



## Physiological effects of exercise on the cardiopulmonary system

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### **Basic cardiopulmonary physiology**

In order to appreciate the effects of exercise on the cardiopulmonary system, it is important to know the basic physiological principles of each system. It is common practice to view the cardiovascular system as the pulmonary circulation, composed of the right heart pump and the lungs, and the systemic circulation, in which the left heart pump supplies blood to the systemic organs (all structures except the gas exchange portion of the lungs) [1]. The left and right sides of the heart pump an equal volume of blood each minute. The amount of blood pumped is known as the cardiac output, and is approximately five to six liters per minute in resting individuals.

The cardiac output must respond to a metabolic challenge, such as exercise, in order to provide the body with the nutritional needs required for a given workload. Changes in either the heart rate or stroke volume have an effect on cardiac output. The greatest influence on the heart rate comes from the autonomic nervous system, composed of the sympathetic nervous system, which has a positive chronotropic effect, and the parasympathetic nerve system, which has a negative chronotropic effect. Stroke volume increases as a result of an increase in venous return caused by the pumping action of exercising muscles. The Frank-Starling law states that stroke volume increases as cardiac filling increases. In addition, the sympathetic nervous system also has an effect on stroke volume, and thus cardiac output, by increasing cardiac muscle-cell contractility and causing a more forceful ejection during systole.

The main goal of the respiratory system is to provide oxygen to the tissues and remove carbon dioxide from the body. Increased muscle activity develops during exercise, and in order to maintain the demands of aerobic metabolism, there must

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be an increase in oxygen delivery. There must be adequate airflow and gas exchange by the respiratory system to maximize arterial oxygenation. Variables such as tidal volume, respiratory rate, distribution of ventilatory volume, chest-wall mechanics, altitude, inspired and alveolar oxygen pressures, red blood cell volume, pulmonary blood flow distributions, pulmonary capillary transit time, and others clearly contribute to overall effective arterial oxygenation, however [2].

There are four major functional events in respiration: (1) pulmonary ventilation, which means an inflow and outflow of air between the atmosphere and the lung alveoli; (2) diffusion of oxygen and carbon dioxide between the alveoli and blood; (3) transport of oxygen and carbon dioxide in the blood and body fluids to and from cells; and (4) regulation of ventilation and other facets of respiration [3]. When describing pulmonary ventilation, it is helpful to divide the air in the lungs into four volumes and four capacities (Fig. 1). The minute respiratory volume is equal to the respiratory rate times the tidal volume and is normally about 6 L/minute. The rate at which new air reaches the alveoli, alveolar sacs, alveolar ducts, and bronchioles is referred to as alveolar ventilation. It is equal to the respiratory rate times the amount of new air that enters the alveoli and gas exchange areas with each breath.  $\dot{V}_A = \text{Freq} \times (V_T - V_D)$ , where  $\dot{V}_A$  is the volume of alveolar ventilation per minute, Freq is the frequency of respiration per minute,  $V_T$  is the tidal volume, and  $V_D$  is the physiologic dead space [3]. The alveolar ventilation is a major factor for determining the amount of oxygen and carbon dioxide in the alveoli.

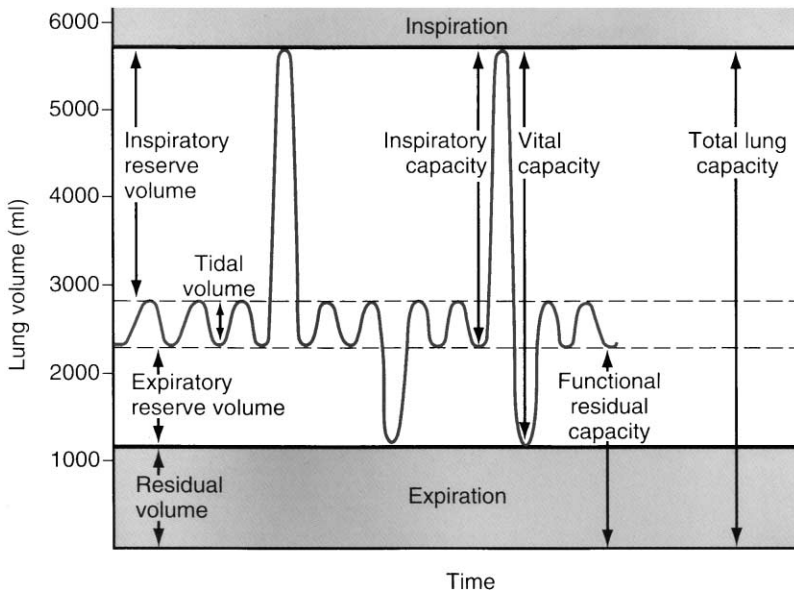


Fig. 1. Diagram showing respiratory excursions during normal breathing and during maximal inspiration and maximal expiration. (From Guyton AC, Hall JE. Pulmonary ventilation. In: Textbook of medical physiology. 10th edition. Philadelphia: WB Saunders; 2000. p. 432–43; with permission.)

## Acute effects of exercise

Exercise can generally be divided into dynamic (endurance) or static (power) exercise [4]. Most sports activities involve a combination of the two exercise types to varying degrees, and although this article focuses on one type or the other, understand that a significant overlap can exist. Walking, running, and swimming are examples of dynamic exercise, whereas hand grip and power lifting best represents static exercise. Weight lifting is largely a static exercise; however, components of dynamic exercise are present when one considers circuit training and high repetitions. Cycling is an example of an activity in which there is a combination of the two types of exercise, with dynamic exercise being performed by the legs and static exercise by the arms. The cardiovascular and pulmonary systems will be discussed separately, but it is important to remember that the two systems work in an integrated fashion during exercise to meet the metabolic demands.

