Effects of Exercise on Cardiac Output
(A Review)

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Abstract
This paper reviewed cardiac functions during exercise; cardiac output (CO) is an indicator for fitness. It is the total volume of blood pumped per minute by each ventricle. It’s normally increases during exercise. The measurement of cardiac output was first proposed by Fick, he published an equation in 1870, and it calls for the measurement of the contents of oxygen or CO$_2$ in pulmonary arterial and systemic arterial blood. Simultaneous measurements of oxygen consumption ($V_O_2$) and CO during exercise which allows for the calculation of arterial – venous oxygen difference. Methods used in determining CO are essential in combination with cardiopulmonary exercise testing (CPET). During exercise, muscles receive 66% of the CO, but kidneys only receive 3%. Number of studies have shown the effect of maximal and sub maximal exercise on the cardiovascular responses of HR, SV, and CO. Cardiovascular drift is characterized by a continues, gradual increase in (HR) after 10 min of moderate – intensity aerobic exercise, despite maintenance of a constant work rate. Furthermore, many studies have revealed the response of SV to exercise. Traditionally, it has been agreed that during incremental exercise, SV plateaus at 40% of $V_O_2$max. However, recent research studies have documented that SV progressively increase $V_O_2$max in both trained and untrained subjects. An increase in exercise causes a proportional increase in oxygen consumption $V_O_2$max by skeletal muscles. The increase demand for oxygen causes an increased CO, which increases blood flow to skeletal muscles and leads to a greater amount of oxygen extraction from the blood. The paper recommends that additional research be conducted to evaluate the importance of blood
volume, or other mechanisms, that influence the stroke volume response to exercise in healthy and sick individuals.

**Key words:** Effect, Exercise, Cardiac output

**Introduction**

Effective cardiac output (CO) is an indicator for fitness (Dimkpa, 2009). Thus, a cardiovascular fit person is expected to have an increased cardiac output. Cardiac output is referred to as the total volume of blood pumped per minute by each ventricle. Cardiac output plays an important role in meeting the oxygen demand for work. As the work increases, the CO increases in a nearly linear manner to meet the increased oxygen demand, but only up to the point it reaches its maximal capacity.

The common formula for determining cardiac output is by multiplying heart rate with SV. That is CO = HR×SV (Michigan State University, 1998). Heart rate (HR) refers to the number of times the heart beats each minute; and stroke volume (SV) refers to the amount of blood, measured in milliliters, ejected by each ventricle with each beat (Prabhu, 2007; Lohrey, 2010).

A number of comprehensive papers from scientific organizations (American Psychological Society, 1997; MSU, 1998) and numerous research articles (Stark-Leyva, Beck, & Johnson, 2004; Macklem, 2006; Tol, Huijsmans, Kroo, Schothorst, & Kwakkel, 2006; Miller, Smith, Hemaner, & Dempsey, 2007; Sports Fitness Advisor, 2012) summarized the effect of exercise on cardiac output, with the consistent conclusion that exercise can significantly affect cardiac output, particularly during high intensity exercise bouts. Increase in cardiac output is due primarily to an increase in heart rate and stroke volume (SFA, 2012). Cardiac output increases proportionally with exercise intensity - which is predictable from understanding the response of HR and SV to activity. At rest the cardiac output is about 5.5 L/min. the values is typically for a resting, average sized adult. During intense exercise, this can increase to 20-40 L/min. as the exercise progresses in relation to the intensity.
Objectives of the Review
The objective of this paper is to review and synthesize current literature on the effect of exercise on cardiac output. It specifically focuses on how exercise affects heart rate, stroke volume cardiac output.

Rationale for the Review
Heart failure is defined as a syndrome in which cardiac output is insufficient to meet metabolic demands. Gandhi, Powers, Fowle, Rankin, Nomeir, Kitzman. (2004). Research evidence has shown that effective cardiac output is an indication for absolute fitness in an individual. Therefore, the rationale for the review is that effects of diseases and physical non performance upon exercise can be measured by comparing cardiac output of healthy individuals and that of patients.

