}max. In fact, this relationship is very useful in clinical and other settings for estimating VO\textsubscript{2} and VO\textsubscript{2}max on the basis of the HR response. You are probably well aware that many people monitor the intensity of exercise on the basis of HR. While the basic relationship between HR and VO\textsubscript{2} is rectilinear under a variety of conditions, many factors (e.g., environmental stress, psychological stress) can affect this relationship, especially at low intensities of exercise. Let me remind here that increases in HR during exercise are necessary for large increases in cardiac output, but the changes in HR are permissive rather than causative.

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Stroke volume (SV) increases during acute exercise. SV increases approximately in proportion to increases in VO\textsubscript{2} up to about 50-60% of VO\textsubscript{2}max, and then changes little, if at all, beyond that. In other words, SV increases as an important part of the increase in C.O., but maximal SV is typically reached at a relatively low exercise intensity. Responses of SV at higher exercise intensities apparently vary among individuals and under different conditions, and this topic is still a focus of research. There is no question, however, that SV normally increases in response to volume overload exercise, and that this is an
essential part of the increase in C.O. that supports an increased VO2. SV increases as a result of increased contractility of the left ventricle, which is evidenced by an increased ejection fraction. Often, there is both increased end-diastolic volume (i.e., greater venous return and filling of the ventricle prior to systole) and decreased end-systolic volume (i.e., the ventricle contracts more forcefully, contracting down to a smaller volume and thus ejecting out a greater volume of blood with each contraction).

Systolic blood pressure (SBP) increases in approximate proportion to changes in VO2; values as high as 250 mm Hg could be normal in certain individuals during exhaustive exercise, but most would not have values this high. Diastolic blood pressure (DBP) typically changes very little during acute, volume-overload exercise. Since mean arterial pressure (MAP) depends on SBP and DBP, MAP also increases in approximate proportion to increases in VO2. But the changes are not large, usually no more than 50% above the resting pressure. Note: Measurement of arterial blood pressures with the standard cuff and stethoscope technique is difficult during exercise, and it is impossible during certain exercise. Furthermore, even valid measurements of pressure with this technique during exercise often differ from pressures measured directly with a transducer in an artery. This is especially true for diastolic pressure. I urge extreme caution in interpreting exercise blood pressures measured in this way, especially but not only when done by an inexperienced technician.
- Cardiovascular Responses to Acute Volume-Overload Exercise (cont)
**Total peripheral resistance** (TPR) decreases during exercise, in proportion to the amount of arteriolar dilatation in active skeletal muscles. This decreased resistance is essential to the increase in stroke volume and cardiac output. TPR is inversely and curvilinearly related to VO\(_2\). The greatest decreases in TPR occur at relatively low exercise intensities, with additional smaller decreases at higher exercise intensities.

Certain changes in a-v \(O_2\) difference reflect pulmonary function, but some changes reflect cardiovascular function. I am including changes in a-v \(O_2\) difference here for completeness.

**a-v \(O_2\) difference** increases with increases in VO\(_2\). The relationship between these two variables is approximately rectilinear. **Arterial hemoglobin saturation** and **CaO\(_2\)** usually do not change, or change only little, over the entire range of exercise intensities (with the exception noted in Lesson 2). This reflects the excellent normal pulmonary function, even during intense exercise. Since CaO\(_2\) changes little, if at all, the increase in a-v \(O_2\) difference is brought about by a decrease in **mixed venous saturation** and **CvO\(_2\)**. These decrease in inverse proportion to increases in VO\(_2\). These decreases reflect primarily the increased rate of diffusion of oxygen from tissue capillary blood into active skeletal muscle fibers. An important aspect of this is the redistribution of blood flow controlled by the arteriolar resistance vessels. The shift of a greater proportion of the total blood flow to active muscle tissue results in a greater amount going to the tissues that are extracting oxygen at a faster rate. Therefore, the mixed venous blood is “biased” toward a lower oxygen content.
Cardiovascular Adaptations to Volume-Overload Training