### *Dynamic exercise*

#### *Cardiovascular adaptations*

In dynamic exercise there is a linear relationship between oxygen consumption and exercise intensity up to the maximal amount of oxygen an individual can consume [5]. Factors influencing the oxygen consumption ( $\dot{V}O_2$ max) are best represented by the Fick equation  $\dot{V}O_2 = CO \times a-vO_2$ , where CO is cardiac output and a- $vO_2$  is the arteriovenous oxygen difference. Increases in either CO or a- $vO_2$  can subsequently increase oxygen consumption and improve performance. Maximal dynamic exercise results in a four to six fold increase in cardiac output, a threefold increase in heart rate, and a twofold increase in stroke volume [6].

As previously mentioned, cardiac output will increase with increases in either heart rate or stroke volume. A linear relationship exists between cardiac output and oxygen uptake. Cardiac output during exercise is largely related to an increase in the heart rate [7]. The central nervous system acts early in exercise by decreasing vagal tone and causing a rapid increase in heart rate. These centrally mediated changes are due to cerebral mechanisms, and reflex changes caused by activation of mechanical receptors in the skeletal muscles that are being activated [8]. As exercise continues, the sympathetic nervous system increases activity levels, allowing for further increases in heart rate.

Early in exercise, cardiac output increases secondary to an increase in stroke volume. This is caused by an increased venous return from pumping skeletal muscles. The major causes of increased stroke volume during exercise in humans are increased cardiac contractility and increased venous return to the heart [5]. The overall increase in stroke volume from rest to peak exercise is typically 30% to 40% [9,10]. During dynamic exercise, there is an increase in venous return, aided by a redistribution of blood flow and venous volumes from the viscera to active skeletal muscles [5]. Relative blood flow remains constant to the brain, is reduced by half to the kidney and splanchnic organs, and is increased to the heart

and exercising skeletal muscles by four and ten times respectively [1,6,11]. The increase in blood flow to the cardiac and skeletal muscle produced by exercise is called exercise hyperemia [5].

Vasodilation and decreased vascular resistance in active skeletal muscle cause a decrease in total peripheral resistance. There is an increase in systolic and mean arterial pressure and the diastolic pressure decreases slightly [6]. The changes in blood pressure are not linearly related to workload, because blood pressure is influenced by the magnitude of muscle mass being exercised [12]. For example, dynamic exercise performed with the arms will cause a greater increase in arterial pressure when compared with that performed by the legs, because there is less of a decrease in total systemic vascular conductance, based upon the difference in muscle mass. Although we see a large decrease in systemic vascular resistance during dynamic exercise, the mean arterial pressure actually increases, due to the larger effect of an increased cardiac output.

Maximal  $\dot{V}O_2$  is a measure of aerobic capacity and is determined by the maximal oxygen delivery and oxygen extraction by working muscle [13]. A major determinant of maximal oxygen consumption is the arteriovenous oxygen difference. Arterial oxygen content depends on oxygen-carrying capacity and the ability of the respiratory system to load oxygen in the pulmonary capillaries [5]. During exercise, splenic constriction allows for a slight increase in arterial oxygen-carrying capacity by increasing the hematocrit level. The increase found in the arteriovenous oxygen difference comes from the decreased mixed venous oxygen content. The mixed venous oxygen content decreases as the intensity level of the exercise increases. This is secondary to the redistribution of cardiac output from tissues that extract small amounts of oxygen, such as the liver and kidney, to tissues that extract large amounts of oxygen, such as cardiac and active skeletal muscle [5]. The oxygen content of the blood in the coronary veins and skeletal muscle is low, and with intense exercise the venous return to the heart is coming from these tissues. The mixed venous content decreases as an increased amount of blood is returned to the heart from exercising muscle. As a result, oxygen consumption will go up, based upon the principles of the Fick equation. With prolonged, intense exercise, oxygen consumption increases until maximal  $\dot{V}O_2$  is reached. If the intensity exceeds maximum oxygen consumption, increased work can be performed for a period of time via anaerobic processes that result in excess lactic acid production.

The circulation of blood during upright exercise involves a two-pump system: a central cardiac pump, and a peripheral skeletal muscle pump responsible for systemic venous return [14]. It is known that a peripheral pump is a necessary component for sustaining cardiac output during exercise [15]. The skeletal muscle pump promotes venous return by compressing intramuscular venous vessels, causing blood to be propelled centrally and preventing the increased heart rate and cardiac contractility from significantly lowering central venous pressure. In addition, the effect of muscular contractions reduces intramuscular venous pressure, which creates an increased arterial-venous gradient for inflow during periods of muscle relaxation [14].

Table 1  
Alterations in the Fick equation variables from rest to maximal exercise

	VO <sub>2</sub> (mL/min)	=	HR (beats/min)	x	SV (L/beat) (EDV-ESV)	x	(aO <sub>2</sub> - vO <sub>2</sub> ) (mL/L) (mL/L)
Subjects							
Untrained at rest	280	=	80	x	0.070 (0.120 - 0.050)	x	200 - 15
Untrained during exercise	3080	=	200	x	0.110 (0.155 - 0.045)	x	200 - 60
Trained during exercise	3458	=	190	x	0.130 (0.165 - 0.035)	x	200 - 60
Endurance-trained during exercise	5595	=	190	x	0.190 (0.220 - 0.030)	x	200 - 45

From Brown DD. Pulmonary response to exercise and training. In: Garrett WE Jr, Kirkendall DT, editors. Exercise and sport science. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 117–134; with permission.

