

# Cardiovascular Responses to Exercise





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## Introduction

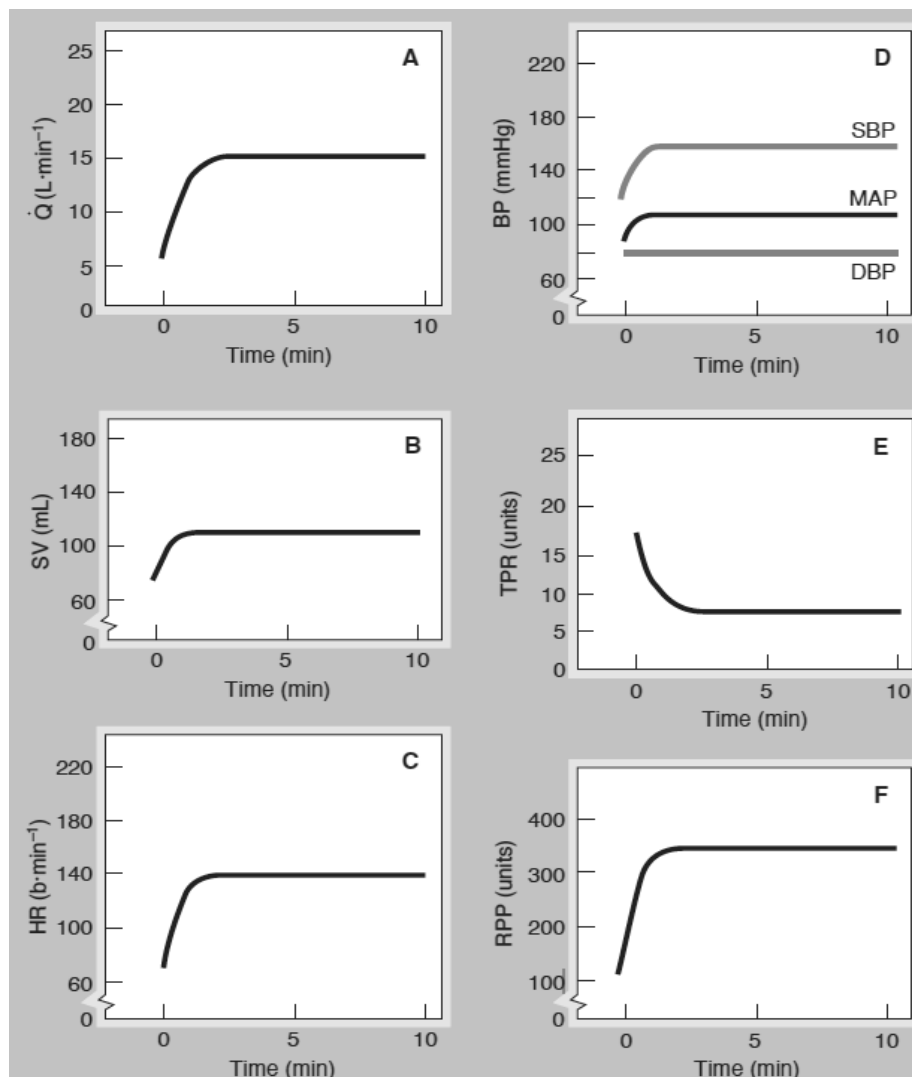
All forms of human kinesis require an expenditure of energy that is above resting values, with much of this energy coming from the use of oxygen. To supply the working muscle tissues with oxygen, the cardiovascular and respiratory systems work collectively. This e-book explains the cardiovascular responses to dynamic aerobic activity, static exercise, and dynamic resistance exercise. Importantly, minimal consideration is given here to short-term, high-intensity anaerobic exercise since this mode of activity is normally implemented to stress the metabolic system.

## Cardiovascular Responses to Aerobic Exercise

When we engage in aerobic exercise our body requires more energy resulting in more oxygen (consider that the term aerobic means “with oxygen”) than static or dynamic resistance exercise respectively. How much oxygen is required is contingent principally on the intensity of the activity and its duration. This e-book classifies exercises as short-term (5–10 minutes), light (30–49% of maximal oxygen consumption,  $VO_{2max}$ ) to moderate (50–74% of  $VO_{2max}$ ) submaximal exercise; long-term (>30 minutes), moderate to heavy (60–85% of  $VO_{2max}$ ) submaximal exercise; or incremental exercise to maximum (increasing from ~30% to 100%  $VO_{2max}$ ).

## Short-Term, Light to Moderate Submaximal Aerobic Exercise

**Figure 1** illustrates the general cardiovascular responses to short-term, light to moderate submaximal aerobic exercise. It is important to understand that the magnitude of each variable’s change is contingent on the work rate or load, environmental settings, and the client’s genetics and physical fitness level. At the start of light- to moderate-intensity exercise, cardiac output ( $Q$ ) initially increases to plateau at a steady-state (**Figure 1A**). Cardiac output levels off within the initial two minutes of exercise, reflecting that  $Q$  is sufficient to transport the oxygen required to support the metabolic requirements of the activity. Cardiac output increases due to an initial increase in stroke volume (SV) and heart rate (HR) (**Figure 1C**); both plateau within two minutes.



**Figure 1.** Cardiovascular Responses to Short-Term, Light to Moderate Submaximal Aerobic Exercise. **A.** Cardiac output (Q). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Blood pressure (SBP, MAP, and DBP). **E.** Total peripheral resistance (TPR). **F.** Rate-pressure product (RPP).

During exercise of light to moderate submaximal aerobic exercise, the cardiorespiratory system can sustain the body's metabolic requirements; hence, this mode of exercise is frequently termed **steady-state or steady-rate exercise**. During steady-state exercise, energy provided aerobically is balanced with the energy needed to perform the exercise. The plateau in cardiovascular variables (see **Figure 1**) suggests that a steady state has been attained. Stroke volume (SV) increases quickly at the onset of exercise due to an increase in venous return, thereby increasing the end-diastolic volume (EDV) (preload). The increased preload stretches the



myocardium (the heart muscle) resulting in a forceful contraction, as described by the Frank-Starling law of the heart.

Contractility of the myocardium is likewise increased by the sympathetic nervous system, which is initiated during physical activity and exercise. The increase in the EDV and the reduction in the end-systolic volume (ESV) contribute to the increase in the SV during light to moderate dynamic exercise (Poliner et al., 1980). Heart rate increases at the onset of the activity because of parasympathetic withdrawal. However, as exercise continues, additional increases in HR are a result of sympathetic nervous system activation (Rowell, 1986).

Systolic blood pressure (SBP) increases in a means comparable to that of cardiac output (i.e., an initial increase followed by a plateau once a steady state is achieved [Figure 1D]). The upsurge in SBP results from increased Q. SBP would be higher if it was not for the resistance reductions, thereby offsetting the increase in Q. When blood pressure (BP) is assessed intra-arterially, diastolic blood pressure (DBP) does not alter. However, when it is measured by auscultation, it either does not alter or may only reduce marginally. Diastolic blood pressure remains somewhat constant due to peripheral vasodilation, which facilitates blood flow to the working muscles. The slight increase in SBP and the insignificant change in DBP produce the mean arterial pressure (MAP) to rise only marginally, following a similar pattern of SBP.

Total peripheral resistance (TPR) decreases due to vasodilation in the active muscles (Figure 1E). This vasodilation results mainly from the effect of local chemical elements (i.e., lactate, K<sup>+</sup>, etc), which reflect increased metabolism. The TPR can be calculated using the following equation below:

$$TPR = \frac{MAP}{\dot{Q}}$$

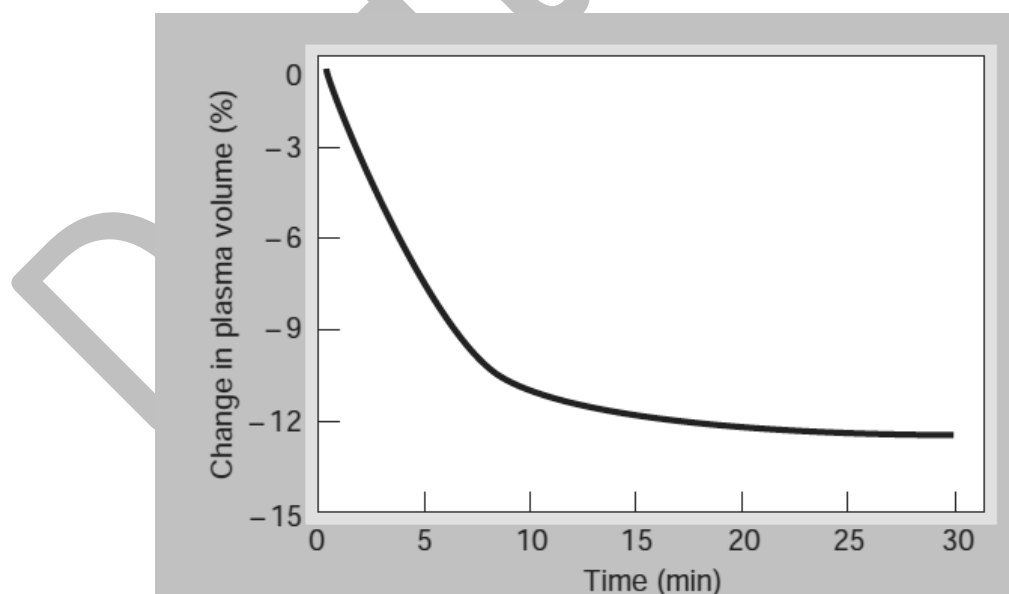
The reduction in TPR has two critical effects. Firstly, vasodilation of the vessels supplying the active muscle produces diminished resistance that leads to increased blood flow, thus increasing the availability of oxygen and nutrients. Secondly, the