Measurement of Cardiac Output
Different techniques with different levels of invasiveness can be used to measure the CO which includes indirect flick, electrical bioimpedance and pulse contour methods. The measurement of cardiac output was first proposed by Fick, who published his equation in 1870. Fick's calculation called for the measurement of the contents of oxygen or CO₂ in pulmonary arterial and systemic arterial blood (Laszlo, 2004; Truijen, Lieshout, Wesselink, & Westrhof, 2012). Different techniques with different levels of invasiveness can be used to measure the cardiac output which includes indirect Fick method (Laszlo, 2004; Geerts, Aarts, & Jansen, 2010), electrical bioimpedance (Holmes & Williams, 2010; Gujjar, 2010), pulse contour method, (Schattenkerk, 2009; Stover et al., 2009; Bogert, Wesseling, Schraa, Lieshout, Bajm, & Goudever, 2010; Martina, 2010; Bogers, Burg, Schepp & Klein, 2009), acetyline rebreathing and doppler ultrasound methods (Agostoni, Cattadori, Apostolo, Contini, Palermo, Marenzi, & Wasserrman, 2005).
A new Australian study (University of Queensland, 2012) has confirmed the accuracy of a modern non-invasive cardiac output monitor that can replace a 40-year-old standard in this field. The collaborative paper by researchers at the University Of Queensland School Of Medicine, the
Florey Neuroscience Institute and the University of Melbourne compared the current accepted method of measuring cardiac output with a non-invasive accurate ultrasound monitor called USCOM. The current gold standard cardiac monitor is the pulmonary artery catheter (PAC), which involves insertion of a catheter into a patient's neck or groin. The catheter is then positioned in their arteries through the heart before heating or cooling the blood. The researchers surgically implanted accurate measurement devices onto the great cardiac arteries, and then monitored their cardiac output using USCOM and PAC at rest and as medications were introduced. They further found that USCOM had a 1 percent error compared with the surgical device, while the PAC error was 17 per cent, and that USCOM was six to eight times more accurate than the PAC for detecting changes associated with the common drugs used in cardiovascular management.

**Measurement of Cardiac Output in Heart Failure**

Giving that the average total blood volume is about 5.5L, nearly all the blood is pumped around the circuit on each minute. During periods of strenuous exercise in well trained atheles, the CO may reach 35L/min; the entire blood volume is pumped around the circuit almost seven times per minute. Even sedentary, untrained individual can reach CO of 20 – 25L/min during exercise. The volume of blood each ventricle pumps as function of time, usually expressed in liters per minute, is called the CO. In a steady state, the CO following through the systemic and the pulmonary circuits is the same. The CO is calculated by multiplying the heart HR the number of beats per minutes per minute and the SV, the blood volume ejected by each ventricle with each beats. $\text{CO} = \text{HR} \times \text{SV}$.

For example, if each ventricle has a rate of 72 beats/min and ejects 70mL of blood with each beats, the CO is $\text{CO} = 72\text{beats/min} \times 0.07\text{L/beats} = 5.0\text{L/min}$. (Eric, Hershel, and Kevin, 2014).

In heart failure (HF), measurement of CO during exercise is important in defining severity of the disease (Schwaiblmair et al., 2012). Simultaneous measurements of oxygen consumption ($\text{VO}_2$) and CO during exercise
allow calculation of arterial-venous oxygen difference \([C(a-v)O_2]\), and plotting these three variables together, to discriminate exercise limitations due to altered left ventricle pump function from those due to other causes, including muscle enzyme deficiency and deconditioning (Agostoni et al., 2005).

**Cardiac Output and Cardio-respiratory Exercise Testing**

This computerized test provide a breath – by analysis of respiratory gas exchange and cardiac function at rest and during a period of exercise, the intensity of which increased incrementally until symptoms limit testing. Information on airflow, \(O_2\) consumption, \(CO_2\) production, and heart rate are collected and used for computation of other variables, Exercise was done on a treadmill or on a bicycle ergometer, the ergometer may be preferable because work rate can be directly measured and the test was affected less by obesity. This test primarily determines whether patients have normal or reduced maximal exercise capacity (\(VO_2\) max), and if so, suggest probable causes. CPET was used to define which organ system contributes to a patient’s symptoms of exertion dyspnea and exercise intolerance and to what extent. The test was also more sensitive for detecting early or subclinical disease which is less comprehensive test usually done at rest, the example include:

Assessment of exercise capacity for disability evaluation, Preoperative assessment, Determination of whether dyspnea symptoms result from cardiac or pulmonary problems in patients who have disorders of both organ systems, Selection of candidates for cardiac transplantation, Assessment of prognosis in selected disorders (e.g. heart diseases, pulmonary vascular disorders, and cystic fibrosis) (Pepin, Saey, Laviolette and Maltais, 2007).
Rest to Exercise Cardiac Output
During exercise (Figure 1), muscles receive 66% of the cardiac output, but the kidneys only receive 3%. At rest, the liver has the highest percentage of cardiac output (27%), while the muscles only receive 15%, only 15% of the resting cardiac output goes to muscle, but during Olympic cross country skiing, the muscles receive 60% to 70% of the CO (MSU, 1998). The American Council on exercise reports that pulse rate and in turn, CO increases during exercise because the heart is trying to meet up with the demands of muscles needs for increased level of fuels. This fuel comes in the form of blood and oxygen, and is required for the muscle to continue the exercise. (ACE, 2015).

Figure 1:

![Cardiovascular Responses to Exercise](image)

Source: MSU, 1998

Cardiovascular Responses to Exercise
A number of studies have been carried out (Stark-Leyva et al., 2004; Macklem, 2006; Miller, Smith, Hemaner & Dempsey, 2007) confirming the effect of maximal and sub maximal exercises on the cardiovascular responses. The following sections discuss the responses of HR, SV and CO to exercise.
Heart rate Response to Exercise

Cardiovascular drift is characterized by a continuous, gradual increase in heart rate (HR) after 10 min of moderate-intensity aerobic exercise, despite maintenance of a constant work rate (Mikus, Earnest, Blair, & Church. 2009). In their findings, Heffernan, Fahs, Shinsako, Jae, & Fermhall (2007) suggested that resistance exercise training increases heart rate after exercise (Myers, Gjuja, & Neelaqaru, 2009). A number of studies investigated whether HR response during exercise test independently predicts cardiovascular disease (CVD) mortality, and whether exaggerated blood pressure response to heart rate during exercise is predictive of future hypertension independent of other important risk factors. One study by Savonen, Lakka, Laukkanen, Halonen, Rauraramaa, Salonen, & Rauramaa (2012), found that a blunted HR increase at 40–100% of maximal workload was associated with increased CVD mortality. Ohuchi, Suzuki, Yasuda, Arakaki, Echigo, & Kamiya, (2000) investigated the difference in HR recovery after exercise between children and young adults. The findings suggest that the early phase of HR recovery after light to severe exercise is influenced by the cardiac parasympathetic nervous activity at rest, and that the greater central cholinergic modulation of HR in children than in young adults may be responsible in part for children's faster HR recovery after exercise. Another study by Miyai, Arita, Miyashita, Morioka, Shiraishi, & Nishio. (2002) suggested that exaggerated full blood pressure response to heart rate during exercise predicts CHD and future hypertension independent of other important risk factors. The study further supports the concept that blood pressure measurement during exercise test is a valuable means of identifying normotensive individuals at high risk for developing hypertension (Miyai, Arita, Miyashita, Morioka,Shiraishi & Nishio, 2002).

Stroke Volume Response to Exercise

Many studies investigated the responses of stroke volume to exercise (Alonso, 2008; Yu & Bil, 2010). Traditionally, it has been accepted that
during incremental exercise, stroke volume plateaus at 40% of \( \text{Vo}_2\text{max} \). However, recent research has documented that stroke volume progressively increases \( \text{Vo}_2\text{max} \) in both trained and untrained subjects. (O’Meagher, Munoz, Alison, Younq, Tanous, Celermajor & Puranik, 2012). Gledhill, et' al (2015). Support the hypothesis that trained subjects show an increased SV during moderate level of exercise, whereas untrained subjects shows a plateau at an early phase of exercise with the same exercise lord. Several lines of evidence indicate SV increase to between 91% and 100% of \( \text{VO}_2\text{max} \) in endurance athlete. Krip, Gledhill, Jamnik, and Warburton. (2014). In contrast, there are doubts to SV during MLE in moderately trained subjects, with an SV plateau at sub – maximal exercise intensity, Spriet, Gledhill, Froeses, and Wilkes. (2004). This evidence shows that a high level of physical fitness may be required to obtain increase SV during MLE.