Dynamic exercise causes an increase in cardiac output, due to improved pump function and increased venous return. These physiologic alterations are necessary to meet the metabolic demands of exercising muscles. A vast majority of the blood flow is diverted to exercising skeletal muscles and away from visceral organs. Vasodilation of the skeletal muscles causes a decrease in total peripheral resistance, but mean arterial pressure is increased due to an increase in cardiac output. These impressive adaptations in the components of the Fick equation from the resting state to physical exercise are shown in Table 1. The maximum heart rate does not vary between untrained and trained subjects, but impressive changes in  $\dot{V}O_2$ , stroke volume, and venous oxygen content are found among the endurance-trained group. The relationship of heart rate, stroke volume, blood pressure, and arteriovenous oxygen difference to oxygen uptake during progressive dynamic exercise is summarized graphically in Fig. 2.

### *Pulmonary adaptations*

The response of the healthy pulmonary system to the physiologic requirements of exercise is typically one of impressive efficiency. Respiratory rate has a four to five fold increase during exercise, tidal volume increases five to seven fold, and minute ventilation can increase 20 to 30 times over resting airflow values [2]. Maintaining arterial oxygenation (PaO<sub>2</sub>) and O<sub>2</sub> content is a difficult task for the lung, because the mixed venous return has a decreased O<sub>2</sub> content, a rising PCO<sub>2</sub> level, and a decreased transit time in the capillary beds from the increased cardiac output [16]. Despite these alterations, arterial oxygenation is maintained, and a threefold increase in arteriovenous oxygen difference occurs, as venous oxygenation decreases because of greater cellular extraction of arterial oxygen. The net effect of these and other physiological adaptations to exercise is an amazing 20 to 25 fold increase in oxygen consumption over resting values [2].

Ventilation is the process of providing sufficient airflow through the respiratory passageways, filling gas-exchange areas in an attempt to accommodate the cellular needs of oxygen delivery and carbon dioxide removal [2]. Ventilation increases over tenfold with maximal  $\dot{V}O_2$ , and generally occurs in a two-

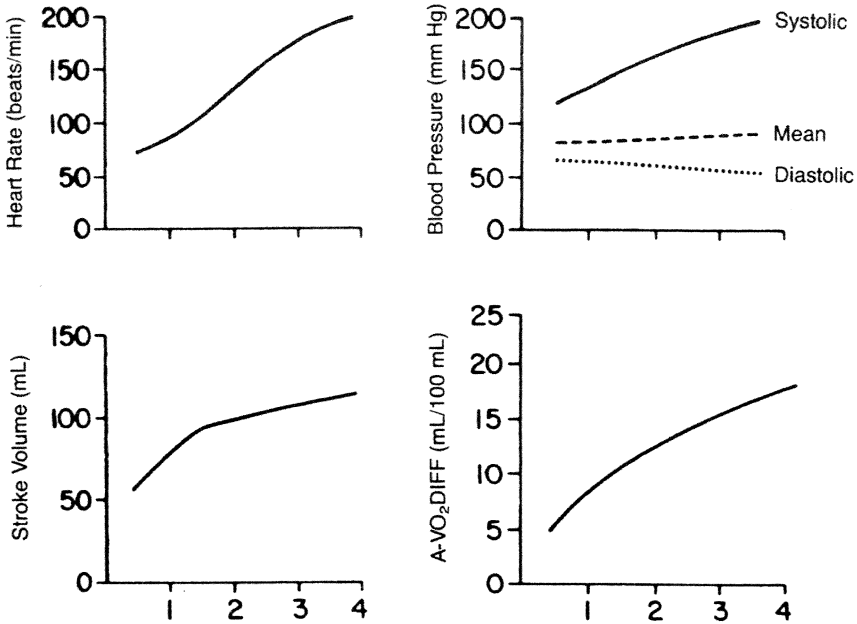


Fig. 2. Relationship of heart rate, stroke volume, blood pressure, and arteriovenous oxygen difference to oxygen uptake during progressive dynamic exercise. (From Crawford MH. Physiologic consequences of systematic training. *Cardiol Clin* 1992;10:210–13; with permission.)

phase response, with a less steep increase until about 50% of the max  $\dot{V}O_2$ , and thereafter the ventilation increases more steeply [16,17]. Ventilation is a function of the respiratory rate (RR) and the tidal volume ( $V_T$ ) ( $\dot{V}_E = RR \times V_T$ ). The increases in minute ventilation ( $\dot{V}_E$ ) that occur in people during incremental exercise are a result of increases in both tidal volume and respiratory frequency [17]. Once the tidal volume reaches approximately 50% to 60% of the individual's vital capacity, the tidal volume ceases to increase, and further increases in minute ventilation occur secondary to increases in the respiratory rate [18]. The net effect on minute ventilation is an astonishing 30 to 40 fold increase over resting airflow values, with airflow rates approaching and exceeding 200 liters per minute during maximal exercise in highly trained athletes [2].

In the average person, resting ventilation typically averages 8 to 10 liters per minute, but at maximal exercise can reach 100 to 130 liters per minute. The maximum ventilatory capacity is commonly estimated by multiplying the forced expiratory volume in one second ( $FEV_1$ ) by 40, and in the average person is approximately 200 liters per minute. The ventilatory reserve is determined by the ventilatory ceiling minus the ventilation at maximal exercise [16]. A moderately trained subject with a normal  $FEV_1$  of 5 liters per minute who is at maximum exercise, would have a reserve of 80 liters per minute based on ventilatory reserve (200 L/min–120 L/min). Because ventilation can be increased further, it is

commonly held that ventilatory function does not limit exercise performance [18]. One exception where ventilation is a limiting factor occurs when one considers the elite athlete.

The respiratory system's adaptation to the cellular demands during exercise is immediate, with an initial increase in ventilatory response occurring before exercise actually begins [19,20]. There are three different phases to the ventilatory response for pulmonary minute ventilation from rest to exercise. Table 2 summarizes the ventilatory changes before, during, and after exercise.