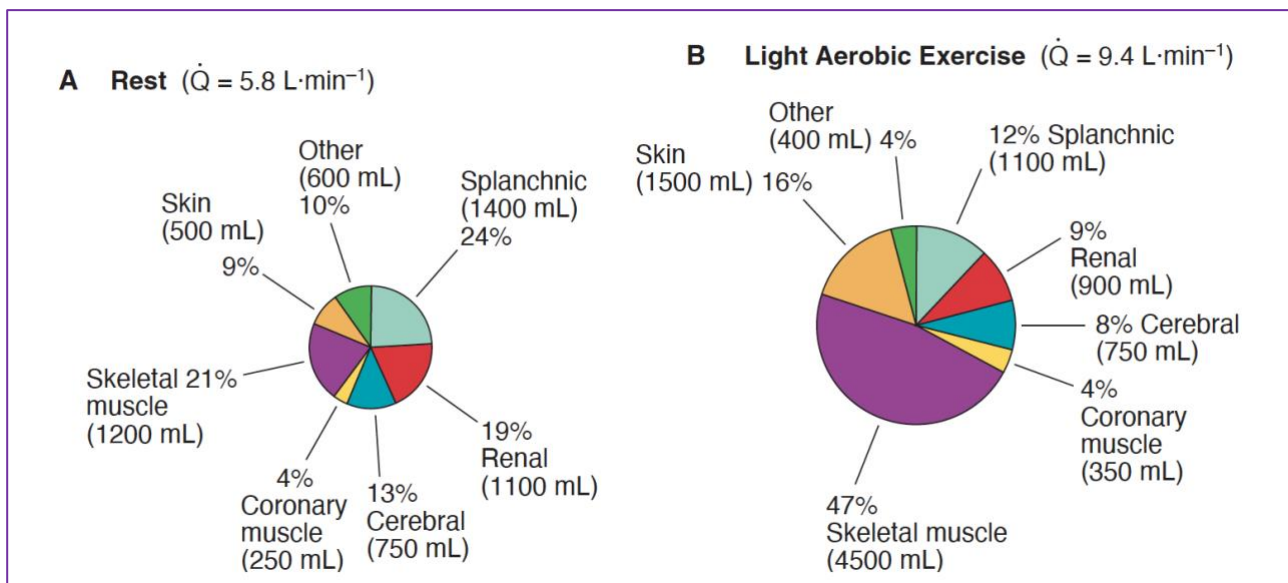


decreased resistance refrains MAP from increasing dramatically. The increase in MAP is determined by the relative changes in Q and TPR. Since Q increases more than resistance decreases, MAP increases marginally during dynamic aerobic exercise. Myocardial oxygen consumption increases during dynamic aerobic exercise because the heart must do more work to increase cardiac output to supply the working muscles with additional oxygen. The rate pressure product (RPP) increases due to increases in the HR and the SBP. This increase reflects the greater myocardial oxygen demand of the heart during exercise (**Figure 1F**).

Blood volume decreases during submaximal aerobic exercise. **Figure 2** displays the reduction of plasma volume during 30 minutes of moderate-intensity cycle ergometer exercise (60–70%  $\text{VO}_2\text{max}$ ) in a warm environmental setting (Fortney et al., 1981). As can be observed the main decrease transpires during the initial 5 minutes of exercise, and then the plasma volume stabilises. This decrease in plasma volume suggests that it is fluid shifts, rather than fluid loss, that elucidate the initial decrease (Wade and Freund, 1990). The magnitude of the decrease in plasma volume depends on the intensity of exercise, environmental influences, and the client's hydration status.



**Figure 2.** Cardiovascular Responses to Long-Term, Moderate to Heavy Submaximal Aerobic Exercise. A. Cardiac output (Q). B. Stroke volume (SV). C. Heart rate (HR). D. Blood pressure (SBP, MAP, and DBP). E. Total peripheral resistance (TPR). F. Rate pressure product (RPP).



**Figure 3.** Distribution of Cardiac Output at Rest and during Light Aerobic Exercise.

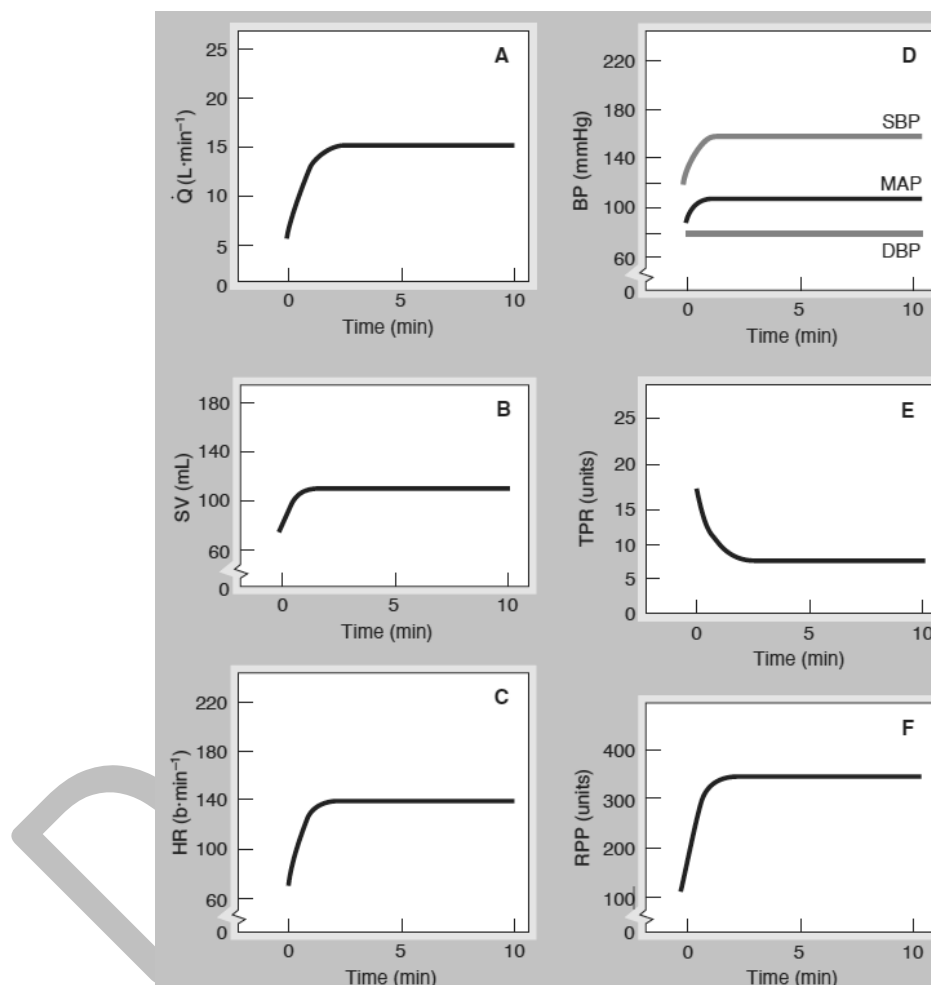
Above in **Figure 3** shows the distribution of  $\dot{Q}$  at rest and during light aerobic exercise. Observe that  $\dot{Q}$  increases from 5.8 to 9.4  $\text{L}\cdot\text{min}^{-1}$  in this illustration. The most significant change in  $\dot{Q}$  distribution with light exercise is the increased percentage from 21 to 47% and the increased blood flow from 1200 to 4500 mL to the working muscles to support energy production. The blood flow of the skin also increases to address the thermoregulatory requirements of exercise. The absolute blood flow to the coronary muscle also increases, although its percentage of  $\dot{Q}$  remains comparatively constant. The absolute amount of cerebral blood flow remains constant while the percentage of  $\dot{Q}$  distributed to the brain decreases. Both renal and splanchnic blood flow is moderately decreased during light exercise.

### Key Point

- The key point, with aerobic exercise, is that as  $\dot{Q}$  increases it is redistributed so that tissues that require increased blood flow, such as the muscles and skin, receive it and other tissues receive either equal or less blood flow.

## Long-Term, Moderate to Heavy Submaximal Aerobic Exercise

The cardiovascular responses to long-term, moderate to heavy submaximal aerobic exercise (60–85% of  $\dot{V}O_{2\max}$ ) are displayed in **Figure 4**. Comparable to light to moderate workloads,  $\dot{Q}$  increases quickly during the two minutes of exercise and then plateaus and remains relatively stable throughout the exercise (**Figure 4A**). However, observe, that the absolute  $\dot{Q}$  reached is higher during heavy exercise than during light to moderate exercise. The increase in  $\dot{Q}$  is due to increased SV and HR.



**Figure 4.** Cardiovascular Responses to Long-Term, Moderate to Heavy Submaximal Aerobic Exercise. A. Cardiac output ( $\dot{Q}$ ). B. Stroke volume (SV). C. Heart rate (HR). D. Blood pressure (SBP, MAP, and DBP). E. Total peripheral resistance (TPR). F. Rate pressure product (RPP).





There is an initial increase in stroke volume before it plateaus, and then has a descending drift as the exercise duration exceeds approximately >30 minutes. Stroke volume increases quickly during the initial few minutes of exercise and then plateaus after a workload of approximately 40–50% of  $VO_{2max}$  is attained (Åstrand et al., 1964) (See **Figure 4B**). Therefore, during work that requires > 50% of  $VO_{2max}$ , the SV response does not depend on intensity. Stroke volume remains somewhat constant during the initial 30 minutes of heavy exercise. Like short-term, light to moderate submaximal aerobic exercise, the increase in SV is suggested to be an outcome of increased venous return (leading to the Frank-Starling mechanism) and increased contractility due to sympathetic nerve stimulation.

Consequently, changes in SV happen due to increases in EDV and decreases in ESV (Poliner et al., 1980). End diastolic volume increases mainly because of the increased venous return of blood to the heart by the active muscle pump and increased vasoconstriction, which reduces venous pooling. End diastolic volume decreases because of increased contractility of the heart, which efficiently ejects more blood. If the exercise continues past 30 minutes, SV progressively drifts downward while continuing to remain above resting values. This downward shift is generally recognised as thermoregulatory stress, which leads to vasodilation, plasma loss, and a redirection of blood to the cutaneous vessels to dissipate heat, effectively reducing venous return and therefore SV. This concept suggests that HR increases to counterbalance the decrease in SV to sustain Q. An alternative notion is that the downward drift in SV is due to an increase in HR that leads to a reduced filling time, therefore leading to a reduced SV (Rowland, 2005b).

