Journal of Strength and Conditioning Research, 1999, 13(1), 90–96

A Review of the Acute Cardiovascular Responses to Resistance Exercise of Healthy Young and Older Adults

JERRY J. MAYO AND LEN KRAVITZ

Exercise Science and Leisure Management Department, The University of Mississippi, Turner Center, University, Mississippi 38677.

ABSTRACT

The purpose of this review was to summarize the acute cardiovascular responses of healthy young and older adults to resistance exercise and to review studies that have compared healthy younger and older populations. Intensity, duration, and active muscle mass are known to play important roles in the degree of pressor response elicited during resistance exercise in both young and older participants. Systolic, diastolic, and mean arterial pressure as well as heart rate rise in response to resistance exercise. Stroke volume generally remains unchanged but may significantly decrease with greater exercise intensity and active muscle mass. Cardiovascular variables such as cardiac output, rate–pressure product, and oxygen consumption increase comparably in the 2 groups. Also, total peripheral resistance may increase or decrease based on involved muscle mass and resistance type. Findings from this review suggest that acute cardiovascular responses to resistance exercise are similar in healthy young and older adults. This supports the inclusion of resistance exercise as part of an overall fitness program designed for healthy older adults.

Key Words: blood pressure, heart rate, isometric

Reference Data: Mayo, J.J., and L. Kravitz. A review of the acute cardiovascular responses to resistance exercise of healthy young and older adults. *J. Strength and Cond. Res. 13(1):90–96. 1999.*

Introduction

Many positive physiological adaptations occur in older persons as a result of resistance exercise. Physical changes, such as muscle hypertrophy, decreased adiposity, and enhanced muscular strength, have been reported in several contemporary studies (3, 9, 15, 32). These adaptations have the cumulative effect of increasing quality of life while slowing some age-related processes.

Until recently, resistance exercise in the older population had been considered risky, in part because of exaggerated responses secondary to the placement of large demands on the cardiovascular system (34). However, limited research has been completed that compares the acute cardiovascular responses to resistance exercise of younger and older adults. The objective of this article is to present a concise summary of the acute cardiovascular responses to resistance exercise and to review studies that have compared healthy younger and older populations. Results will provide support for the participation of older adults in resistance exercise.

Factors Affecting the Acute Responses of Resistance Exercise

During resistance exercise, a number of physiological changes occur in the heart rate, blood pressure, metabolic rate, hormone secretion, nerve conductivity, muscle activity, and respiration. These distinctive acute responses are influenced by numerous factors, including active muscle mass, relative intensity of the exercise, number of repetitions, type of exercise (isometric, isotonic, or isokinetic), duration of exercise, use of rest periods between exercises, and the intermittent nature of the exercise performance (4, 13, 17, 35). The varying methods and protocols used by some investigators add confusion to interpretation of the acute physiological responses to resistance exercise. Another challenge noted in investigating the effects of different types of exercise is comparison of the amount of work accomplished; there are inherent differences among these amounts that correspond to the different types of exercise (19). Although resistance exercise involves static and dynamic activity, it is misleading to assume that the specific cardiovascular responses to isometric exercise are completely equivalent to those associated with traditional dynamic resistance exercise (13). However, MacDougall and associates (24) suggest that resistance exercise is analogous to a series of static contractions that are performed dynamically. The major-

ity of research comparing the effects of age on acute resistance exercise has been conducted using isometric exercise (36, 38–40, 43, 49, 51); this is probably due to the more stringent control associated with this exercise and with the ability to generalize this type of exercise to everyday tasks (38).

Heart Rate Response

The heart rate (HR) response to exercise encompasses an integration of the cardiovascular, muscular, and central nervous systems (35). Contraction of skeletal muscle, activation of afferent fibers by stretch, and increase in metabolites produced from raised cellular activity can contribute to changes in HR during resistance exercise (48). In addition, there is an increase in sympathetic stimulation (4), a rise in plasma catecholamines (5), and a decrease in parasympathetic drive (26) at the onset of exercise. Collins et al. (4) propose that the increased sympathetic activity can be partially attributed to the sustained static component of resistance exercise and to the performance of the Valsalva maneuver. The Valsalva maneuver has been shown to cause tachycardia and sympathetic nerve activity because of a decreased sympathoinhibition in both arterial and low pressure baroreceptors (6). As well, a decrease in muscle cell pH may stimulate chemosensitive afferent fibers, thereby elevating HR (52). The forces and intensity of resistance exercise (16), greater involvement of the fast-twitch muscle fibers (4), and size of activated muscle mass (35) may also stimulate increases in HR.