With regard to gas exchange, the alveolar ventilation ( $\dot{V}_A$ ) increases with carbon dioxide production. This occurs in a proportional manner at low work loads (<65% of max  $\dot{V}O_2$ ) but once the anaerobic threshold is crossed,  $\dot{V}_A$  increases more than is required to hold  $PCO_2$  constant [18]. With exercise, alveolar ventilation increases more than the increase in blood flow to the lung, and as this occurs, the concentration of oxygen within the alveolus ( $PaO_2$ ) rises. The ratio of alveolar ventilation to cardiac output increases from 0.9 at rest to 3.5 at the conclusion of heavy exercise [16]. During exercise, the dead space actually increases from around 200 ml to a maximum of 400 ml, but because the tidal volume increases three to four fold (500 ml to 1500–2000 ml), the dead space to tidal volume ratio ( $VD/V_T$ ) falls from around 33% at rest to 20% with exercise [16]. This allows for a greater volume of the lung to participate in gas exchange.

The increase in airflow is typically linearly related to the metabolic demand of increased oxygen consumption and carbon dioxide elimination [2]. Once exercise intensity exceeds 55% to 65% of maximal aerobic capacity, the increased ventilation is related more to the physiologic need of carbon dioxide elimination

Table 2  
Ventilatory changes before, during, and after exercise

Phase	Change	Controlling mechanism
1. Rest	—	Central and peripheral influencing intrinsic pattern established by the medulla
2. Before exercise	Moderate increase	↑ Central command (cerebral cortex)
3. During exercise		
a. Immediate	Rapid increase	↑ Central command and possibly ↑ neural stimuli to medulla caused by activation of muscle/joint receptors
b. Mid	Steady-state or slower increase	Central or peripheral chemoreceptors reacting to ↑ $P_{CO_2}$ and ↓ pH in blood or csf
c. End	Continued or rapid (hyperventilation) increase	Same as above, with possible additional input from ↑ blood potassium, ↑ blood catecholamines, ↑ body temperature, and ↑ central command
4. Recovery		
a. Immediate	Rapid decrease	↓ Central command
b. Later	Slower decrease toward rest	↓ Input from central and peripheral chemoreceptors as $P_{CO_2}$ and pH normalize

From Foss ML, Keteyian SJ. Fox's physiological basis for exercise and sport. 6th edition. Boston: WCB McGraw-Hill; 1998. p. 170; with permission.

than to oxygen consumption [19,21]. As it becomes more difficult to sustain energy levels with aerobic energy systems, a greater reliance on anaerobic glycolysis for metabolic energy production emerges. The net result is a buildup of by-products (eg, lactic acid) that need to be buffered in order to maintain homeostasis [21]. Buffering via the bicarbonate system yields nonmetabolically produced carbon dioxide in addition to the metabolically produced amount (catabolism of acetyl-coenzyme A in the Krebs cycle). Regardless of the source for carbon dioxide production, respiratory centers of the central nervous system respond to the regulatory feedback by increasing pulmonary minute ventilation that mirrors the change in carbon dioxide production [2,19].

In order to accommodate the physiological adaptations at the respiratory membrane during exercise, pulmonary capillary blood volume expands to three times its resting value, and its increase is linearly related to the changes in pulmonary blood flow [22]. The pulmonary capillary transit time decreases from 0.75 seconds at rest to 0.4 seconds during exercise. This adaptation does not significantly affect the exchange of oxygen and carbon dioxide in the pulmonary capillary bed [22]. The overall effect of the respiratory system adaptations is to maintain arterial oxygen and carbon dioxide concentrations at or near resting values throughout a wide range of exercise intensities.

### *Static exercise*

#### *Cardiovascular adaptations*

The physiological effects of static exercise are different from those seen with dynamic exercise. The central cardiovascular effects of static exercise training primarily reflect the heart's response to increased afterload or blood pressure, whereas training responses to dynamic exercise are the result of a volume load on the heart. There is a marked increase in intramuscular pressure during static contraction, which leads to an impairment of blood flow to the muscle secondary to arterial compression by muscle fibers [23]. Once muscle tension exceeds 70% of maximal voluntary contraction (MVC), there is a complete vascular occlusion [24]. Energy requirements during static muscle contraction are met by either partial or complete conversion to anaerobic metabolism [23,25].

With static muscle contraction, the mean arterial pressure, heart rate, and cardiac output all increase, and in general the heart rate and blood pressure increases seen during static exercise exceed those of dynamic exercise when duration, intensity, and active muscle mass are similar [5]. Arterial blood pressure changes can be dramatic, with systolic pressure increasing as high as 250 to 450 mm Hg [26]. This increase in blood pressure is necessary to maintain blood flow to exercising muscles whose arterial supply is being compressed. Cardiac stroke volume, ejection fraction, and systemic vascular resistance remain essentially unchanged during static exercise [27]. The increase in cardiac output during static exercise is largely due to an increase in heart rate, because the stroke volume does not increase appreciably until the immediate recovery period. Stroke volume is maintained, however, because of an increase in venous return immediately after

the initial contractile effort squeezes blood out of the exercising muscles and sympathetic nervous system activation causes increased cardiac contractility [6].

Maximal oxygen consumption is unchanged during static exercise, because a large component involves anaerobic metabolism [28,29]. Studies have shown either that maximal oxygen consumption of power lifters is not increased relative to age-matched and sex-matched controls [30], or is increased slightly [31]. Arteriovenous oxygen difference is not changed during static exercise, because the muscle contraction impedes access of blood. After muscle contraction, arteriovenous oxygen difference increases as a result of increased flow to the muscles and increases in muscle oxygen extraction in the postexercise bed [6].