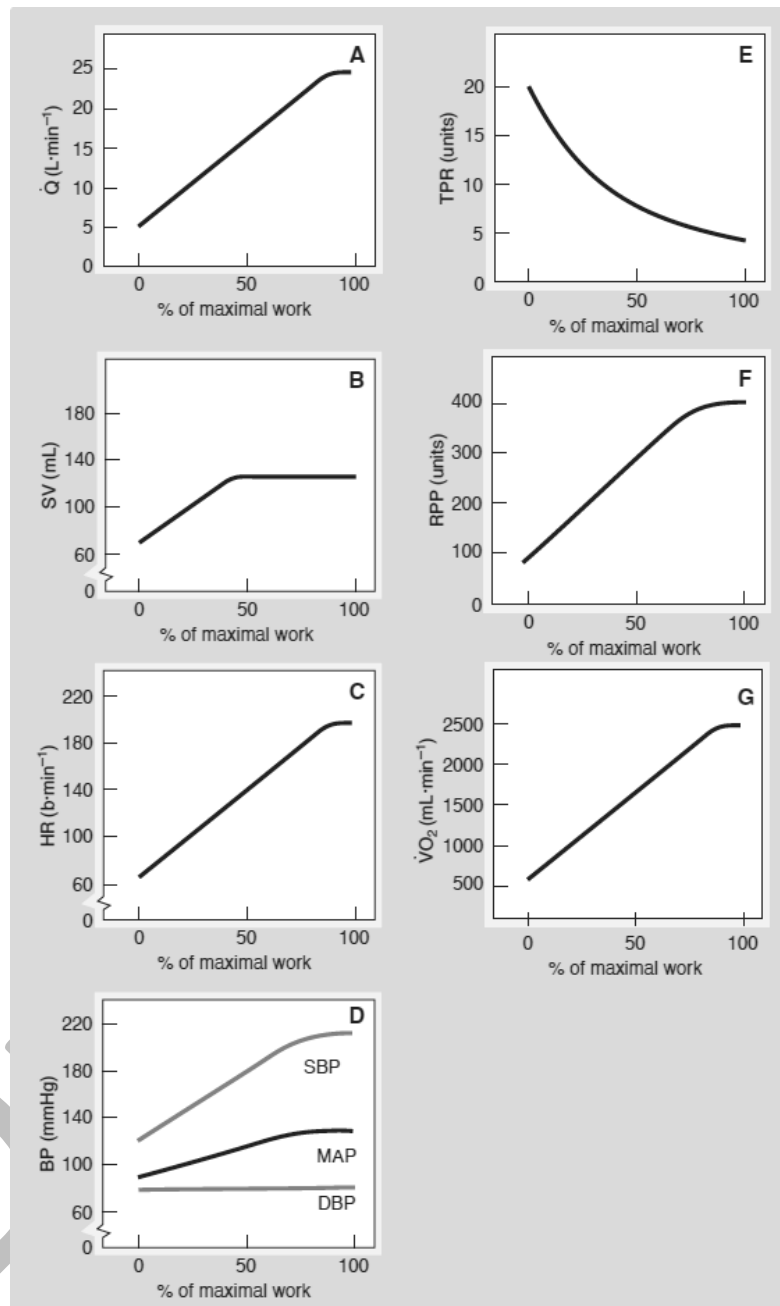


## Incremental Aerobic Exercise to Maximum

**Figure 5** displays the cardiovascular responses to incremental aerobic exercise to the maximum. Observe that unlike the figures previously for light to moderate and heavy exercise, the cardiovascular variables are displayed here with the percentage of maximum work on the x-axis (bottom axis). Incremental exercise to maximum (or a max test) comprises a succession of work stages, each progressively becoming more difficult, that continue until volitional fatigue. The duration of each work stage alters from 1 to 3 minutes to allow a steady state to happen, particularly at the lower workloads. Maximum tests are typically performed in laboratory settings to quantify the physiological responses to the maximal work (intensity) that an individual can achieve. Incremental tests to maximum may include the direct measurement of oxygen consumption.

During an incremental test, cardiac output has a rectilinear increase and plateaus at maximal exercise (**Figure 5A**). The initial increase in cardiac output reflects an increase in the SV and the HR; however, at workloads greater than 40–50%  $VO_{2max}$ , the continued increase in cardiac output in untrained individuals is achieved almost completely by an increase in the HR. As shown in Figure 5B, in untrained individuals, the SV increases rectilinearly initially and then plateaus at approximately 40–50% of  $V\& O_{2max}$  (Åstrand et al., 1964; Higginbotham et al., 1986). The exact SV response to incremental exercise continues to be debated (González-Alonso, 2008; Rowland, 2005a, b; Warburton and Gledhill, 2008). As indicated above, it has traditionally been believed that the SV plateaus at approximately 50% of  $V\& O_{2max}$  in untrained individuals. However, there appears to be considerable interindividual variability in this response, and many laboratories have reported an increase in the SV at maximal exercise in most endurance athletes and some untrained individuals (Ferguson et al., 2001; Gledhill et al., 1994; Warburton et al., 1999). In contrast, other researchers have documented a decrease in the SV at the maximal exercise (Mortensen et al., 2005; Stringer et al., 1997), and some researchers contend that after an initial increase (due to the skeletal muscle pump returning the pooled venous blood to the heart), the SV remains essentially unchanged during the incremental maximal exercise (Rowland, 2005b). Much of the debate is related to the

difficulties in assessing the SV during maximal exercise, with the use of different exercise protocols, and with individual variability.



**Figure 5.** Cardiovascular Response to Incremental Aerobic Exercise to Maximum; (A) Cardiac output (Q); (B) Stroke volume (SV); (C) Heart rate (HR); (D) Blood pressure (SBP, MAP, and DBP); (E) Total peripheral resistance (TPR); (F) Rate-pressure product (RPP); (G) Oxygen consumption (VO<sub>2</sub>).



**Figure 5** illustrates the changes in EDV and ESV that account for variations in the SV during gradually increasing exercise (Poliner et al., 1980). The EDV increases mainly due to the return of blood to the heart by the active muscle pump and the increased sympathetic outflow to the veins initiating vasoconstriction and increasing venous return. ESV decreases because of increased contractility of the heart, which ejects more blood and leaves less in the ventricle. Heart rate increases linearly throughout most of the submaximal ( $\sim 120\text{--}170\text{ b}\cdot\text{min}^{-1}$ ) division of incremental exercise and plateaus at maximal exercise (**Figure 5C**) (Astrand and Rhyning, 1954). Myocardial cells can contract at  $>300\text{ b}\cdot\text{min}^{-1}$  but seldom exceed  $210\text{ b}\cdot\text{min}^{-1}$  because a quicker HR would not permit sufficient ventricular filling, leading to a decrease in SV and Q.

The arterial BP responses to incremental dynamic exercise to the maximum are shown in **Figure 5D**. Systolic blood pressure increases rectilinearly and plateaus at maximal exercise, often attaining values of  $> 200\text{ mmHg}$  in well-trained athletes. This increase is produced by the increased Q, which offsets the concurrent decrease in resistance. Systolic blood pressure and HR are usually examined during exercise tests to ensure the safety of the client. If either of these variables fails to rise with an increasing workload, cardiovascular insufficiency, and an inability to adequately perfuse tissue may result, and the exercise test should be stopped. A drop in SBP of  $10\text{ mmHg}$  or more that occurs despite an increase in workload indicates that the exercise test or session should be discontinued.

Similarly, an upsurge in SBP  $>250\text{ mmHg}$  or DBP  $>115\text{ mmHg}$  suggests that the exercise test or session should be discontinued. Diastolic blood pressure normally remains somewhat constant or deviates insignificantly so that it has no physiological meaning, though it may decrease at high levels of exercise. Diastolic pressure remains relatively stable because vasodilation in the vasculature of the active muscle is balanced by vasoconstriction in other vascular beds. Diastolic pressure is most prone to decrease when exercise is performed in a hot environment setting because the skin vessels are dilated more and there is diminished resistance to blood flow. An increase in DBP  $>115\text{ mmHg}$  may indicate that the exercise assessment should be stopped. Individuals with a hyperbolic BP response to exercise are at an increased risk of hypertension, stroke, and cardiovascular disease mortality compared to those with a



typical exercise BP response (American College of Sports Medicine, 1993; Jae et al., 2006; Kurl et al., 2001; Miyai et al., 2002).

Total peripheral resistance reduces in a negative curvilinear pattern and extends to its lowest level at maximal exercise (**Figure 5E**). Reduced resistance suggests maximal vasodilation in the active tissue in response to the need for increased blood flow during maximal exercise. The considerable decline in resistance is also vital for keeping MAP from becoming too high. The rate pressure product (RPP) increases in a rectilinear manner plateauing at maximum in an incremental exercise test (**Figure 5F**), paralleling the increases in HR and SBP.

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## Maximal Oxygen Consumption

**Figure 5G** shows that oxygen consumption increases linearly in relation to the exercise intensity during an incremental exercise to maximum (**Figure 5G**). The greatest amount of oxygen an individual can absorb, transport, and utilise to generate ATP aerobically while breathing air during heavy exercise is termed **maximal oxygen consumption** ( $\dot{V}O_{2\max}$ ).

As previously discussed, maximal oxygen intake is assessed during an incremental maximal exercise test. Maximal oxygen consumption can be defined by rearranging the Fick equation (**see Equation 2**) to the following equation:

$$\dot{V}O_{2\max} = (\dot{Q}_{\max}) \times (a - vO_{2\text{diff max}})$$

The rectilinear increase in  $\dot{Q}$  during a maximal incremental exercise test has been previously discussed. The changes in the  $a-vO_{2\text{diff}}$  are an increase with a plateau at approximately 60% of  $\dot{V}O_{2\max}$ . The outcome of these changes is a rectilinear increase in oxygen up to maximum. Maximal exercise tests that include the assessment of oxygen consumption for the determination of  $\dot{V}O_{2\max}$  are regularly administered by sport and exercise scientists to establish an athlete's fitness or to monitor fluctuations in fitness. Furthermore, researchers administer these tests to attempt to comprehend the various mechanisms that restrict the exercise or to investigate other areas related to physiological function under stressful conditions.

$\dot{V}O_{2\max}$  is frequently used as the criterion measure of aerobic (also termed cardiorespiratory) fitness. However,  $\dot{V}O_{2\max}$  is an amalgamated measure of fitness that involves the capability of the body to absorb, transport, and use oxygen. Consequently,  $\dot{V}O_{2\max}$  may be suggested as a cardiovascular, respiratory, and metabolic variable. However,  $\dot{V}O_{2\max}$  has important consequences for cardiovascular health and is limited by cardiovascular function.





## Criteria for Determining $VO_{2max}$

When an individual reaches volitional fatigue, the highest oxygen consumption may or may not denote an explicit maximal value. Before considering the highest value as  $VO_{2max}$ , the sport and exercise scientist must consider whether a test accurately is a maximal one. Therefore, the often-used term  $VO_{2\ peak}$  is used to signify the highest value attained during the test if the exercise physiologist is not assured that a true maximal value was reached.