Taylor and colleagues (49) had 14 younger (26 ± 1.0 years) and 14 older (66 ± 1.0 years) healthy males perform a 30% maximal voluntary contraction (MVC) isometric handgrip exercise to the point of exhaustion (inability to maintain target force). MVC for the younger (402 ± 20 N) and older (392 ± 20 N) subjects was similar, as was time to exhaustion (315 ± 27 seconds in younger vs. 339 ± 17 seconds in older men). During the sustained trial, electromyography activity and ratings of perceived exertion were similar between groups, indicating comparable voluntary efforts. HR rose above baseline within the first 20% of the exercise duration and continued to increase ($p < 0.05$) throughout the handgrip in both groups. However, at every point during the exercise, the absolute level of HR was lower and the magnitude of the increase from control was less ($p < 0.05$) in the older subjects.

Similarly, Petrofsky and Lind (33) showed that the increase in HR during isometric exercise is less in older groups. In this investigation, 100 men, aged 22 to 66 years and grouped by decade (20–29, 30–39, 40–49, and 50–62 years) performed a 40% MVC handgrip exercise to the point of exhaustion. The mean MVC strength of the men did not differ with increasing decades (48.8, 52.5, 48.5, and 47.5 kg, respectively). For all subjects, HR increased steadily from a mean HR (at rest) of 80 beats per minute (bpm) to an average peak value of 100 bpm. As with Taylor et al. (49), the largest increase in average HR took place in the first 20% of the duration of exercise, when it rose from an average of 80 to 90 bpm during the contraction. The greatest increase in mean HR at the end of the 40% MVC was found in the youngest decade (114 bpm), followed by 105, 101, and 100 bpm in each increasing decade.

Another comparative investigation (28) of 10 younger (25.3 ± 1.3 years) and 12 middle-aged (46.8 ± 0.8 years) men who performed 5-minute static forearm contractions at 33% MVC demonstrated a comparable (but not a significant) HR response to that observed by Taylor et al. (49) and Petrofsky and Lind (33). HR increased linearly from mean resting values of 58.3 ± 1.9 bpm in the older group and 57.3 ± 3.6 bpm in the younger group to 89.6 ± 4.9 bpm and 95.7 ± 5.5 bpm, respectively, at the fifth minute of the contraction.

Van Loan et al. (51) compared 24 younger (23.7 ± 3.8 years) and 24 older (57.8 ± 5.6 years) males performing small muscle (finger flexor) and large muscle (leg extensor) isometric muscle contractions (45-second duration) at 4 different intensities (15, 30, 45, and 60% MVC). Results indicated that higher HR responses were found during leg extension exercises at each increase in % MVC workload and in the younger subjects (with both muscle groups) at all exercise levels. Researchers noted that the fastest rise in HR occurred at the higher % MVC.

Sagiv et al. (39) reported results that contrasted with those of Van Loan et al. (51) in an investigation comparing the effects of an isometric deadlift at 30% MVC (held for 3 minutes) in 25 younger (25 ± 4.0 years), 25 older (51.3 ± 3.2 years), and 25 elderly (68.4 ± 4.0 years) subjects. Groups differed significantly in the MVC of the dead lift (136.6 ± 18.0, 111.3 ± 7.3, 89.6 ± 15.3 kg for the younger, older, and elderly group, respectively) and in HR for the 30% MVC (82.8 ± 19.0, 88.2 ± 8.0, and 104.6 ± 33.0 bpm for the younger, older, and elderly groups, respectively). The authors suggested that the increase in HR in the older and elderly subjects was attributable to the their limited ability to increase stroke volume (SV), whereas young adults increased both HR and SV to augment cardiac output (CO).

In order to determine the acute HR response to training status and age, Sagiv et al. (38) compared the effect of a dead lift isometric exercise, held at 30% of MVC for 3 minutes, in 14 younger (28 ± 6.0 years) and 10 older (51 ± 3.0 years) weight lifters and in 14 younger (29 ± 6.0 years) and 10 older (52 ± 1.1 years) ''normal'' individuals. During the dead lift, all subjects increased mean peak HR significantly from rest (but not between groups), attaining values of 94.2 ± 8, 96.0 ± 9, 90.4 ± 10, and 89.1 ± 6 bpm for the young

normals, young weight lifters, old normals, and old weight lifters, respectively. Age comparison results are similar to those of other studies using the isometric dead lift exercise at 30% MVC, as conducted by Sagiv et al. (40, 42).