In summary, the cardiovascular effects of static exercise include an increased heart rate, increased cardiac output, increased arterial pressure, and an increased sympathetic drive. There is a greater increase in diastolic, systolic, and mean arterial pressure than that observed with dynamic exercise. This leads to a higher afterload on the heart and a higher cardiac workload during static exercise. Following static exercise, a postischemic hyperemic response occurs in which there is a transient increase in  $\dot{V}O_2$  and  $CO$ , due to vasodilation within the muscle bed in order to pay back the oxygen debt that incurred during exercise [32]. A summary of the effects of static exercise is depicted in Fig. 3.

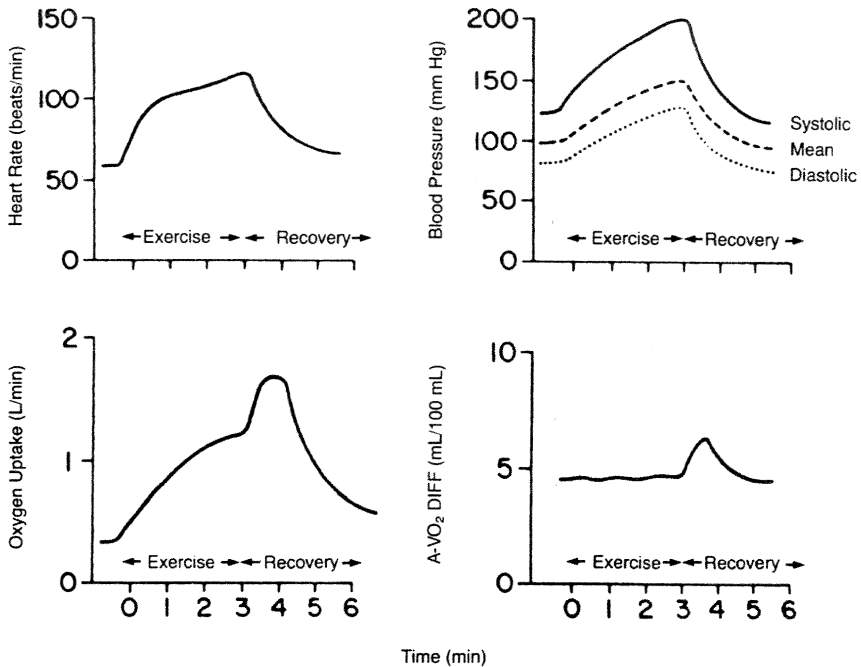


Fig. 3. Heart rate, oxygen uptake, blood pressure, and arteriovenous oxygen difference during static exercise and recovery. (From Crawford MH. Physiologic consequences of systematic training. *Cardiol Clin* 1992;10:210–13; with permission.)

### *Pulmonary adaptations*

As far as the pulmonary responses to static exercise, all responses are the same as those observed for light- to moderate-intensity, short-term, submaximal exercise. Some of the changes that are observed include: minute ventilation increases rapidly then levels off; tidal volume increases rapidly then levels off; respiratory rate slowly increases then levels off; ventilatory dead space decreases; alveolar ventilation increases; venous oxygen concentration decreases rapidly; venous carbon dioxide concentration shows a slight linear increase; and arterial carbon dioxide concentration is level then decreases rapidly. There is no change in alveolar or arterial oxygen concentration [33].

## **Chronic effects of exercise**

As with acute changes, the specific alterations depend on the type, intensity, age, duration of exercise, and original fitness level. There are morphological changes that occur with both dynamic and static exercise. Adaptations in the respiratory system are not as extensive as those seen in the cardiovascular system.

### *Dynamic exercise*

#### *Cardiovascular adaptations*

From a physiological standpoint, aerobic fitness and performance have traditionally been linked to the body's maximal capacity for utilizing oxygen ( $\dot{V}O_{2max}$ ), which in turn is determined by peak cardiac functional capacity [34]. Maximal oxygen consumption is the best indicator of aerobic exercise capacity, and is highly reproducible.  $\dot{V}O_{2max}$  reflects the maximal capacity of the cardiovascular system to increase blood flow, as reflected in a substantial rise in cardiac output, to the exercising skeletal muscle and heart. This adaptation is made in order to meet the metabolic demand and the maximal capacity of the exercising skeletal muscle to extract oxygen from the nutrient blood flow, as reflected in an increase in arteriovenous oxygen difference [35]. Dynamic training for 30 to 60 minutes at 60% to 70% maximal  $\dot{V}O_2$ , three to four times a week, is required to obtain a significant training effect [36]. In normal young men, the maximal  $\dot{V}O_2$  is 45 ml/kg/minute and it can increase to 60 ml/kg/minute with training, and up to 80 ml/kg/minute in world-class dynamically trained athletes [13].

There is an increase in cardiac output secondary to increased stroke volume, and an increase in  $a-vO_2$  due to improved systemic oxidative capacity and vascular conductance [37]. The maximal heart rate is not different between dynamically trained athletes and untrained individuals, so the increase in cardiac output comes from an increased stroke volume. In fact, the heart rate itself is decreased at rest and during submaximal training, making the increased cardiac output in training primarily a cardiac response to the increased stroke volume [38]. In a study of endurance athletes, it was found that stroke volumes of endurance trained athletes'

increases progressively to maximum with no plateau. In addition, to augment stroke volume even at high heart rates, endurance athletes rely on enhancements in ventricular emptying, and especially in ventricular filling [39].

The decreases in heart rate at rest and at submaximal  $\dot{V}O_2$  are the primary cardiovascular alterations seen in dynamically trained athletes. There is an increase in parasympathetic tone in trained athletes and not a decrease in sympathetic activity or circulating catecholamine levels [6]. Once the heart rate exceeds 130 beats per minute during exercise there is no longer a vagal effect, and further increases in heart rate come from sympathetic stimulation and circulating catecholamine levels [40]. In trained athletes, a lower circulating level of catecholamines is found at any point of submaximal  $\dot{V}O_2$ , but the responsiveness of the sinoatrial node does not change before and after training [6,38].