Criteria used to determine whether the test is maximal include:

1. A lactate value  $> 8-9 \text{ mmol}\cdot\text{L}^{-1}$  (Åstrand, 1956; Åstrand et al., 2003).
2. A heart rate  $\pm 12 \text{ b}\cdot\text{min}^{-1}$  of predicted maximal heart rate  $(220 - \text{age})$  (Durstine and Pate, 1988).
3. A respiratory exchange ratio of 1.0 or 1.1, mainly depending on the age of the subject (Holly, 1988; MacDougall et al., 1982).
4. A plateau in oxygen consumption. The classic definition of a plateau is a rise of  $2.1 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  or less, or a rise of  $0.15 \text{ L}\cdot\text{min}^{-1}$  or less, in oxygen consumption ( $VO_2$ ) with an increase in workload that represents a change in grade of 2.5% while running at  $7 \text{ mi}\cdot\text{hr}^{-1}$  ( $11.2 \text{ km}\cdot\text{hr}^{-1}$ ) with 3-minute stages (Taylor et al., 1955).



## Factors Restricting $VO_{2max}$

At certain points, an individual cannot continue to increase the exercise load or is capable to work at a maximum effort due to the body being unable to deliver and use more oxygen to support further workload. Hypothetically, maximal oxygen uptake could be restricted by any system along the route of transporting oxygen into the body and distributing it to the mitochondria to produce ATP. Therefore, any of the subsequent systems may restrict  $VO_{2max}$ :

1. The respiratory system, due to insufficient ventilation, oxygen diffusion limitations, or an inability to maintain the gradient for the diffusion of  $O_2$  ( $a-vO_{2diff}$ ).
2. The cardiovascular system, because of restricted blood flow ( $Q$ ) or oxygen-carrying capacity ( $Hb$ ).
3. The metabolic functions within the skeletal muscle, include the inability to produce further ATP due to the restricted number of mitochondria, limited enzyme levels or activity, or inadequate substrates.

Bergh and colleagues (2000) have suggested that each of these above systems may constrain  $VO_{2max}$  settings. For instance, a reduction in the partial pressure of oxygen ( $PO_2$ ) at altitude or with asthma produces a reduction in  $VO_{2max}$ . Certain medications including beta-blockers may limit  $Q$  causing a reduction in  $VO_{2max}$ . Furthermore, diseases in which muscle enzymes involved in metabolism are deficient can also generate a reduction in  $VO_{2max}$ .

However, factors in the above systems may limit  $VO_{2max}$  resulting in various unanswered questions including What restricts  $VO_{2max}$  in healthy humans when they perform the maximal exercise? This question has interested exercise physiologists since the work of A. V. Hill in the 1920s and is currently being debated among physiologists today (Bassett and Howley, 2000; Bergh et al., 2000; Grassi, 2000; Hale, 2008; Saltin, 1985).



Contemporary evidence suggests that maximal oxygen uptake is limited by the ability of the cardiorespiratory system to deliver oxygen to the muscle, rather than the ability of the muscle mitochondria to use oxygen (Bergh et al., 2000; Hale, 2008; Rowell, 1993; Saltin, 1985). Particularly,  $Q$  appears to be the limiting factor in  $VO_{2max}$  (Bergh et al., 2000; di Prampero, 2003; Saltin, 1985). Furthermore, evidence suggests that oxygen uptake is not limited by pulmonary ventilation in normal, healthy athletes without exercise-induced arterial hypoxemia. Normally, the functional capacity of the respiratory system is considered to exceed the demands of maximal exercise (Rowell, 1993). The only variable likely to impose a limitation on oxygen transport is  $a-vO_{2diff}$ .

Various studies have reported that skeletal muscles could utilize more oxygen than can be supplied by the respiratory and cardiovascular systems (Richardson, 2000; Rowell, 1993; Saltin, 1985). However, this has been debated with others proposing that failure of muscle performance may explain exhaustion during maximal exercise (Noakes, 1988).

Feasibly, the factors limiting  $VO_{2max}$  vary with the fitness level of the individual. According to this hypothesis, in an untrained individual, the respiratory capacity for gas exchange exceeds the cardiovascular system's capacity to deliver oxygen. A training program results in little change in the respiratory capacity but large changes in the cardiovascular capacity. Thus, in some highly trained individuals who have exercise-induced arterial hypoxemia, the increased cardiovascular capacity may exceed the respiratory capacity (Dempsey, 1986; Legrand et al., 2005; Powers et al., 1989). In this case, the respiratory system becomes the factor limiting  $VO_{2max}$ .

One final point to consider is even though it is interesting to investigate the question, "what limits  $VO_{2max}$ ?". According to Mitchell and Saltin (2003), we should resist the lure to let the search for an answer cloud the fact that a close interaction exists among the various systems ensuring continuous delivery of oxygen to the working tissue during exercise. The reduction in plasma volume during submaximal exercise also happens in incremental exercise to the maximum. Because the magnitude of the reduction depends on the intensity of exercise, the reduction is greatest at maximal exercise. A decrease of 10–20% can be seen during incremental exercise to maximum (Wade and Freund, 1990).



Considerable changes in  $Q$  occur during maximal incremental exercise. **Table 1** illustrates the distribution of  $Q$  at rest and maximal aerobic exercise. The maximum  $Q$  in this instance is  $25 \text{ L}\cdot\text{min}^{-1}$ . Again, the most noticeable variation is the amount of  $Q$  that is directed to the working muscles (88%). At maximal exercise, skin blood flow is reduced to direct the necessary blood to the muscles. Renal and splanchnic blood flows also decrease considerably. Blood flow to the brain and cardiac muscle is maintained.

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**Table 1. Cardiovascular Responses to Exercise\***

	Short-Term, Light to Moderate Submaximal Aerobic Exercise	Long-Term, Moderate to Heavy Submaximal Aerobic Exercise†	Incremental Aerobic Exercise to Maximum	Static‡ Exercise	Resistance‡ Exercise
<b>Q</b>	<ul style="list-style-type: none"> <li>Increases rapidly plateau at a steady state within 2 min</li> </ul>	<ul style="list-style-type: none"> <li>Increases rapidly; plateaus</li> </ul>	<ul style="list-style-type: none"> <li>Rectilinear increase with a plateau at max</li> </ul>	<ul style="list-style-type: none"> <li>Modest gradual increase</li> </ul>	<ul style="list-style-type: none"> <li>Modest gradual increase</li> </ul>
<b>SV</b>	<ul style="list-style-type: none"> <li>Increases rapidly plateau at a steady state within 2 min</li> </ul>	<ul style="list-style-type: none"> <li>Increases rapidly; plateaus; negative drift</li> </ul>	<ul style="list-style-type: none"> <li>Increases initially; plateaus at 40– 50% <math>VO_{2max}</math></li> </ul>	<ul style="list-style-type: none"> <li>Relatively constant at low workloads, decreases at high workloads; rebound rise in recovery</li> </ul>	<ul style="list-style-type: none"> <li>Little change, a slight decrease</li> </ul>
<b>HR</b>	<ul style="list-style-type: none"> <li>Increases rapidly plateau at a steady state within 2 min</li> </ul>	<ul style="list-style-type: none"> <li>Increases rapidly. plateaus; positive drift</li> </ul>	<ul style="list-style-type: none"> <li>Rectilinear increase with a plateau at max</li> </ul>	<ul style="list-style-type: none"> <li>Modest gradual increase</li> </ul>	<ul style="list-style-type: none"> <li>Increases gradually with numbers of reps</li> </ul>
<b>SBP</b>	<ul style="list-style-type: none"> <li>Increases rapidly plateau at a steady state within 2 min</li> </ul>	<ul style="list-style-type: none"> <li>Increases rapidly. plateaus; slight negative drift</li> </ul>	<ul style="list-style-type: none"> <li>Rectilinear increase with a plateau at max</li> </ul>	<ul style="list-style-type: none"> <li>Marked steady increase</li> </ul>	<ul style="list-style-type: none"> <li>Increases gradually with numbers of reps</li> </ul>
<b>DBP</b>	<ul style="list-style-type: none"> <li>Shows little or no change</li> </ul>	<ul style="list-style-type: none"> <li>Shows little or no change</li> </ul>	<ul style="list-style-type: none"> <li>Shows little or no change</li> </ul>	<ul style="list-style-type: none"> <li>Marked steady increase</li> </ul>	<ul style="list-style-type: none"> <li>No change or increase</li> </ul>
<b>MAP</b>	<ul style="list-style-type: none"> <li>Increases rapidly, plateaus at steady state within 2 min</li> </ul>	<ul style="list-style-type: none"> <li>Increases initially, little if any drift</li> </ul>	<ul style="list-style-type: none"> <li>Small rectilinear increase</li> </ul>	<ul style="list-style-type: none"> <li>Marked steady increase</li> </ul>	<ul style="list-style-type: none"> <li>Increases gradually with numbers of reps</li> </ul>
<b>TPR</b>	<ul style="list-style-type: none"> <li>Decreases rapidly; plateaus</li> </ul>	<ul style="list-style-type: none"> <li>Decreases rapidly; plateaus; slight negative drift</li> </ul>	<ul style="list-style-type: none"> <li>Curvilinear decrease</li> </ul>	<ul style="list-style-type: none"> <li>Decreases</li> </ul>	<ul style="list-style-type: none"> <li>Slight increase</li> </ul>
<b>RPP</b>	<ul style="list-style-type: none"> <li>Increases rapidly, plateaus at steady state within 2 min</li> </ul>	<ul style="list-style-type: none"> <li>Increases rapidly; plateaus; positive drift</li> </ul>	<ul style="list-style-type: none"> <li>Rectilinear increase with a plateau at max</li> </ul>	<ul style="list-style-type: none"> <li>Marked steady increase</li> </ul>	<ul style="list-style-type: none"> <li>Increases gradually with numbers of reps</li> </ul>

\*Resting values are taken as baseline.

†The difference between a plateau during the short-term, light to moderate and long-term, moderate to heavy submaximal exercise response is one of magnitude; that is, a plateau occurs at a higher value with higher intensities.