The lower HR in older men during static effort supports the view that the CO is smaller compared with that of the younger men, assuming that they do not increase their SV (33). This blunted tachycardia in the older persons may be attributable to lesser withdrawal of cardiac vagal tone and to impairment in cardiac sympathetic stimulation or to both (49). Some of the discrepancies in HR responses between younger and older men (reported above) may be partially attributable to the varying effects of the different-sized muscles used (forearm vs. legs and back). It has been shown that the magnitude of the HR response is directly influenced by the contracting muscle mass (26). When the % MVC increases above 20% and is continually maintained, the HR will increase in proportion to the duration, tension exerted, and amount of muscle mass utilized (39, 43, 46). In addition, the dead lift requires the use of several ancillary muscles for postural control, as compared with the handgrip exercise, which is typically performed while the individual is in the supine position.

Stroke Volume

SV is influenced by preload, afterload, cardiac contractility, and HR (2). Also, extrinsic factors, such as catacholamine release, may increase the inotropic state of the heart (7). Resistance exercise, in contrast to aerobic exercise, imposes an increased afterload stress on the heart, which produces only minimal variations in SV (13, 22, 31). In general, SV remains unchanged during light work (11, 38), while reductions occur at a greater % MVC (1, 20). High intensity muscle contraction results in decreased preload, increased afterload, and overall diminished SV response (29). The accentuated intrathoracic and intra-abdominal pressures from the Valsalva maneuver may further inhibit venous return, thereby significantly decreasing SV (20).

Research reporting SV comparisons using submaximal isometric contractions for 3 minutes has shown minor increases from rest, ranging from 2.6–6.2% for younger and from 1.0–13% for older adults (37, 38, 40, 42). Using echocardiography, Sagiv et al. (42) observed slight increases of 6 and 13%, respectively, in the younger (23 ± 3.0 years) and older (67 ± 4.0 years) subjects during isometric handgrip and negligible increases from rest while performing a dead lift exercise (younger 4%; elderly 3%).

Other researchers, utilizing isometric exercise for extended periods, have observed significant reductions in SV (28, 49). Using impedance cardiography, Taylor et al. (49) observed decreases ($p < 0.05$) in SV but failed to note age-related changes in SV or in catacholamine response during 5 minutes of isometric handgrip exercise at 30% MVC. Likewise, McDermott et al. (28) noted a 36% decrease in SV for younger subjects (from rest), compared with a 4% decline in older subjects while subjects sustained a maximal isometric handgrip for 5 minutes. The authors attributed these differences to a slightly reduced total peripheral resistance (TPR) and to the greater epinephrine levels found in the older men. Van Loan et al. (51) reported lower ($p < 0.05$) SV measurements between age groups when he employed small and large muscle isometric exercises that subjects maintained for 45 seconds. However, resting SV was significantly different between the 2 age groups (55.6 ± 3.0 vs. 34.3 ± 2.0 ml for younger and older adults, respectively). Sagiv et al. (38) suggest that the slight reductions in SV at rest are due to changes in left ventricular systolic function, changes which are attributable to age alone. When expressed as the percent change from rest, these differences were negated. For example, decreases in SV at 15, 30, 45, and 60% MVC during leg extensor exercise were 13.1, 17.8, 25.9, and 27.3% for the younger subjects and 11.4, 21.6, 22.7, and 24.2% for the older subjects at each respective exercise intensity.

SV responses during resistance exercise reflect the interplay of factors controlling end-systolic volume and end-diastolic volume. Acute resistance exercise affects circulatory dynamics by placing an increased afterload stress on the heart; yet, there appear to be no age-related differences in SV responses between healthy younger and older adults at similar relative intensities (28, 37, 38, 40, 42, 49). The observed SV increases to resistance exercise are present during smaller muscle group exercises that are performed at low percentages of MVC (42). Several investigators indicate that increased intensity and/or the utilization of greater active muscle mass during resistance exercise produce unchanged or significantly lower SV measurements compared to those obtained during resting levels (28, 37–39, 49, 51).

Cardiac Output

CO is based on both inotropic and chronotropic responses (8) that determine the rate of oxygen delivery to the skeletal muscle (7). Peripheral circulation factors control venous return to the heart and thus influence CO through the Frank–Starling mechanism (10). Unlike aerobic activities, the acute responses to resistance exercise do not extensively activate the Frank–Starling mechanism (31). Arterial occlusion in response to muscular contraction elevates mean arterial pressure (MAP), creating an increased systemic resistance, which impedes venous return of blood to the heart (29). These acute responses to resistance exercise limit the influence of SV, producing only mild (1, 29) to

moderate (11, 20) increases in the CO that is primarily mediated by an increased HR.