The decrease in sympathetic nervous system activity in trained individuals is possibly due to an attenuated reflex heart-rate response to myocardial stretch [41]. Training allows for improvements in cardiac output by increasing stroke volume. A lower heart rate is then needed to achieve a given workload and  $\dot{V}O_2$ . Training certain muscle groups may have an effect on exercise heart rate. If training involves primarily the lower extremities, exercising the upper extremities will elicit the same heart rate response as in an untrained individual [6,36].

Studies of the effects of dynamic exercise on blood pressure have shown mixed results with regard to changes in systolic and diastolic levels. Kelley and colleagues did a meta-analysis in 1995 in order to examine the effects of aerobic exercise on resting systolic and diastolic blood pressure among normotensive adults [42]. For all trial comparisons (randomized controlled trials, controlled trials, and no nonexercising controls), systolic blood pressure was reduced by 4.4 mm Hg, and diastolic blood pressure was reduced by 3.2 mm Hg. These exercise groups also showed a small decrease in body weight, fat, and mass, and changes of 14% in maximum oxygen consumption and 8% in resting heart rate. The effect that these parameters had on resting blood pressure was not discussed. Fagard reported that aerobic exercise training decreased blood pressure along a continuum, with systolic blood pressure (SBP) and diastolic blood pressure (DBP) decreased by an average of 3 mm Hg in normotensive subjects, 6 to 7 mm Hg in subjects with high normal blood pressure, and 8 to 10 mm Hg in subjects with documented hypertension [43]. Although it is commonly believed that dynamic exercise lowers blood pressure, studies that do not support this finding are common [44,45].

A recent analysis was done to determine the aftereffects of dynamic exercise on ambulatory blood pressure [46]. It is believed that ambulatory blood pressure more accurately reflects the blood pressure a person maintains during daily activity [47]. The meta-analysis study raised concerns about several experimental flaws within the various studies, and called for more rigorous investigations into the manner. Nevertheless, the final conclusion was that dynamic exercise studies indicate that day, night, and 24-hour ambulatory blood pressure is reduced after exercise among sedentary, white, middle-aged men and women who are overweight and not taking medications. The reductions were most notable in hypertensive individuals and less so in the normotensive population.

### *Pulmonary adaptations*

In the respiratory system, the changes that occur with prolonged exercise are far less than those described in the cardiovascular system. The respiratory system responses are similar between trained and untrained individuals. This fact is not surprising, given the reserve found within the respiratory system, even without prolonged training. With the capacity to increase ventilation by 20 to 40 fold and of respiratory rate and tidal volume to increase 4 to 6 fold from resting values, the respiratory system is designed for maximal function without the requirement of adaptations through prolonged physical training [2]. It is generally believed that the pulmonary system is not the limiting factor in exercise in the majority of trained and untrained individuals. Studies examining wrestlers, football players, distance runners, and other types of athletes have been performed that showed no significant differences in pulmonary-function responses to prolonged exercise when compared with matched controls for body weight, gender, and age [48,49]. The exception came with swimming, in which greater lung volumes and capacities were found in subjects in comparison with those in matched land-based trained and untrained controls [48].

In contrast, in the elite, highly trained individual, with significant improvements to their cardiovascular and musculoskeletal systems from long-term training and minimal pulmonary adaptations, the respiratory system may become the rate-limiting step in the oxygen consumption equation [2]. The elite athlete develops a different response to the alveolar-to-arterial gas exchange than that of healthy untrained and moderately trained individuals. In the elite athlete, there may be anatomic and physiologic constraints to the respiratory system that results in a widening of the alveolar and arterial oxygen pressure, producing an exercise-induced hypoxemia [22,50]. The mechanism and prevalence for such a finding is not yet entirely known and research has yet to effectively prove such a theory.

The change in pulmonary ventilatory dynamics has been shown to be a consistent adaptation to prolonged training. Pulmonary minute ventilation is increased during maximal exercise and decreased during submaximal exercise intensities. Tidal volume increases both at rest and during submaximal and maximal exercise. The respiratory rate is reduced at rest and submaximal exercise, but increases during maximal exercise when compared with matched controls [2]. During maximal exercise, the demand for airflow is extremely high and can best be met through an increase in both respiratory rate and tidal volume, yielding the large airflow rates observed in highly trained athletes [51]. Finally, respiratory muscle strength and endurance have been shown to develop secondary to prolonged physical activity [52].

### *Static exercise*

Although dynamic exercise leads to increases in  $\dot{V}O_{2\max}$  and cardiac output, the general belief is that static exercise does not effect these parameters to any significant degree [31,53]. It has been demonstrated that the overall cardiovascular hemodynamic response to static exercise is similar in trained

and untrained individuals [54]. The improvements in performance in static exercise are seen through adaptations of targeted muscle groups. There are no quality studies concerning pulmonary adaptations to static exercise.

Research does exist that has demonstrated cardiovascular adaptations to static exercise. Studies of untrained healthy subjects who underwent an 8- to 10-week regimen of weight training and circuit training were found to have decreases in resting heart rates of five to seven beats per minute [31,55]. Other studies have noted a decrease in resting systolic and diastolic blood pressure with static exercise training [31,56,57]. Following termination of training, systolic and diastolic blood pressures started to return to pretraining levels within 4 weeks. Overall findings comparing weight lifters or body builders with untrained controls suggest that chronic weight lifting does not appear to have an effect on resting blood pressure, and more importantly, does not induce a resting hypertension [27,58,59].

Although the magnitude of cardiovascular training effects is lower in static exercise than in purely dynamic exercise, static exercise does appear to have some physiological effects on the cardiovascular system. Strength training that entails the use of frequent repetitions and moderate weight has a more effective impact upon the cardiovascular system. In this case a larger dynamic component may influence cardiovascular changes. The cardiovascular adaptations may include small decreases in resting heart rate and possible decreases in systolic and diastolic blood pressure; reductions in sympathetic nerve activity [60,61], blood pressure, and heart rate response to the same absolute workload; and increases in muscle capillary density and blood flow during static contraction [62,63].