‡The magnitude of a plateau change depends on the %MVC/load

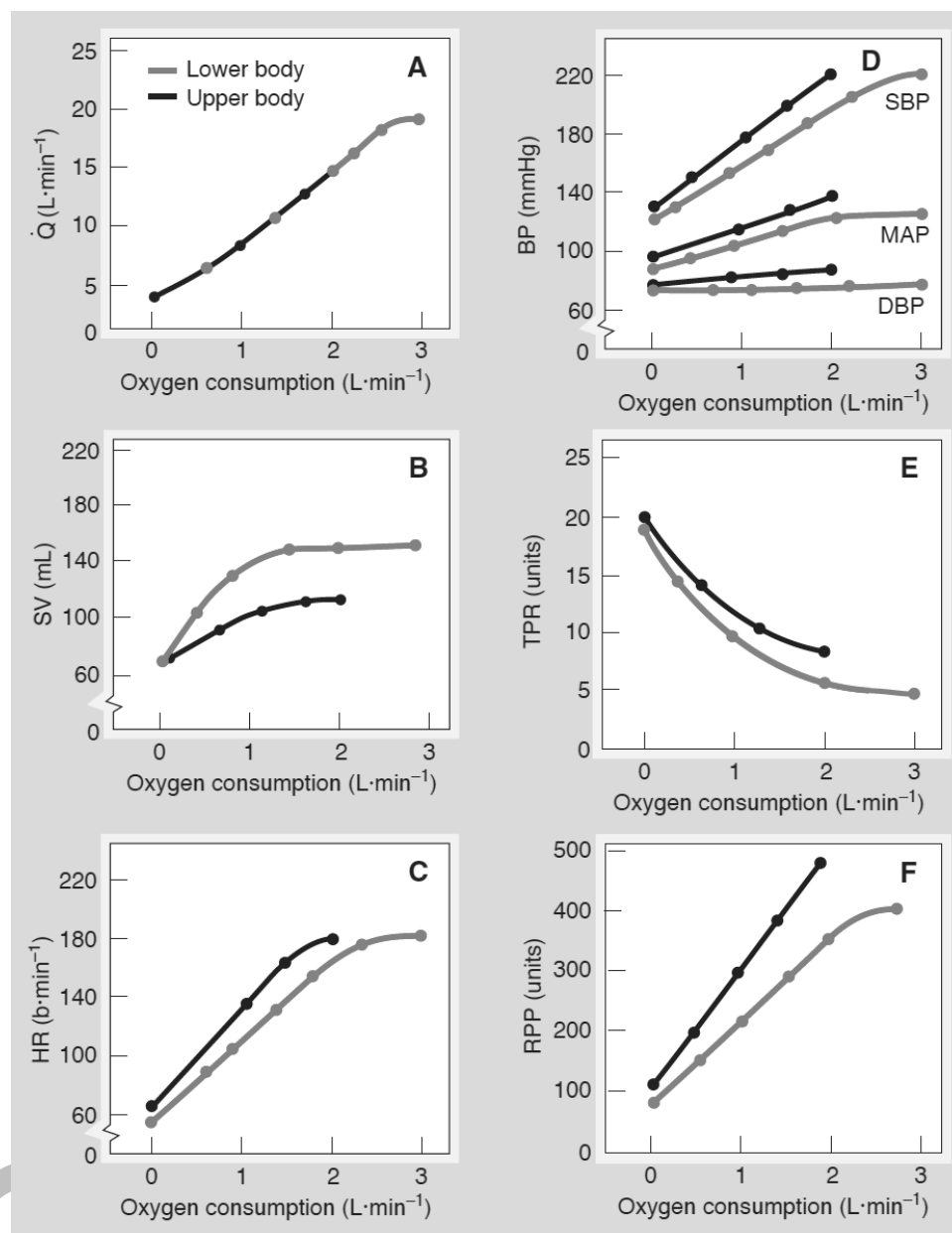


## Upper-Body versus Lower-Body Aerobic Exercise

Upper-body exercise is consistently performed throughout our day-to-day life including recreational, and sporting activities. The cardiovascular responses to exercise using muscles of the upper body are different in several significant ways from exercise performed using the lower body muscles. **Figure 6** below displays data regarding the cardiovascular responses to incremental exercise to the maximum in healthy subjects using the upper body (arm cranking on an arm ergometer) vs. lower body (cycling on a cycle ergometer). Observe that a higher peak  $\text{VO}_2$  was attained during lower-body exercise. Comparisons at any given level of oxygen consumption also demonstrate the differences in cardiovascular responses to submaximal upper- and lower-body exercise. When the oxygen consumption needed to perform a submaximal workload is equivalent,  $\dot{Q}$  is comparable for both upper- and lower-body exercise (**Figure 6A**). Conversely, the mechanism to attain the required increase in  $\dot{Q}$  is not equivalent. As displayed in **Figures 6B** and **6C**, upper-body exercise results in a lower SV and a higher HR at any submaximal workload (Clausen, 1976; Miles et al., 1989; Pendergast, 1989). SBP, DBP, MAP (**Figure 6D**), total peripheral resistance (**Figure 6E**), and rate pressure product (**Figure 6F**) are also considerably higher in upper-body exercise than compared to lower-body exercise performed at the same oxygen consumption.

There are various reasons for the above differences: (i) The higher HR reported during upper-body exercise is understood to reflect a greater sympathetic stimulation (Åstrand et al., 2003; Davies et al., 1974; Miles et al., 1989); (ii) Stroke volume (SV) is lower during upper-body exercise because of the lack of the skeletal muscle pump increasing venous return from the legs. The greater sympathetic stimulation that occurs during upper-body exercise may also be partly responsible for the increased BP and total peripheral resistance. Upper-body exercise often includes a static element that produces a hyperbolic BP response. For example, using an arm-cranking ergometer has a static element because the client must grasp the hand cranks.





**Figure 6.** Cardiovascular Response to Incremental Aerobic Maximal Upper Body and Lower-Body Exercise. **A.** Cardiac output ( $\dot{Q}$ ). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Blood pressure (SBP, MAP, and DBP). **E.** Total peripheral resistance (TPR). **F.** Rate-pressure product (RPP).

When maximal exercise is performed using upper body muscles,  $VO_{2max}$  values are almost 30% lower than when maximal exercise is implemented using lower-body muscles (Miles et al., 1989; Pendergast, 1989). Maximal HR values for upper-body exercise are approximately 90–95% of those for lower-body exercise, with the SV circa 30–40% less during maximal upper-body exercise. Maximal SBP and the rate-



pressure product are typically similar, but DBP is normally 10–15% higher during upper-body exercise (Miles et al., 1989).

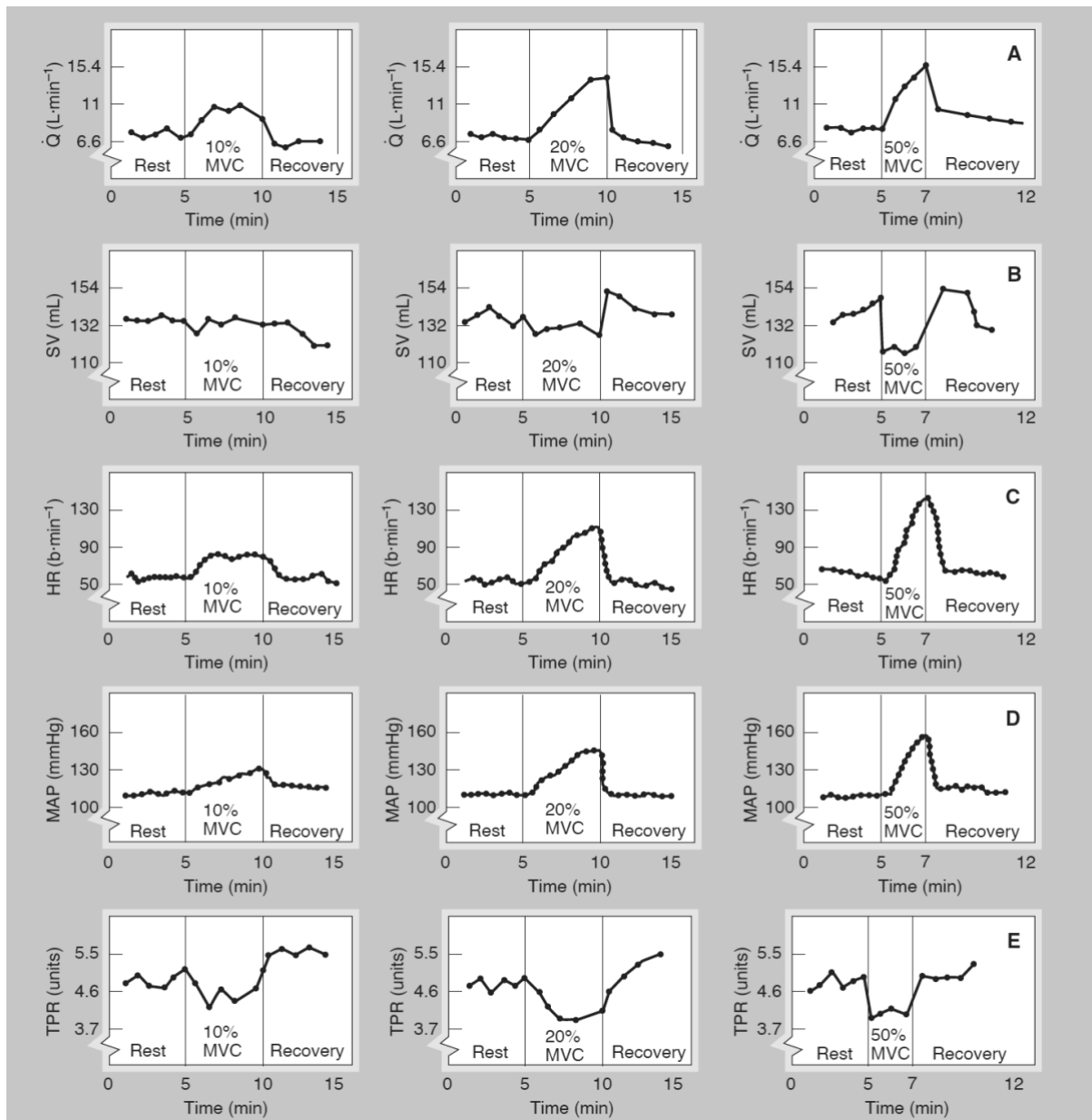
The various cardiovascular responses to an absolute workload performed with the upper body versus the lower body dictate that exercise prescriptions for arm work cannot be based on data obtained from testing with leg exercises. Additionally, the greater cardiovascular strain associated with upper-body work must be kept in mind when one prescribes exercise for individuals with cardiovascular disease.

### Cardiovascular Responses to Static Exercise

Static work occurs repeatedly during daily activities, such as lifting and carrying heavy objects. It is also a common form of activity encountered in many occupational settings, particularly manufacturing jobs where lifting is common. Additionally, many sports and recreational activities have a static component associated with their performance. For example, weightlifting, rowing, and racquet sports all involve static exercise. The magnitude of the cardiovascular response to static exercise is affected by several factors, but most noticeably by the intensity of muscle contraction.

### Intensity of Muscle Contraction

The cardiovascular response to static exercise varies on the intensity of contraction, and the specific time the contraction is held. The intensity of a static contraction is normally stated as a percentage of maximal voluntary contraction (%MVC). **Figure 7** displays the cardiovascular response to static contractions of the forearm (via handgrip) muscles at 10%, 20%, and 50% MVC. Observe that at 10% and 20% MVC the contraction could be held for 5 minutes, but at 50%, MVC the contraction could only be held for 2 minutes. Therefore, like aerobic exercise, intensity and duration are inversely associated. Additionally, it is important to note that the data presented in **Figure 7** are from handgrip exercises only. Though the pattern of response seems to be comparable for different muscle groups, the specific values may vary significantly conditional on the quantity of active muscle tissue involved.