CO has been shown to increase mildly from rest in younger (5.8–10.71 L·min^{-1}) and older (5.91–7.98 L·min^{-1}) men during isometric contractions at 30% MVC (1, 37, 42). However, it may rise even higher using resistance at a greater % MVC or during maximal efforts performed to fatigue (49). Three studies reporting CO comparisons between age groups demonstrate no differences (37, 42, 49), while 1 study described significantly higher responses in the younger group (38). These changes in CO are related to the inability to significantly augment acute SV responses during resistance exercise. Thus, to maintain or increase CO, a significant increase in HR will generally occur to overcome the systemic resistance imposed by the cardiac afterload.

Blood Pressure

Resistance exercise induces a sudden, simultaneous elevation of systolic blood pressure (SBP) and diastolic blood pressure (DBP), a process known as the pressor response (8, 13, 23, 31), that is considered discordant to the amount of work performed by the contracting muscle (23, 50). Since both systolic and diastolic pressures increase, a concomitant rise in MAP occurs, with only modest changes in CO (13, 31). The high intramuscular pressures associated with resistance exercise promote the mechanical compression of arterial vessels, occluding blood flow to the active tissues (23). This arterial impedance has the effect of increasing metabolic by-products such as H^+, lactic acid, and adenosine diphosphate. The accretion of by-products activates nerve endings, which in turn elevates MAP (pressor reflex) in an attempt to increase blood flow (10).

The mechanism behind the dramatic rise in blood pressure (BP) during acute resistance exercise is comprised of both central and peripheral components (21, 27). The central mechanism arises from the supraspinal region of the brain and is directly related to level of central motor command (voluntary effort), while the peripheral component originates in the active muscle, and its stimulation is commensurate with muscular force production and the amount of active muscle mass (30, 44).

Studies reporting BP responses to resistance exercise in younger and elderly subjects show dissimilar results. Some studies report no significant age effects (28, 39, 40, 42, 43, 49, 53), while others demonstrate differences between groups (33, 51). Sagiv et al. (42) measured BP of 10 younger (28.0 ± 3.0 years) and 10 older (67.0 ± 4.0 years) healthy males while performing isometric exercises at 30% MVC for 3 minutes. Mean resting auscultatory blood pressures (mean systolic/diastolic BP) were similar between the younger (119/73 mm Hg) and older (119/70 mm Hg) groups. For isometric handgrip exercise, no significant differences in BP were reported between younger (149/95 mm Hg) and older (158/101 mm Hg) subjects. Additionally, performance of isometric dead lift exercise produced higher BP responses compared with those produced by the handgrip exercises, with similar arterial pressures attained between groups (younger = 179/108 mm Hg; older = 182/106 mm Hg). These results are supported by others (37–41, 43). Taylor et al. (49) also reported no differences in BP responses between younger (N = 14, 26 ± 1.0 years) and older (N = 14, 66 ± 1.0 years) men while they were executing isometric handgrip exercises to the point of fatigue. McDermott et al. (28) measured BP by auscultation each minute during 5 minutes of isometric handgrip contraction at 33% MVC, observing progressive increases from control levels (younger = 128/83 mm Hg; older = 117/87 mm Hg) to a peak level of 191/142 mm Hg and 191/137 mm Hg in younger (25.3 ± 1.3 years) and older (46.8 ± 0.8 years) subjects, respectively.

Using dynamic resistance exercise, Wescott and Howes (53) compared auscultatory BP in younger (<38 years) and older (>38 years) subjects (N = 24; 13 males, 11 females). Subjects performed single arm curls using 3 load assignments based on a 10-repetition maximum (RM). Exercise intensity was separated into light (10 RM − 4.53 kg), moderate (10 RM − 2.26 kg), and heavy (10 RM) trials. When grouped by age, the older subjects elicited substantially higher SBP in all 3 exercise trials. But, expressed as a percent increase above baseline (% change), differences between groups disappeared as a result of elevated resting SBP within the older group. Increases in SBP from rest for younger subjects were 13.5, 20.9, and 33%, while the older group recorded 18.0, 23.2, and 34.4% elevations for the light, moderate, and heavy loads, respectively.