### *Cross training*

Cross-training has become more popular over the last decade and is now an integral part of the workout routine for athletes at all levels. Exercise leads to central and peripheral adaptations that are proportional to the amount of work being performed. As already discussed, dynamic exercise of large muscle groups leads to increases in maximal oxygen uptake and cardiac output, and static exercise maximizes peripheral and skeletal muscle training effects. Thus, the type of exercise and magnitude of the muscle groups involved largely determine the difference between the results of static and dynamic exercise training on the cardiovascular system [6].

Cross-training is an attempt to maximize the effect of both modes of training. It has been demonstrated that static exercise will not enhance the cardiovascular response to dynamic exercise [54]. Static exercise, however, will allow for improvements in power and strength within muscle groups that may allow for improved performance, despite no appreciable effect on maximal  $\dot{V}O_2$ .

On the other hand, it is believed that dynamic training of athletes primarily using static training does alter cardiovascular response to static exercise [59]. The increase in left ventricular volume and increased stroke volume at a lower heart rate allows a greater tolerance of the pressure load on the heart during static

exercise, and thus improve static performance [6]. There does not appear to be an effect on long-term adaptations with the sequence of cross-training. Collins and Snow found no difference in maximal  $\dot{V}O_2$  or muscle strength whether endurance exercise or strength training was done first [64].

### **Morphological changes from exercise**

Resistance training stimulates hypertrophy with normal cavity dimensions, whereas endurance training stimulates hypertrophy and cavity dilatation [65,66]. Most sports and training programs use both types of exercise to some degree, and as a result there is a range of morphological changes. Growth occurs in the normal heart muscle that matches the workload imposed on the ventricle to maintain a constant relationship between systolic cavity pressure and the ratio of wall thickness to ventricular radius [67]. These alterations in cardiac structure are determined by the law of Laplace, in which wall tension ( $T$ ) depends upon both intraventricular pressure ( $P$ ) and ventricular radius ( $r$ ) ( $T = P \times r$ ). In endurance exercise the load on the heart is primarily of the volume type, whereas in resistance exercise the load is primarily of a pressure type.

As mentioned, endurance training stimulates hypertrophy and cavity dilatation. With chronic volume loading, the increase is primarily in ventricular end-diastolic cavity size, with a proportional increase in septal and free wall thickness to normalize myocardial wall stress. These cardiac adaptations allow for the effects that have been discussed to develop. An increased diastolic volume allows the ejection of a given stroke volume, with less myocardial shortening and lower frictional and tension energy losses [67]. Increased myocardial mass is needed to compensate for efficiency losses created by greater wall tension caused by an increased end-diastolic volume.

Endurance trained athletes have an increase in left ventricular end-diastolic dimension that is present even when corrections are made for weight and body surface area. Studies of the heart in several sports have shown consistent increases in cavity size, but these findings usually fall within the normal range at about 5.7 cm [68]. Some studies do demonstrate values higher than this, but a well-known study evaluated 947 athletes and found only 38 who had left ventricular end-diastolic dimension of greater than 6 cm [69]. Echocardiographic studies have shown that athletes have a 10% increase in left ventricular end-diastolic dimension when compared with matched sedentary controls, and due to the cube relationship of dimension to volume, this represents a 33% greater end-diastolic volume [68]. Even when comparing widely differing age groups (24 to 32 years old and 60 to 82 years), the cardiovascular changes in response to endurance training are similar [70].

Resistance training primarily induces a pressure overload that causes a thickening of the septum and posterior wall without left ventricular cavity enlargement [65]. Power exercise causes brief bursts of cardiac output against high systemic vascular resistance, and lacks the volume loading that is seen in

endurance exercise. Most studies show that when correction for body surface area is made, weight lifters and shot putters have normal or nearly normal left ventricular end-diastolic dimensions [71].

In endurance athletes, the increase in left ventricular cavity size is compensated with an increase in the thickness of the posterior wall and septum. Again, the values obtained typically fall within the normal range or just beyond. Average values for accumulated data show an increase in septal thickness to 1.04 cm (14% greater than matched controls) and posterior wall to 1.06 cm (19% greater than normal) [68]. Examination of posterior wall thickness in the study by Pelliccia and colleagues revealed a frequency distribution of only 1.7% out of the 947 athletes with values greater than 1.3 cm. The greatest values of wall thickness in this study came from athletes such as rowers and cyclists with a mixed endurance and resistance regimen. Hypertrophy seen in hypertrophic cardiomyopathy is usually asymmetric or regionally distributed in some way; whereas in athletes, even with relatively pronounced hypertrophy, the changes are symmetric, with only a small variation between segments [69]. Hypertrophy as a result of physiologic causes is not accompanied by fibrosis and other structural changes seen in pathologic states [72].

Greater absolute values for wall thickness have been obtained in athletes who undertake predominantly power training, which leads to symmetric left ventricular hypertrophy from pressure overload (judo wrestlers, mean 1.4 cm, maximum 1.7 cm; shot putters, maximum 1.6 cm) [65,67].

Left ventricular mass is also found to be increased, along with left ventricular end-diastolic dimension and wall and septal thickness. This finding is consistently demonstrated in highly trained athletes, and remains elevated after correction for body surface area and weight. Pooled data show that highly trained athletes show an average increase in mass of 45% beyond that of controls, despite only small increases in cavity size and wall thickness [69].

A resting bradycardia is another marker of the cardiovascular effects of training. The bradycardia in athletes is mainly mediated by increased parasympathetic and reduced sympathetic input during the resting state [73]. The result of increased parasympathetic input can lead to sinus pauses with junctional escape rhythms, first-degree heart block, Wenckenbach-type second-degree heart block, atrial or ventricular ectopic activity, and in some cases, atrial fibrillation [74,75].