**Figure 7.** Cardiovascular Response to Varying Intensities of Static Handgrip Exercise. **A.** Cardiac output (Q). **B.** Stroke volume (SV). **C.** Heart rate (HR). **D.** Mean arterial pressure (MAP). **E.** Total peripheral resistance (TPR).

Cardiac output increases during static contractions due to an increase in HR, with the magnitude of the increase dependent on the intensity of exercise. SV (**Figure 7B**) remains relatively constant or decreases slightly during low-intensity contractions and decreases during high-intensity contractions. There is a marked increase in SV immediately following the cessation of high-intensity contractions (Lind et al., 1964; Smith et al., 1993). This is the same rebound rise in recovery as seen in  $a\text{-}v\text{O}_{2\text{diff}}$ , VE,



and  $\text{VO}_2$ . The reduction in SV during high-intensity contractions probably results from both a decreased preload and increased afterload. Preload is decreased because of high intrathoracic pressure, which compresses the vena cava and thus decreases the return of venous blood to the heart. Because arterial BP is markedly elevated during static contractions (increased afterload), less blood is ejected at a given force of contraction. HR (**Figure 7C**) increases during static exercise. The magnitude and the rate of the increase in HR depend on the intensity of contraction. The greater the intensity, the greater the HR response.

Static exercise is characterized by a rapid increase in both systolic pressure and diastolic pressure, termed the pressor response, which appears to be inappropriate for work produced by the contracting muscle (Lind et al., 1964). Since both systolic and diastolic pressures increase, there is a marked increase in MAP (**Figure 7D**) (Donald et al., 1967; Lind et al., 1964; Seals et al., 1985; Tuttle and Horvath, 1957). As in any muscular work, static exercise increases the metabolic demands of the active muscle. However, in static work, high intramuscular tension results in mechanical constriction of the blood vessels, which impedes blood flow to the muscle. The reduction in muscle blood flow during static exercise results in a build-up of local by-products of metabolism. These chemical by-products [ $\text{H}^+$ , adenosine diphosphate, and others] stimulate sensory nerve endings, which leads to a pressor reflex, causing a rise in MAP (pressor response). This rise is substantially larger than the increase during aerobic exercise requiring similar energy expenditure (Asmussen, 1981; Hanson and Nagle, 1985). Observe in **Figure 7D** that holding a handgrip dynamometer at 20% MVC for 5 minutes resulted in an increase of 20–30 mmHg in MAP and holding 50% MVC for 2 minutes caused a 50-mmHg increase in a MAP!

Total peripheral resistance, indicated by TPR in **Figure 7E**, decreases during static exercise, although not to the extent seen in dynamic aerobic exercise. The smaller decrease in resistance helps to explain the higher BP response to static contractions. The high BP generated during static contractions helps overcome resistance to blood flow from mechanical occlusion. Because the SBP and the HR both increase during static exercise, there is a large increase in myocardial oxygen consumption and thus rate-pressure product.



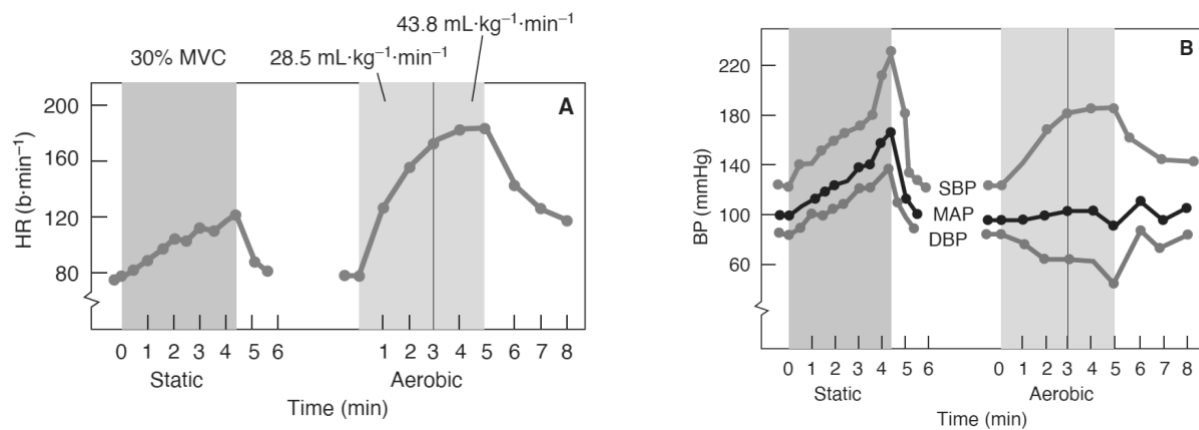
## Blood Flow during Static Contractions

Blood flow to the working muscle is impeded during static contractions because of the mechanical constriction of the blood vessel supplying the contracting muscle (Freund et al., 1979; Sjogaard et al., 1988). Figure 12.13 depicts blood flow in the quadriceps muscle when a 5% and 25% MVC contraction were held to fatigue. The 5% MVC load could be held for 30 minutes; the 25% load could be held for only 4 minutes. Quadriceps blood flow is greater during the 5% MVC, suggesting that at 25% MVC there is considerable impedance to blood flow. Blood flow during the 25% MVC load was very close to resting levels despite the metabolic work done by the muscle. The response occurring during recovery suggests that when contraction ceases, a mechanical occlusion to the muscle is released. The marked increase in blood flow during recovery compensates for the reduced flow during sustained contraction. The relative force at which blood flow is impeded varies greatly among different muscle groups (Lind and McNichol, 1967; Rowell, 1993).

Mechanical constriction also occurs during dynamic aerobic exercise. However, the alternating periods of muscular contraction and relaxation during rhythmical activity allow—and, indeed, encourage—blood flow, especially through the venous system.

## Comparison of Aerobic and Static Exercise

**Figure 8** compares the HR and the BP responses to fatiguing handgrip (static) exercise (30% MVC held to fatigue) and a maximal treadmill (incremental aerobic) test to fatigue. The incremental aerobic exercise is characterized by a large increase in the HR, which contributes to increased cardiac output. The treadmill exercise response also shows a modest increase in the SBP and a relatively stable or decreasing DBP. Aerobic exercise is said to impose a “volume load” on the heart. Increased venous return leads to increased SV, which contributes to increased cardiac output. In contrast, fatiguing static exercise is characterized by a modest increase in the HR, but a dramatic increase in the BP (pressor response). Mean BP increases because of increased SBP and DBP. Static exercise is said to impose a “pressure load” on the heart. Increased MAP means that the heart must pump harder to overcome the pressure in the aorta.



**Figure 8.** Comparison of HR (A) and BP (B) Response to Static and Incremental Aerobic Exercise to Maximum.

### Cardiovascular Responses to Dynamic Resistance Exercise

Weightlifting or resistance exercise includes a combination of dynamic and static contractions (Hill and Butler, 1991; MacDougall et al., 1985). At the beginning of the lift, a static contraction exists until muscle force exceeds the load to be lifted and movement occurs, leading to a dynamic concentric (shortening) contraction as the lift continues. This is then followed by a dynamic eccentric (lengthening) contraction during the lowering phase (McCartney, 1999). A static component is always associated with gripping the barbell. During dynamic resistance exercise, cardiorespiratory system responses are dissociated from the energy demand. In contrast, during dynamic endurance activity, responses in the cardiorespiratory system are directly related to the use of oxygen for energy production. In part, the reason for this dissociation between oxygen use and cardiovascular response to resistance exercise is that much of the energy required for resistance exercise comes from anaerobic (without oxygen) sources. Another important difference between resistance exercise and aerobic exercise is the mechanical constriction of blood flow during resistance exercise because of the static nature of the contraction.

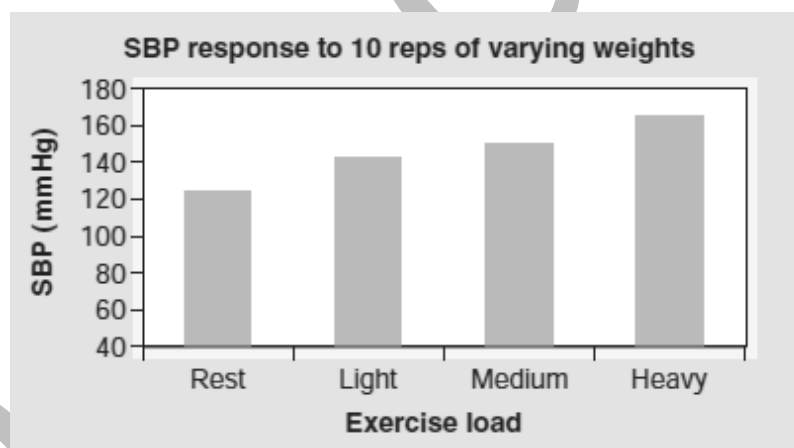
The magnitude of the cardiovascular response to resistance exercise depends on the intensity of the load (the weight lifted) and the number of repetitions performed. Cardiovascular responses also depend on how the load and repetitions are combined.





## Varying Load/Constant Repetitions

As expected, cardiovascular responses are greater when heavier loads are lifted, assuming the number of repetitions is constant (Fleck, 1988; Fleck and Dean, 1987). For example, as shown in **Figure 9**, when participants performed 10 repetitions of arm curling exercises with dumbbells of three different weights (identified as light, moderate, and heavy), the SBP was highest at the heaviest set (Wescott and Howes, 1983). The SBP increased 16%, 22%, and 34% during the light, moderate, and heavy sets, respectively. The DBP, measured by auscultation, did not change significantly with any of the sets. There is disagreement about the DBP response to resistance exercise; some authors report an increase, while others report no change (Fleck, 1988; Fleck and Dean, 1987; Wescott and Howes, 1983). These discrepancies may reflect differences in measurement techniques (auscultation versus intra-arterial assessment) and the timing of the measurement.