In contrast, Van Loan et al. (51) found a significant but modest increase in BP (10 mm Hg for SBP and 7 mm Hg for DBP) between the younger (23.7 ± 3.8 years) and older (57.8 ± 5.6 years) subjects. Similarly, Petrofsky and Lind (33) observed increased SBP (no change in DBP) in older (>50 years) compared to younger (<50 years) male industrial workers. It should be noted that the resting SBPs were also significantly higher in the older adults.

It can be concluded from the existing literature that BP responses are similar for healthy younger and older adults (38, 39, 42, 49). In both groups, BP rapidly increases, with the magnitude of the response being driven by central motor command and by the relative percent of muscular effort (24, 45). The majority of studies report progressive increases in BP relative to intensities of effort and greater muscle mass involvement in both groups (28, 35, 40, 41, 43 47, 51). However, 2 studies comparing healthy younger and older

groups report small (<10 mm Hg) elevations in the older adults (32, 49), which may be attributed to differences in resting BP and not to changes induced by the resistance exercise.

Rate Pressure Product

Rate pressure product (RPP), or double product (HR × peak SBP), is regarded as an important noninvasive means of estimating myocardial oxygen demand (8, 18). A significant increase in RPP is produced during moderate to heavy resistance exercise in response to coupled increases in HR and SBP. Because HR changes occur less rapidly compared with those of SBP, HR is the most important determinant of myocardial oxygen demand (8). The increases in myocardial work during resistance exercise have been reported to be comparable between resistance types (11, 19).

Older adults have achieved RPPs comparable to those of younger groups, which reflects the comparable HR and SBP responses of the 2 groups. Sagiv et al. (37) saw similar rises in RPP in younger and older groups from pre-exercise levels of 89 ± 12 and 90 ± 10 to 164 ± 18 and 176 ± 20 during peak exercise, respectively. Van Loan et al. (51) reported that RPP was slightly higher in younger compared with older subjects during isometric handgrip exercise at 30, 45, and 60% of MVC. The authors suggest that this is attributable to the higher HR achieved by the younger subjects.

Total Peripheral Resistance

Total peripheral resistance (TPR) represents the impedance to blood flow through the systemic circulation. Factors affecting TPR include blood viscosity, hydrostatic pressure, and, most importantly, vessel radius (7). The predominant controls of TPR during resistance exercise are the vasodilator and vasoconstrictor mechanisms of the sympathetic nervous system. Additionally, humoral (catacholamine, prostaglandins) and local control (pH, lactic acid concentration, PCO_2) factors influence resistance to peripheral blood flow (7). TPR during resistance exercise has been shown to increase (11, 20, 49), while other investigators have found decreases (11, 22). This discrepancy is purported to be the result of differences in active muscle mass (22).

Taylor et al. (49) reported increases in TPR for both age groups (younger = 21%; older = 12%) during the final portion of an isometric handgrip maintained at 30% MVC until the point of exhaustion. No significant differences in TPR were observed between groups expressed in absolute or as a percent change from rest. Similarly, McDermott et al. (28) and Sagiv et al. (37) have corroborated these results, finding a lesser increase in TPR in healthy older men compared with younger males. Contrasting results were found by Sagiv et al. (38), who observed modest but significant differences between groups with increases in TPR of 10 and 16%, respectively, for younger and older men. The increased arterial impedance in the older group was likely responsible for the observed differences in this study (38).

Although limited, age group comparison studies suggest that TPR is similarly affected in younger and older groups (28, 38, 47). TPR is predominately unchanged or may increase during isometric exercises (1, 28, 38, 47). In contrast, 2 studies have shown decreases in TPR through the use of large muscle dynamic resistance exercise. Lewis et al. (22) attribute this discrepancy to the process in which the metabolically activated vasodilation drive overcomes the vasoconstrictor drive observed in larger muscle masses (not smaller muscles), especially at greater exercise intensities.

Oxygen Consumption

Oxygen consumption ($\dot{V}O_2$) increases commensurate with exercise intensity. $\dot{V}O_2$ is controlled by the rate of oxygen (O_2) transport, the carrying capacity of O_2 in the blood, and O_2 extraction by the tissue (2). Several studies have observed small increases in $\dot{V}O_2$ secondary to resistance exercise (1, 47), while others report greater elevations (4), with the magnitude of the response being associated with the type of resistance exercise and with the intensity of contraction. The elevations in intramuscular pressure associated with static contractions limit muscle perfusion pressure, restricting blood flow and O_2 delivery (22). However, intermittent contractions performed in dynamic resistance exercise promote a slightly higher $\dot{V}O_2$ response. For example, $\dot{V}O_2$ during circuit training has routinely been observed to be 30–50% of $\dot{V}O_2$ max (4, 12, 14, 54).