I have just summarized the typical responses of the cardiovascular system to acute, volume overload exercise. **What changes can be expected if one trains with these types of exercise, that is, does “volume-overload training”?**

We have already dealt with this topic to a certain extent. First, the Principle of Specificity strongly suggests that this training would result in adaptations in some cardiovascular variables to improve functional capacity and/or endurance. Second, we know that VO\(_2\)\text{max} typically increases in response to this type of training, especially in a person who was relatively untrained at the start. And, the Fick Equation tells us that cardiorespiratory changes necessarily occur if VO\(_2\)\text{max} changes. **What are the typical training adaptations?**

A major change, and probably the single most important one in most individuals, is an increase in maximal cardiac output that is secondary to an increase in maximal stroke volume. Several adaptations contribute toward the increase in maximal SV:

- Structurally, the left ventricle hypertrophies so that the internal dimensions increase with little or no change in thickness of the myocardial wall. This allows greater filling of the ventricle during diastole.

- Training usually increases myocardial contractility. This leads to a greater maximal ejection fraction, and perhaps to an increase in maximal mean arterial pressure.
Total peripheral resistance can be decreased even more during exercise after training, which facilitates ejection of blood from the left ventricle.

Blood volume is increased. This “fills up” the vascular system more, enhancing venous return and ventricular filling prior to systole.

Elite endurance athletes have awesome hearts! Whereas maximal SV may be 100 mL and maximal C.O. 15.0-20.0 L/min in a “normal person,” in these athletes maximal SV may be 200 mL and maximal C.O. 40.0 L/min! No wonder they can transport so much oxygen and their tissues can generate ATP at such high rates in aerobic metabolism! One other bit of data about the world’s best endurance athletes that is amazing to me: A maximal ejection fraction of 70% is considered very high. These athletes may have ejection fractions of 90% during intense exercise! That means that the left ventricle is emptying almost completely during each contraction, reflecting a powerful contraction of the ventricle.

Maximal heart rate (maxHR) is typically unchanged as a result of training. Some have suggested that there is an interactive effect of training with age in terms of change in maximal heart rate. You are probably aware that maxHR normally declines with age. The most common estimate of max HR is 220 – age in years, which reflects this trend with aging. Some suggest that maxHR tends to decrease with training in young adults and increase with training in older persons. In other words, that training reduces the normal differences in maxHR due to aging. I’m not sure there is strong research evidence for this.

Even if maxHR does not change with training, there are important training effects on the HR response to submaximal exercise. At any given power output or VO2, HR is lower after training. And C.O. is usually the same, which indicates that stroke volume is higher at a given power output or VO2 after training. This lower heart rate and higher stroke volume at a given C.O. is more economical for the heart.

Maximal a-v O2 difference sometimes increases with volume-overload training, though such increases are usually maximized before VO2max stops increasing. This should not be surprising; there are mathematical limits to increases in a-v O2 difference, unless blood [Hb] increases. And [Hb] does not increase with training; if anything, it decreases, secondary to a greater increase in plasma volume. If [Hb] does not change (or it decreases), changes in a-v O2 difference are dependent entirely on changes in % saturation of Hb. In arterial blood, Hb
is normally almost 100% saturated, so no increase can occur in SaO₂ or CaO₂. That leaves only changes in saturation of the mixed venous blood as a possible mechanism of change in a-v O₂ difference. SvO₂ does decrease some with training, reflecting (a) enhanced blood flow to trained skeletal muscles with improved capillarization and exchange of oxygen, and perhaps (b) even greater redistribution of total blood flow to active muscles during exercise. But SvO₂ can only decrease so far. Mathematically, of course, SvO₂ cannot go below 0%. Physiologically, this value probably cannot go below about 15-20%. If mixed venous SvO₂ were 0%, that would indicate that every bit of oxygen had been removed from every milliliter of blood that was pumped to the peripheral tissues. This is not possible. Nearly all of the oxygen is removed from the blood by active skeletal muscles, but not absolutely all. And some blood is pumped to tissues that require little oxygen during exercise. These tissues remove relatively little oxygen from the blood perfusing them, leaving a relatively large amount in the venous blood. In summary, there is a limit to how low the CvO₂ can go, and this limits maximal a-v O₂ difference.