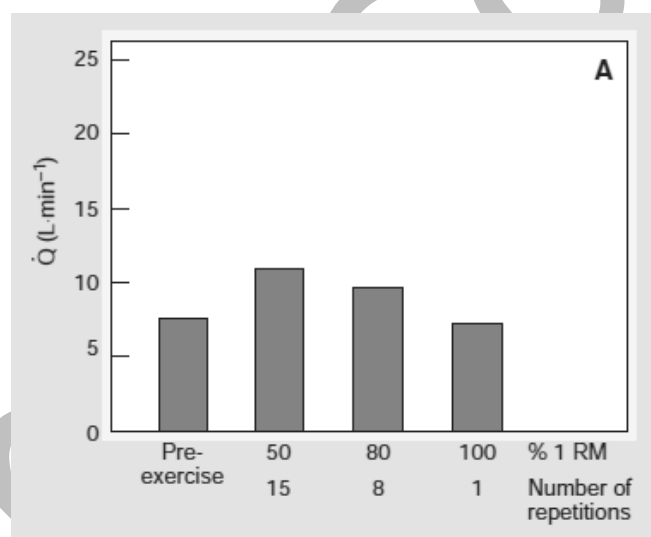


**Figure 9.** Systolic Blood Pressure (SBP) Response at the Completion of 10 Reps of Arm Curls Using Different Weights.



## Varying Load/Repetitions to Failure

A different pattern of response is seen when a given load is performed to fatigue, which lifters typically call failure. In this case, the individual performs maximal work regardless of the load. **Figure 10** shows the cardiovascular response at the complete after exercises performed to failure. Participants performed 50%, 80%, and 100% of their one-repetition maximum (1-RM) as many times as they could, and cardiovascular variables were recorded at the end of each set (Falkel et al., 1992). Participants could perform the 100% load only one time, of course, but they could perform the 80% and 50% loads an average of 8 and 15 times, respectively. Thus, the greatest amount (volume) of work was performed when the lightest load was lifted the greatest number of times. Cardiac output at the completion of the set after lightest load was lifted for the most repetitions—that is, when the total work was greatest (**Figure 10A**).

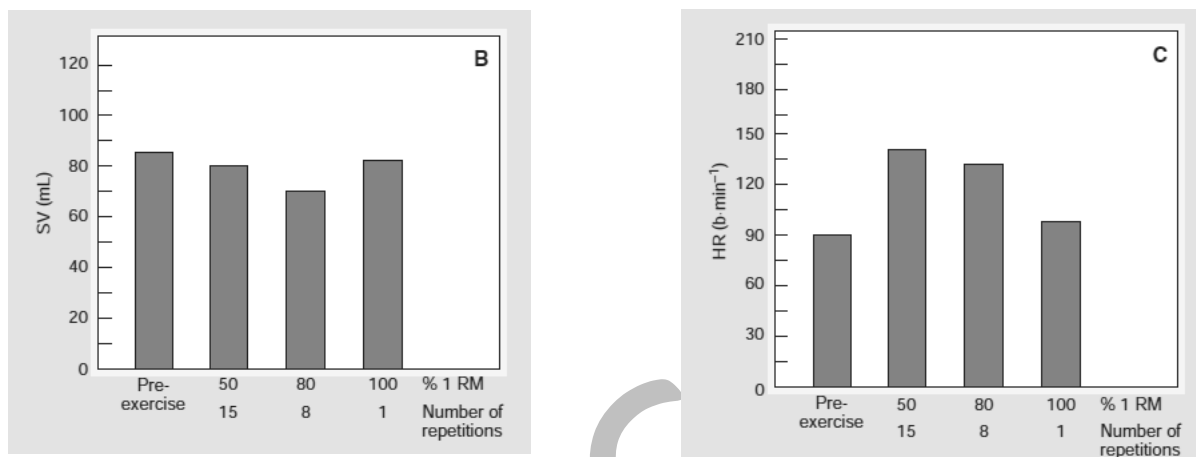


**Figure 10A.** Cardiovascular Response at the Completion of Dynamic Resistance Exercise [Concentric Knee Extension Exercise] to Failure with Varying loads. A. Cardiac output (Q).

The SV at the end of a set was similar for each condition (**Figure 10B**) and was slightly below resting levels. This contrasts with significant increases in the SV that occur during aerobic exercise. Thus, dynamic resistance exercise does not produce the SV overload of dynamic aerobic exercise (Hill and Butler, 1991; McCartney, 1999). The HR was highest after completion of the set using the lightest load and lifting it the most times (**Figure 10C**). The HR was lowest when a single repetition using the



heaviest weight was performed, and hence when the least amount of work was done. HRs between 130 and 160  $\text{b}\cdot\text{min}^{-1}$  have been reported during resistance exercise (Hill and Butler, 1991). There is some evidence that the HR and the BP attained at fatigue are the same when loads between 60% and 100% of 1-RM are used, regardless of the number of times the load can be performed (Nau et al., 1990).



**Figure 10B and 10C.** Cardiovascular Response at the Completion of Dynamic Resistance Exercise [Concentric Knee Extension Exercise] to Failure with Varying loads.

### Constant Load/Repetitions to Failure

When the load is substantial, MAP and HR increase with subsequent repetitions in a set to failure (Fleck and Dean, 1987; MacDougall et al., 1985). **Figure 11A** displays the MAP, measured intra-arterially, during a set of leg press exercises that represented 95% of 1-RM; **Figure 11B** displays the HR during these exercises. In this study, peak SBP averaged 320 mmHg, and peak DBP averaged 250 mmHg! The study demonstrated that increase in BP during dynamic resistance exercise results from the mechanical compression of blood vessels and performance of the Valsalva manoeuvre. The TPR is higher during dynamic resistance exercise than during dynamic aerobic exercise because of the vasoconstriction caused by the pressor reflex. Moreover, several studies have reported a minor increase in the TPR during resistance exercise, rather than the decrease reported with aerobic exercise (Lentini et al., 1993; McCartney, 1999; Miles et al., 1987). Myocardial oxygen consumption and therefore the rate-pressure product can reach high levels due to the tachycardia



and the hyperbolic SBP response. Dynamic resistance exercise also produces large (about 15%) but transient decreases in plasma volume (Hill and Butler, 1991).

Resistance exercises are commonly performed to enhance muscle size or improve muscular health. The aim, therefore, is not to stress the cardiovascular system. Consequently, there is insufficient evidence to sufficiently compare cardiovascular responses to resistance exercise among different populations (male versus female, children versus adult, and young versus older adults).

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## Cardiovascular Responses of Older Adults to Exercise

Ageing is associated with diminishing function in many systems of the body. Thus, ageing is characterized by a decreased ability to respond to physiological stress (Skinner, 1993). There is considerable debate, though, about how much loss of function is inevitably related to age, how much is related to disease, and how much can be attributed to a sedentary lifestyle often accompanying ageing. Each of these factors causes decrements in function, but for an individual, it is often difficult to know which one or which combination may cause an observed change.

Many older adults remain active into their later years and perform amazing athletic feats. Many studies on physical activity suggest that by remaining active in the older years, individuals can markedly reduce loss of cardiovascular function.

### Short-Term, Light to Moderate and Long-Term, Moderate to Heavy Submaximal Exercise

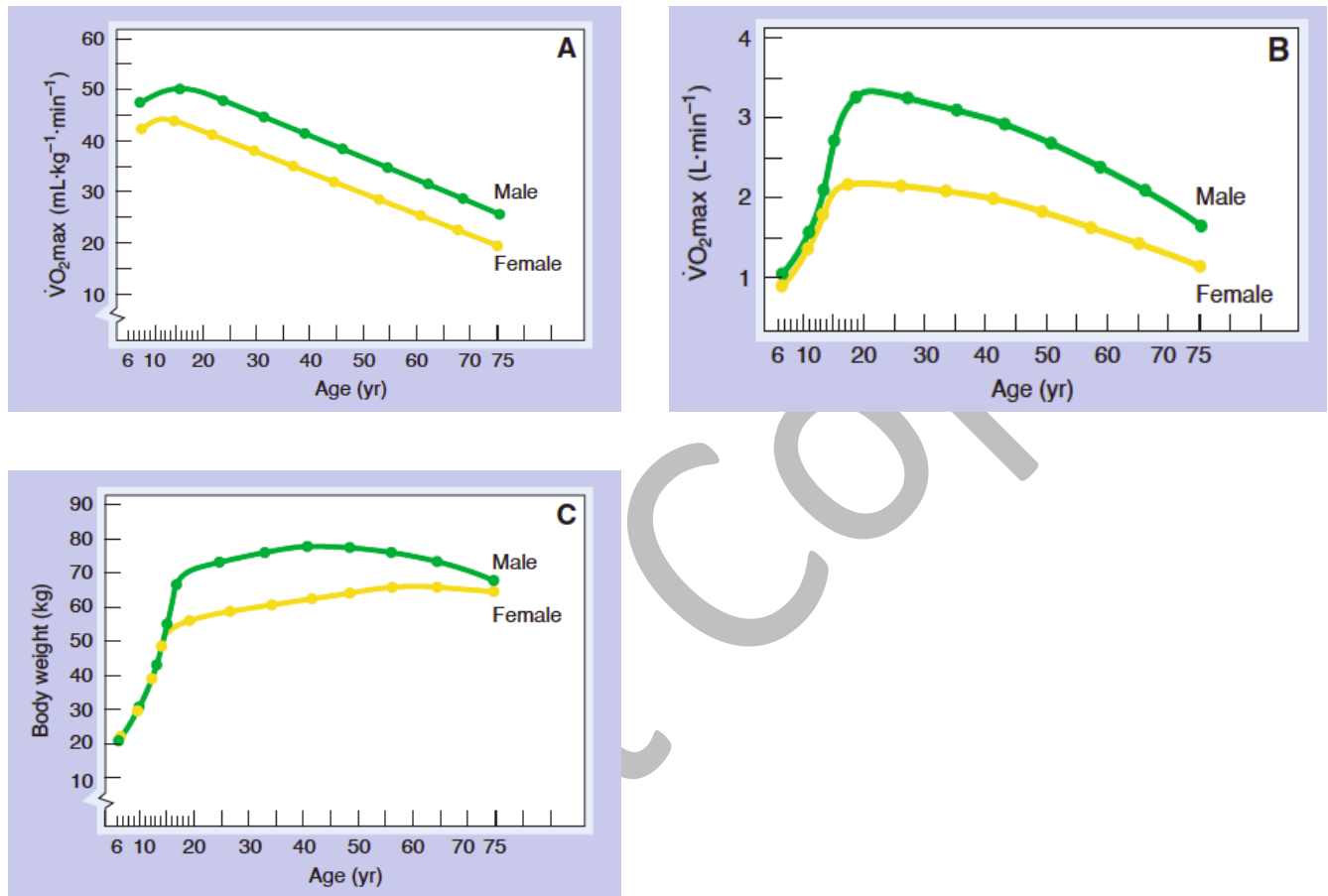
At the same absolute submaximal workload, cardiac output and SV are lower in older adults, but HR is higher than in younger adults. The pattern of systolic and diastolic pressure is the same for younger and older individuals. The difference in resting BP is maintained throughout the exercise, so that older individuals have a higher SBP, DBP, and MAP at any given level of exercise (Ogawa et al., 1992). The higher BP response is related to a higher TPR in older individuals, resulting from a loss of elasticity in the blood vessels. Because HR and SBP are higher for any given level of exercise in older adults, myocardial oxygen consumption and thus rate pressure products are also higher in older individuals than in younger adults.