Reports comparing $\dot{V}O_2$ among younger and older subjects have found increases ($p < 0.05$) in younger men (38–40, 43), with 1 study revealing no differences (37). Sagiv et al. (39) demonstrated a progressively lower $\dot{V}O_2$ response with age. In younger (27.4 ± 2.3 years), older (51.0 ± 3.2 years), and elderly (67.8 ± 3.8 years) males who performed an isometric dead lift for 3 minutes at 30% MVC, $\dot{V}O_2$ measurements were 10.71 ± 3.5, 7.98 ± 1.17, and 7.90 ± 2.26 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively. Sagiv et al. (42) suggest that these results may be caused by the greater amounts of absolute tension developed or by enhanced O_2 delivery and extraction capabilities in younger men.

Practical Applications

Resistance exercise has become quite popular among older adults over the last decade; research in this area has primarily focused on the chronic effects of resistance training. This article adds support to the growing body of research that is establishing the safety and efficacy of encouraging older populations to incorpo-

rate resistance exercise into their daily lives. The results of this review indicate that low-to-moderate intensity resistance exercise elicits similar cardiovascular responses in healthy younger and older populations. In all of the cardiovascular variables reviewed, no exaggerated responses, which might indicate significant health risks to older adults who are performing resistance exercise, were observed. Since the fastest growing segment of the population consists of those over 65 years of age, interested applied exercise professionals are encouraged to reach out to these members of society with resistance training programs that may improve bodily function and quality of life.