## **Gender influences**

In dynamic training, an increase in maximal  $\dot{V}O_2$  is one of the physiological adaptations that occurs due to increased cardiac output and a- $\dot{V}O_2$ . Gender influences the acute response to exercise and the results of training. There are no differences in maximal  $\dot{V}O_2$  until puberty, when girls demonstrate 20% to 30% less maximal  $\dot{V}O_2$  than boys for the same amount of training [76]. Possible factors that may be involved include lower body weight and lean body mass for girls, and a lower hemoglobin level.

In recent years, there have been cross-sectional studies investigating cardiac morphologic changes in women athletes who participated in distance running and swimming. Data showed that left ventricular cavity dimensions increased by 5% to 15% and wall thickness increased by 4% to 25% [77,78]. In addition, longitudinal studies have found a mild enlargement in left ventricular cavity size (2% to 7%) and wall thickness (4% to 12%) as a consequence of endurance training in previously unconditioned women [79]. Pelliccia and colleagues investigated 600 highly trained female athletes from 27 different sports. They found that the magnitude of increase in cavity dimension (6%) and wall thickness (14%) identified in these elite female athletes relative to those in sedentary matched controls was similar to that reported in elite male athletes [69]. The type of sport had similar effects on females, with endurance athletes (cross-country skiing, cycling) showing the greatest effects on left ventricular cavity size and wall thickness.

Finally, elite female athletes who were compared with a large group of males of the same ethnic origin, with similar intensity of training and duration of athletic conditioning, showed substantially lower absolute left ventricular wall thickness (–23%), cavity dimension (–11%), and mass index (–31%) [80]. The differences in absolute left ventricular dimensions are related to several factors, which include: (1) lower body size and lean body mass; (2) different gender-related hemodynamic responses to exercise, such as lower absolute cardiac output and systolic blood pressure during peak exercise [81–83]; and (3) differences in endocrine milieu (greater availability of androgenic-anabolic hormones in men for cardiac protein synthesis in response to exercise).

### **Effects of physical inactivity**

The cardiovascular system adapts to environmental challenges such as exercise in a manner designed to maintain hemodynamic homeostasis. The cardiovascular adaptations to dynamic exercise have been discussed at length, and many of the changes that occur with exercise are reversible. Left ventricular hypertrophy (LVH) develops with endurance training and has been shown to be entirely reversible after removal of the inducing stimulus and physical activity [84]. Animal studies have found that removal of the training stimuli resulted in regression of cardiac hypertrophy, with approximately 60% of the heart weight declining in the first two weeks of inactivity [85]. In humans, similar results have been found. Cessation of endurance exercise training resulted in a biphasic response characterized by an initial rapid reduction of left ventricular end-diastolic dimension, reaching a plateau after two weeks of detraining, and a significantly more gradual decline in left ventricular wall thickness [86]. Generally, the decrease in left ventricular diameter and posterior wall thickness is proportional. The regression of LVH occurs both in moderately and highly trained endurance athletes [86]. An investigation of highly trained Olympic athletes several weeks after they had stopped or reduced training load showed a

significant reduction in left ventricular wall thickness and mass (24% and 22%), but without significant changes in left ventricular diameter [84]. The consensus remains that removal of training stimulus and cessation of training in moderately trained and elite endurance athletes can result in reduction of left ventricular mass, size, and wall thickness, with loss of physiologic left ventricular remodeling [84,86]. Although the mechanism responsible for these structural adaptations to physical inactivity is unknown, it is plausible to attribute the regression of cardiac hypertrophy to a reduction in plasma volume that occurs after cessation of training [87].

In a study of normal human subjects who were restricted to 21 days bed rest there was a decrease in maximal oxygen consumption, cardiac output, and stroke volume (due to decreased venous return) [88]. In addition, physical inactivity causes a significant reduction of  $\dot{V}O_{2\max}$  in both moderately trained and elite endurance athletes. Athletes who had exercised vigorously for years showed a 16% decline in  $\dot{V}O_{2\max}$  after three months of detraining (7% in the first 3 weeks then 9% over the next 5 weeks) [89].

The endurance athletes studied by Coyle et al also showed an approximately 8% decrease in cardiac output and an 11% decrease in stroke volume during maximal exercise within the first three weeks, but no significant further reductions thereafter [89]. Finally, maximal heart rate and heart rate during submaximal exercise at a given absolute exercise intensity increase after cessation of training [90].

Regression of peripheral adaptations, such as  $a\text{-}vO_2$ , usually occurs later than the central adaptations and reductions in cardiac output and stroke volume. After the third week of physical inactivity,  $a\text{-}vO_2$  begins to decrease, showing a gradual decline, with the lowest value (9% decrease) only after 12 weeks [89]. Based on these findings, it appears that the reduction of aerobic exercise capacity occurs first from a rapid decline in left ventricular size and filling, stroke volume, and cardiac output during maximal exercise, followed by a more gradual decrease that is a consequence of reduced  $a\text{-}vO_2$  [35].

## Summary

The cardiopulmonary adaptations made to dynamic and static exercise show the amazing ability of the human body to alter physiological processes in order to meet metabolic demands. A remarkable partnership that allows individuals to maximize their abilities and obtain goals exists between the cardiovascular and pulmonary systems. The adaptations of the cardiopulmonary system depend heavily on the intensity, duration, frequency, and type of exercise being performed. Although most of this article examined dynamic and static exercise separately, the majority of individuals train using a combination of these two modes. The overall adaptations will vary with the chosen degree of each exercise mode. An appropriate exercise program allows for improvements in the cardiopulmonary system that help develop and maintain fitness levels.

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