### Incremental Aerobic Exercise to Maximum

Maximal Q is lower in older individuals than in younger adults. This results from a lower maximal HR and a lower maximal SV. Maximal SV decreases with advancing age, and the decline is of similar magnitude for both men and women, although women have a much smaller maximal SV initially. Maximal HR decreases with age but does not vary significantly between the sexes. A decrease of approximately 10% per



decade, starting at approximately age 30, has been reported for  $\dot{V}O_{2\max}$  in sedentary and active adults (Åstrand, 1960; Heath et al., 1981; Wilson and Tanka, 2000). There is some indication that the rate of decline in  $\dot{V}O_{2\max}$  is greater in men than in women (Stathokostas et al., 2004; Weiss et al., 2006).



**Figure 11.** Maximal Oxygen Consumption ( $\dot{V}O_{2\max}$ ) is expressed in relative (A) and absolute terms (B) and Body Weight for Males and Females from 6 to 75 Years.

Like resting BP, SBP and DBP responses to maximal aerobic exercise are typically higher in older individuals than in younger individuals of similar fitness (Ogawa et al., 1992). Maximal SBP may be 20–50 mmHg higher in older individuals, and maximal DBP is 15–20 mmHg higher. As a result of an elevated SBP and DBP, MAP is considerably higher at maximal exercise in older than in younger adults.

TPR decreases during aerobic exercise in older adults but not to the same extent as in younger individuals. This difference is a consequence of the loss of





elasticity of the connective tissue in the vasculature that accompanies ageing. Since the decrease in maximal HR for older individuals is greater than the increase in maximal SBP when compared to younger adults, older individuals have a lower rate-pressure product at maximal exercise. **Table 2** presents typical cardiovascular values at maximal exercise in young and old adults of both sexes.

**Table 2.** Cardiovascular Responses to Maximal Exercise in Young and Older Adults

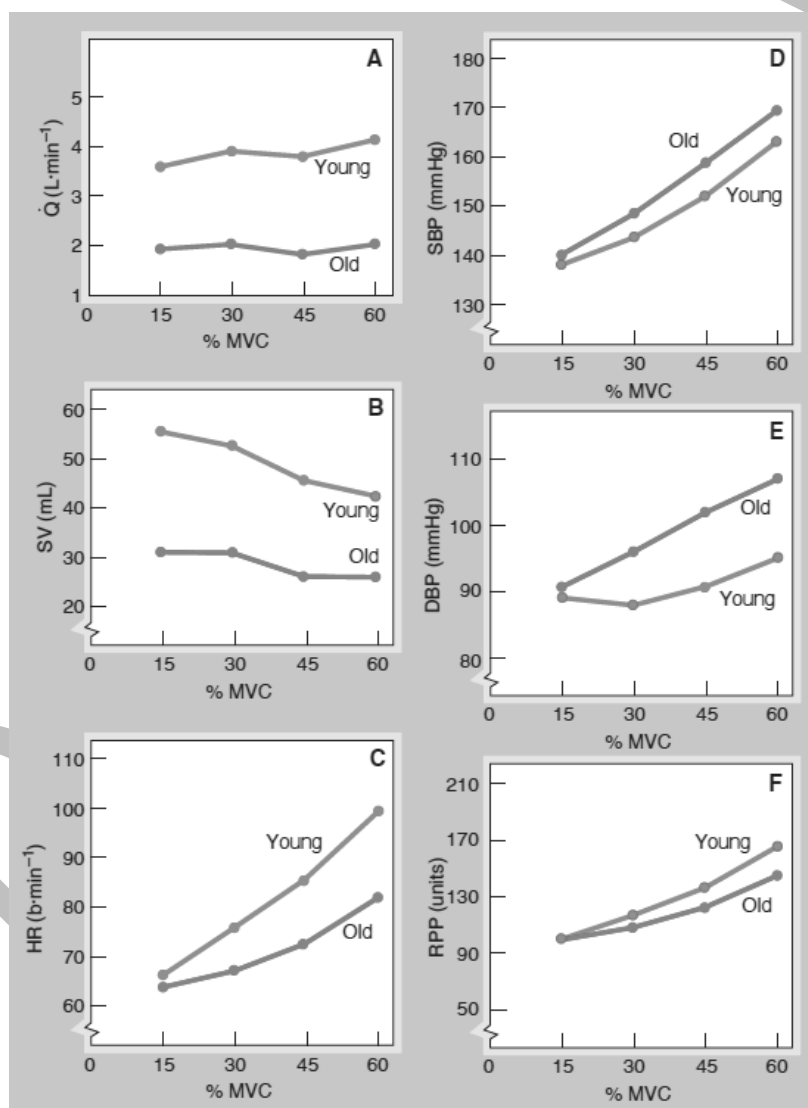
Variable	Men		Women	
	25 years	65 years	25 years	65 years
Q (L.min <sup>-1</sup> )	25	16	18	12
SV (mL.b <sup>-1</sup> )	128	100	92	75
HR (b.min <sup>-1</sup> )	195	155	195	155
VO <sub>2</sub> [L.min <sup>-1</sup> ]	3.5	2.5	2.5	1.5
SBP [mmHg]	190	200	190	200
DBP (mmHg)	70	84	64	84
MAP (mmHg)	130	143	128	143
TPR	5.2	8.9	7.1	11.9
RPP	371	310	371	310

Adapted from: Ogawa, T., R. J. Spina, W. H. Martin, W. M. Kohrt, K. B. Schechtman, J. O. Holloszy, & A. A. Ehsani: Effects of ageing, sex, and physical training on cardiovascular responses to exercise. *Circulation*. 86:494–503 (1992).



## Static Exercise

Many studies have described the cardiovascular responses to static exercise in older adults (Goldstraw and Warren, 1985; Petrofsky and Lind, 1975; Sagiv et al., 1988; VanLoan et al., 1989). As an example, **Figure 12** depicts the cardiovascular responses of young and old men to sustained handgrip and leg extension exercise over a range of submaximal static workloads (VanLoan et al., 1989). Note that cardiac output (**Figure 12A**) and SV (**Figure 12B**) values are lower than normally reported, because of the measurement technique.



**Figure 12.** Cardiovascular Response of Males by Age to Static Exercise. A. Cardiac output (Q). B. Stroke volume (SV). C. Heart rate (HR). D. Systolic blood pressure (SBP). E. Diastolic blood pressure (DBP). F. Rate-pressure product (RPP).



However, the relative differences between the responses of the young and the older participants show that cardiac output, SV, and HR (**Figure 12C**) were lower for the older men than for the younger men at each intensity. In contrast, BP responses (**Figure 12D and 12E**) were higher for the older men at each intensity. As with dynamic aerobic exercise, the differences in the cardiovascular responses between the two age groups are probably due to an age-related increase in resistance due to a loss of elasticity in the vasculature and a decreased ability of the myocardium to stretch and contract forcibly (VanLoan et al., 1989). The rate-pressure product (**Figure 12F**) was higher for the younger participants than for the older participants at 30%, 45%, and 60% MVC. The small difference in rate-pressure product reflected a higher HR in younger participants at each intensity of contraction, which was not completely offset by a lower SBP in the younger participants.

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## Key points

1. During short-term, light to moderate aerobic exercise, cardiac output (Q), stroke volume (SV), heart rate (HR), systolic blood pressure (SBP), and rate pressure product (RPP) increase rapidly at the onset of exercise and reach steady state within approximately 2 minutes. Diastolic blood pressure (DBP) remains relatively unchanged, and resistance decreases rapidly and then plateaus.
2. During long-term, moderate to heavy aerobic exercise, Q, SV, HR, SBP, and RPP increase rapidly. Once a steady state is achieved, cardiac output remains relatively constant owing to the downward drift of SV and the upward drift of HR. SBP and resistance may also drift downward during prolonged, heavy work. This cardiovascular drift is associated with rising body temperature.
3. During incremental exercise to maximum, Q, HR, SBP, and RPP increase in a rectilinear fashion with increasing workload. SV increases initially and then plateaus at a workload corresponding to approximately 40–50% of  $VO_{2max}$  in normally active adults and children. DBP remains relatively constant throughout an incremental exercise test. Resistance decreases rapidly with the onset of exercise and reaches its lowest value at maximal exercise.
4. The decrease in resistance during aerobic exercise has two important implications. It allows greater blood flow to the working muscles and keeps blood pressure from rising excessively. The increase in cardiac output would produce a much greater rise in blood pressure if it were not for the fact that there is a simultaneous decrease in resistance.
5. The highest oxygen consumption during an incremental test may or may not represent an actual maximal value. The term  $VO_{2peak}$  is used to represent the highest value obtained during the test if the tester is not certain that a true maximal value was achieved. Specific criteria should be used to determine whether the test is truly a maximal test.



6. Maximal oxygen uptake may be limited by a system (or step) along the pathway of bringing oxygen into the body and delivering it to the mitochondria to produce ATP. Although factors in each of these systems may limit the  $VO_{2max}$ , research suggests that cardiac output is the limiting factor in  $VO_{2max}$ .
7. Blood volume decreases during aerobic exercise. Most of the decrease occurs within the first 10 minutes of activity and depends on exercise intensity. A decrease of 10% of blood volume is not uncommon.
8. SV initially increases during dynamic aerobic exercise and then plateaus at a level that corresponds to 40–50% of  $VO_{2max}$ . The increase in SV results from changes in end-diastolic volume (EDV) and end-systolic volume (ESV). EDV increases primarily because the active muscle pump returns blood to the heart. ESV decreases owing to augmented contractility of the heart, thus ejecting more blood, and leaving less blood in the ventricle.
9. As adults age, their cardiovascular responses change. Maximal Q, SV, HR, and  $VO_{2max}$  decrease. Maximal SBP, DBP, and mean arterial pressure (MAP) increase.
10. Static exercise is characterized by modest increases in HR and Q and exaggerated increases in SBP, DBP, and MAP, known as the pressor response.
11. Dynamic resistance exercise results in a modest increase in Q, an increase in HR, little change or a decrease in SV, and a large increase in blood pressure.



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