References

1. Bezucha, G.R., M.C. Lenser, P.G. Hanson, and F.J. Nagle. Comparison of hemodynamic responses to static and dynamic exercise. *J. Appl. Physiol.* 53:1589–1593. 1982.
2. Brooks, G.A., T.D. Fahey, and T.P. White. *Exercise Physiology: Human Bioenergetics and Its Applications.* Mountain View, CA: Mayfield, 1996.
3. Charette, S.L., L. McEvoy, G. Pyka, C. Snow-Harter, C. Guido, R.A. Wiswell, and R. Marcus. Muscle hypertrophy response to resistance training in older women. *J. Appl. Physiol.* 70: 1912–1916. 1991.
4. Collins, M.A., K.J. Cureton, D.W. Hill, and C.A. Ray. Relationship of heart rate to oxygen uptake during weight lifting exercise. *Med. Sci. Sports Exerc.* 23:636-640. 1991.
5. Davies, C.T.M., J. Few, G. Foster, and A.J. Sargeant. Plasma catecholamine concentration during dynamic exercise involving different muscle groups. *Eur. J. Appl. Physiol.* 32:195–206. 1974.
6. Delius, W., K.-E. Hagbarth, A. Hongell, and B.G. Wallin. Maneuvers affecting sympathetic outflow in human muscle nerves. *Acta. Physiol. Scand.* 84:82–94. 1972.
7. Durstine, J.L., R.R. Pate, and J.D. Branch. Cardiorespiratory responses to acute exercise. In: *ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription* (2nd ed.). Philadelphia: Lea & Febiger, 1993, p. 66–74.
8. Fardy, P.S. Isometric exercise and the cardiovascular system. *Phys. Sports Med.* 9:43–56. 1981.
9. Fiatarone, M.A., E.C. Marks, and N.D. Ryan. High-intensity strength training in nonagenarians: Effects on skeletal muscle. *JAMA* 263:3029–3034. 1990.
10. Guyton, A.C. *Textbook of Medical Physiology.* Philadelphia: W.B. Saunders Company, 1991.
11. Haennel, R.G., G.D. Snydmiller, K.K. Teo, P.V. Greenwood, H.A. Quinney, and T. Kappagoda. Changes in blood pressure and cardiac output during maximal isokinetic exercise. *Arch. Phys. Med. Rehabil.* 73:150–155. 1992.
12. Hempel, L.S., and C.L. Wells. Cardiorespiratory cost of the Nautilus express circuit. *Phys. Sports Med.* 13:82–96. 1985.
13. Hill, D.W., and D. Butler. Haemodynamic responses to weightlifting exercise. *Sports Med.* 12:1–7. 1991.
14. Hurley, B.F., D.A. Seals, L.J. Ehsani, G.P. Carter, J.M. Dalsky, and J.O. Holloszy. Effects of high-intensity strength training on cardiovascular function. *Med. Sci. Sports Exerc.* 16:483–488. 1984.
15. Hurley, B.F., R.A. Rendmond, R.E. Pratley, M.S. Treuth, M.A. Rodgers, and A.P. Goldberg. Effects of strength training on muscle hypertrophy and muscle celldisruption in older men. *Int. J. Sports Med.* 16:378–384. 1995.
16. Keul, J., G. Haralambie, M. Bruder, and H.-J. Gottstein. The effect of weight lifting exercise on heart rate and metabolism in experienced weight lifters. *Med. Sci. Sports Exerc.* 10:13–15. 1978.
17. Kispert, C.P., and D.H. Nielsen. Normal cardiopulmonary responses to acute- and chronic strengthening and endurance exercises. *Phys. Ther.* 65:1831. 1985.
18. Kitamura, K., C.R. Jorgensen, G.L. Fredarick, H.L. Taylor, and Y. Wang. Hemodynamic correlates of myocardial oxygen consumption during uprightexercise. *J. Appl. Physiol.* 32:516–522. 1972.
19. Kliener, D.M., D.L. Blessing, W.R. Davis, and J.W. Mitchell. Acute cardiovascular responses to various forms of resistance exercise. *J. Strength Cond. Res.* 10:56–61. 1996.
20. Lentini, A.C., R.S. McKelvie, N. McCartney, C.W. Tomlinson, and J.D. MacDougall. Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise. *J. Appl. Physiol.* 75:2703–2710. 1993.
21. Leonard, B., J.H. Mitchell, M. Mizuno, N. Rube, B. Saltin, and N.H. Secher. Partial neuromuscular blockade and cardiovascular responses to static exercise inman. *J. Physiol.* 359:365–379. 1985.
22. Lewis, S.F., P.G. Snell, F. Taylor, M. Hamra, R.M. Graham, W.A. Pettinger, and C.G. Blomqvist. Role of muscle mass and mode of contraction in circulatory responses to exercise. *J. Appl. Physiol.* 58:146–151. 1985.
23. Lind, A.R., G.W. Taylor, P.W. Humphreys, B.M. Kenneally, and K.W. Donald. The circulatory effects of sustained voluntary muscle contraction. *Clin. Sci.* 27:229–244. 1964.
24. MacDougall, J.D., D. Tuxen, D.G. Sale, D.E. Moroz, and J.R. Sutton. Arterial blood pressure response to heavy resistance exercise. *J. Appl. Physiol.* 58:785-790. 1985.
25. MacDougall, J.D., R.S. McKelvie, D.E. Moroz, D.G. Sale, N. McCartney, and F. Buick. Factors affecting blood pressure during heavy weight lifiting and static contractions. *J. Appl. Physiol.* 73:1590–1597. 1992.
26. McCartney, N., R.S. McKelvie, J. Martin, D.G. Sale, and J.D. MacDougall. Weight-training induced attenuation of the circulatory response of older males to weight lifting. *J. Appl. Physiol.* 74:1056–1060. 1993.
27. McCloskey, D.I., and J.H. Mitchell. Reflex cardiovascular and respiratory responses originating in exercise muscle. *J. Physiol.* 224:173–186. 1972.
28. McDermott, D.J., W.J. Stekiel, J.J. Barboriak, L.C. Kloth, and J.J. Smith. Effect of age on hemodynamic and metabolic response to static exercise. *J. Appl. Physiol.* 37:923–926. 1974.
29. Miles, D.S., J.J. Owens, J.C. Golden, and R.W. Gotshall. Central and peripheral hemodynamics during maximal leg extension exercise. *Eur. J. Appl. Physiol.* 56:12–17. 1987.
30. Mitchell, J.H. Neural control of the circulation during exericse. *Med. Sci. Sports Exerc.* 22:141–154. 1990.
31. Mitchell, J.H., and K. Wildental. Static (isometric) exercise and the heart: Physiological and clinical considerations. *Ann. Rev. Med.* 25:369–381. 1974.
32. Nichols, J.D., D.K. Omizo, K.K. Peterson, and K.P. Nelson. Efficacy of heavy-resistance training for active women over sixty: Muscular strength, body composition, and program adherence. *J. Am. Ger. Soc.* 41:205–210. 1993.
33. Petrofsky, J.S., and A.R. Lind. Aging, isometric strength and endurance, and cardiovascular responses to static effort. *J. Appl. Physiol.* 38:91–95. 1975.
34. Rosenthal, J. Aging and the cardiovascular system. *Gerontology* 33:S13–8. 1987.
35. Rozenek, R., L. Rosenau, P. Rosenau, and M.H. Stone. The effect of intensity on heart rate and blood lactate response to resistance exercise. *J. Strength Cond. Res.* 7:51–54. 1993.
36. Sagiv, M., A. Rotstein, J. Watkins, L. Climor, and D. Ben-Sira. Effect of body position on the afterload response to sustained exercise. *J. Sports Med. Phys. Fitness* 32:170–174. 1992.
37. Sagiv, M., A. Sagiv, D. Ben-Sira, J. Rudoy, and M. Soudry. The effects of hypertension and aging on left ventricular func-