**Cardiac Output Response to Exercise**

An increase in exercise (muscle work output) causes a proportional increase in oxygen consumption (\( \text{VO}_2\text{ max} \)) by skeletal muscles. The increased demand for oxygen causes an increased CO, which increases blood flow to skeletal muscles and leads to a greater amount of oxygen extraction from the blood. Elevated CO during steady-state exercise is regulated by the increase in skeletal muscle blood flow and venous return to the heart, whereas the increase in heart rate appears to be secondary to the regulation of CO (MSU, 1998).

The effects of exercise training of low and high intensity on resting blood pressure, cardiac output, and total peripheral resistance were studied by (American Physiological Society APS (1997) in sedentary, low, and high-intensity exercise-trained spontaneously hypertensive rats (SHR). Exercise training was performed on a treadmill for 60 min, 5 times per week for 18 weeks, at 55% or 85% resting heart rate. They concluded that low-intensity exercise training, in contrast to high-intensity exercise training, reduced heart rate, which in turn decreased cardiac output and, consequently, attenuated hypertension in SHR (APS, 1997).
To investigate the role of heart rate and peripheral vasodilatation in the regulation of CO, (Bada, Svendsen, Secher, Saltin & Mortensen, 2012), measured central and peripheral hemodynamics in 10 healthy male subjects’ rats during steady-state exercise. Their results demonstrate that the elevated CO during steady-state exercise is regulated by the increase in skeletal muscle blood flow and venous return to the heart, whereas the increase in heart rate appears to be secondary to the regulation of CO.

**Influence of Age and Sex on Exercise Cardiac Output**

With increasing age, exercise cardiac output was found to be greater despite the fact that there was no such trend in oxygen uptake; in consequence, exercise arteriovenous oxygen difference decreased with age. These trends were observed in both sexes, though the age effects were apparent a decade earlier in men (Vizgirda, Wahlaer, Sondgeroth, Ziolo, & Schwertz, 2002). In addition, in men, the heart rate was lower but the stroke volume higher with increasing age. By contrast, no age effect on exercise pulse rate was recorded in women. When the sexes were compared, exercise cardiac output was higher in women of the younger two decades (20 to 39 years), a difference which was not apparent in subsequent decades (Lang, Karlin, Haythe, Mlin, & Mancini, 2009; Bhella, Prasad, Heinicke, Hastings, Zadeh, Huet, Pacini & Levine, 2011).

The stroke volume response to incremental exercise to VO\textsubscript{2}max may be influenced by training status, age, and sex. For endurance trained subjects, the proposed mechanisms for the progressive increase in stroke volume to VO\textsubscript{2}max are enhanced diastolic filling, enhanced contractility, larger blood volume, and decreased cardiac afterload (Vella & Robergs, 2005). Most of the studies (Mackem, 2006; Miller, Hemaner & Dempsey, 2007; Bada, Svendsen, Secher, Saltin & Mortensen, 2012) on cardiovascular and respiratory responses to exercise were conducted on animals. Whether these findings apply to humans remain uncertain because of the
large differences in cardiovascular anatomy and regulation. Perhaps the greatest difference between humans and quadrupeds is that the directionality of the hydrostatic column is reversed (Miller, Smith, Hemaner, & Dempsey, 2007) that is, during exercise, about 70% of the circulating blood volume is below the heart in humans, whereas 70% of the circulating blood volume is above the heart in the exercising dog.

**Concluding Remark and Recommendations**

Available data substantiated the effect of exercise on cardiac output, with the consistent conclusion that exercise significantly affects cardiac output, particularly during high intensity exercise bouts. It is recommended that additional review is needed to evaluate the importance of blood volume, different techniques to measure cardiac output, and an investigation into the differences in HR recovery after exercise between children and young adults, healthy and unhealthy individuals. In addition to other mechanisms that influence the stroke volume response to exercise.

**References**