### Cardiovascular Responses to Acute Pressure-Overload Exercise

Pressure-overload exercise involves contractions of skeletal muscles against maximal or near-maximal resistances. The best example is heavy resistance weightlifting. Episodes of pressure-overload exercise also occur in wrestling and football, for example. These individual bouts of exercise are necessarily very short in duration, because of their intensity, but often many short bouts are done intermittently over a longer time period. Because the individual bouts are short, aerobic metabolism contributes little to the power input during the exercise. Therefore, although oxygen transport and consumption will increase, these are not critical factors during exercise in terms of successful performance. The key vascular difference between volume-overload and pressure-overload exercise is in total peripheral resistance. The widespread vasodilatation in volume-overload exercise leads to a big decrease in TPR. In contrast, TPR is increased in pressure-overload exercise, primarily due to the occlusion of blood vessels within the contracting muscles during the episodes of straining against heavy resistive loads. An increase in cardiac output is needed, as in all exercise, and the neural and other stimuli are present to cause this. But, if C.O. is to increase with the increased TPR, mean arterial pressure has to increase relatively more than the increase in TPR. Thus, the left ventricle must generate very high pressure. Of course, this is true only during the actual periods of exercise, which are brief. But, it is true nevertheless, and no other exercise situation stresses the heart in this way. Further, these exercise bouts are repeated, so the episodes of generating high blood pressure are repeated also. This is pressure-overload
In contrast with volume-overload exercise, during pressure-overload exercise, diastolic pressure increases and systolic pressure increases relatively more.

For a given cardiac output, heart rate is relatively high and stroke volume relatively low in pressure-overload exercise.
Cardiovascular Adaptations to Pressure-Overload Training

In keeping with the Principle of Specificity, the cardiovascular system adapts to chronic pressure-overload exercise to handle the pressure loads more effectively. Two major changes occur. One is hypertrophy of the left ventricle, with thickening of the ventricular wall, with little or no change in the internal dimensions of the ventricle. (See figure.) This is the opposite of hypertrophic changes in response to volume-overload training. The thicker wall means more muscle mass, analogous to the increased cross-sectional area with training-induced hypertrophy of skeletal muscles. Therefore, the ventricle can generate more force and develop greater blood pressure.

Response to Volume Overload Training

Response to Pressure Overload Training

The second change is less well documented, but it almost certainly must take place: strengthening of the walls of arteries, enabling them to withstand the higher blood pressures. These blood vessels are subjected to the high pressures during the exercise, and the Principle of Overload almost guarantees that they will adapt.
You have come to the end of the online content of Unit 4 – Lesson 3, which also is the end of Unit 4. When you want to review the concepts in this lesson, use the Learning Objectives on Page 2 of this lesson. In addition, I suggest you also use the study questions at the end of Chapter 9 in the textbook (omit Question 3 regarding the electrocardiogram). These should be a good study guide. If you can correctly do what the Objectives and study questions ask, you will have mastered the most important concepts in this lesson. Please realize, however, that these do not exhaustively cover all the information in the lesson.

If you are uncertain about any Objective, or if you want clarification or expansion of any point in the lesson, I urge you to start a threaded conference discussion on WebBoard. Other students may have the same concerns, will probably benefit from the discussion, and may have the information you seek. And, of course, feel free to contact me (Dr. Eldridge) for assistance.

Be sure to check the Announcements Page to see whether there is a specific WebBoard or other assignment associated with this lesson.