tion during isometric exercise. *J. Aging Phys. Activity* 4:69–79. 1996.
38. Sagiv, M., A. Sagiv, D. Ben-Sira, S. Ben-Gal, and M. Soudry. Effects of chronic overload training and aging on left ventricular systolic function. *Gerontology* 43:307–315. 1997.
39. Sagiv, M., D. Ben-Sira, and J. Rudoy. Cardiovascular response during upright isometric dead lift in young, older, and elderly healthy men. *Int. J. Sports Med.* 9:134–136. 1988.
40. Sagiv, M., E. Goldhammer, E. Abinader, and J. Rudoy. Aging and the effect of increased after-load on left ventricular contractile state. *Med. Sci. Sports Exerc.* 20:281–284. 1988.
41. Sagiv, M., N. Fisher, A. Yaniv, and J. Rudoy. Effect of running versus isometric training programs on healthy elderly at rest. *Gerontology* 35:72–77. 1989.
42. Sagiv, M., P. Hanson, E. Goldhammer, D. Ben-Sira, and J. Rudoy. Left ventricular and hemodynamic responses during upright isometric exercise in normal young and elderly men. *Gerontology* 34:165–170. 1988.
43. Sagiv, M., P. Hanson, M. Besozzi, and F.J. Nagle. Left ventricular responses to upright isometric handgrip and deadlift in men with coronary artery disease. *Am. J. Cardiol.* 55:1298–1302. 1985.
44. Sale, D.G., D.E. Moroz, R.S. McKelvie, J.D. MacDougall, and N. McCartney. Effect of training on the blood pressure response to weight lifting. *Can. J. Appl. Physiol.* 19:60–74. 1994.
45. Seals, D., P. Chase, and J.A. Taylor. Autonomic mediation of the pressor responses to isometric exercise in humans. *J. Appl. Physiol.* 64:2190–2196. 1988.
46. Seals, D.R., R.A. Washburn, P.G. Hanson, P.L. Painter, and F.J. Nagle. Increased cardiovascular response to static contraction of larger muscle groups. *J. Appl. Physiol.* 54:434–437. 1983.
47. Sharkey, B.J. A physiological comparison of static and phasic exercise. *Res. Q.* 37:520–531. 1966.
48. Stone, H.L., K.J. Dormer, R.D. Foreman, R. Theies, and R.W. Blair. Neuroregulation of the cardiovascular system during exercise. *Fed. Proc.* 44:2271–2278. 1985.
49. Taylor, J.A., G.A. Hand, D.G. Johnson, and D.R. Seals. Sympathoadrenal-circulatory regulation during sustained isometric exercise in young and older men. *Am. J. Physiol.* 261:R1061–1069. 1991.
50. Tuttle, W.W., and S.M. Horvath. Comparison of effects of static and dynamic work on blood pressure and heart rate. *J. Appl. Physiol.* 10:294–296. 1957.
51. Van Loan, M.D., B.H. Massey, R.A. Boileau, T.G. Lohman, J.E. Misner, and P.L. Best. Age as a factor in the hemodynamic responses to isometric exercise. *J. Sports Med. Phys. Fitness* 29: 262–268. 1989.
52. Victor, R.G., L.A. Bertocci, S.L. Pryor, and R.L. Nunally. Sympathetic discharge is coupled to muscle cell pH during exercise in humans. *J. Clin. Invest.* 82:1301-1305. 1988.
53. Wescott, W., and B. Howes. Blood pressure response during weight training exercise. *NSCA J.* 5:67–71. 1983.
54. Wilmore, J.H., R.B. Parr, P. Ward, P.A. Vodak, T.J. Barstow, T.V. Pipes, G. Grimditch, and P. Leslie. Energy cost of circuit weight training. *Med. Sci. Sports Exerc.* 10:75–78. 1978.

Acknowledgments

We would like to thank Linda Chitwood, Ph.D., and John Alvarez, Ph.D., for their suggestions to the original manuscript.