Chapter 8
Effects of Regular Exercise on Arterial Stiffness

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Abbreviations

AHA	American Heart Association
AI	Augmentation index
CVD	Cardiovascular disease
NOS	Nitric oxide synthase
PWV	Pulse wave velocity

Introduction

It is well established that cardiovascular disease (CVD) is the number one cause of mortality in both men and women in most industrialized countries including the United States [1]. Over 80 % of CVD prevalence can be attributed to the disease of blood vessels as coronary artery disease, stroke, and hypertension are all arterial diseases. The most prominent change in the blood vessels that can contribute to the prevalence of CVD is the stiffening or hardening of arteries. Arterial stiffness is an independent predictor of adverse CVD mortality and morbidity [2, 3] and can induce a number of subsequent cardiovascular sequela including hypertension, left ventricular hypertrophy, coronary ischemia, and stroke [4–6].

The exact cause of arterial stiffening is not well understood, but a number of structural and functional elements would likely contribute to this process (Fig. 8.1) [7–9].

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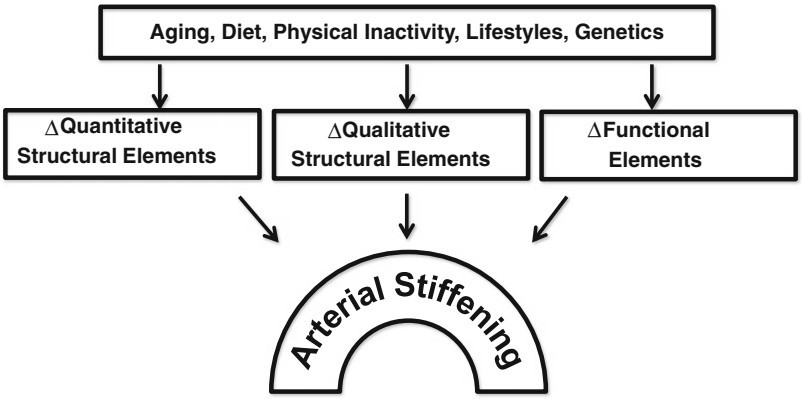

Fig. 8.1 Causes of arterial stiffening

Although epidemiological studies demonstrated relations between arterial stiffening (i.e., arteriosclerosis) and arterial wall thickening (i.e., atherosclerosis) [10], arteries undergo stiffening or hardening independent of atherosclerosis. Marked arterial stiffening with advancing age has been observed in rural Chinese populations where the incidence and prevalence of atherosclerosis are very low [11], in a rigorously screened population in the United States [12–14], and in beagle dogs that do not develop atherosclerosis [15]. Arterial stiffening is not a universal change affecting the entire arterial tree and manifests much more clearly in central elastic or cardiothoracic arteries where the pulsation of pressure pulses must be effectively buffered before it reaches the capillary circulation that lacks defense mechanisms for arterial pulsations [12, 16]. It is thus likely that the mechanisms inducing arterial stiffening would include the interaction between mechanical distension and vasoactive factors.

Purposes of This Chapter

Since CVD has a very long asymptomatic or latent phase of development, primary and secondary prevention is the most effective means to contain the progression and manifestation of CVD [17]. The universal first-line approach for the prevention of CVD is lifestyle modifications including regular exercise [17, 18]. For this reason, there has been increasing interest in evaluating the effects of regular exercise on arterial stiffening [19, 20]. Is habitual aerobic exercise capable of reducing arterial stiffness that is greatly influenced by structural elements in the arterial wall? If so, what physiological factors are responsible for such destiffening effects? What about the influence of resistance training on arterial stiffening? Accordingly, the primary objective of this Chapter is to review and synthesize previous research studies to address the impact of regular exercise on arterial stiffening as an early marker of subclinical CVD.

Key Terminology and Basic Concepts

In this Chapter, aerobic (endurance) exercise and resistance (strength) exercise training studies are discussed separately as these exercise training modalities appear to exert distinct effects on the arterial elasticity. In each section below, cross-sectional findings are discussed first followed by interventional findings.

Much of the confusion in the area of arterial stiffness arises from the different terminologies used to express the elastic properties of arteries. They include arterial stiffness, compliance, distensibility, elasticity, and elastic modulus. The terms "compliance" and "distensibility" are the inverse of "stiffness" in terms of the directions for rigidity. Even though there has been an attempt to standardize the terminologies for arterial stiffness [21], these terms cannot be used interchangeably because each term has different meanings derived from different methodologies as shown below.

Arterial Compliance

The absolute vessel diameter change for a given pressure change.

Arterial Distensibility

The relative diameter vessel change for a given pressure change.

Elastic Modulus

The pressure change required for 100 % stretch from resting vessel diameter.

Methods

There are a number of techniques that have been used to estimate arterial stiffness. Historically, arterial stiffness has been measured in vitro using excised arteries [22, 23]. However, results derived from in vitro measurements may not be applicable to intact vessels [24] because of the sympathetic vasoconstrictor tone and hormonal milieu that the arteries are exposed to in vivo. One of the most frequently used in vivo noninvasive techniques to estimate arterial stiffness is pulse wave velocity (PWV) [22, 25, 26]. PWV is measured from the "foot" of pressure waves recorded

at two points along the path of the arterial pulse wave. The more rapid the pulse wave, the more rigid the artery. PWV can be measured at a variety of arterial segments but the most popular and most established measure of PWV is aortic or carotid-femoral PWV. Another approach is to determine the augmentation index or AI related to the reflected systolic blood pressure waveform obtained on arterial tonometry [27]. AI was used frequently in the past as an index of arterial stiffness, but more recently it is used as an index of arterial wave reflection that is indirectly related to arterial stiffness. Technological advances in ultrasound imaging have significantly improved the image resolution of arteries. When combined with computer-based image analyses and arterial tonometry on the contralateral artery, ultrasound imaging enables robust measurement of arterial distensibility and compliance [13, 28].

In order to review relevant research in this area, a systematic electronic search of the literature on the association between habitual exercise and arterial stiffness was conducted mainly using PubMed. Additionally, cross-referencing of the identified articles was carefully conducted. Although arterial stiffening is clearly an age-associated disease, age-associated changes are difficult to assess in humans especially in relation to the preventive effects of regular or life-long exercise. Accordingly, both cross-sectional and interventional studies were included.

Relevant Research

Aerobic Exercise Training

A number of investigators have reported that arterial stiffness increases during a acute single bout of aerobic exercise [29, 30]. The acute increase in arterial stiffness seems reasonable given the observation that during graded exercise, systolic blood pressure increases markedly while diastolic blood pressure remains unchanged or slightly decreases resulting in a large increase in pulse pressure [31] that is closely associated with arterial stiffness. Following exercise, however, arterial stiffness appears to fall below the baseline levels [32]. The acute effects of exercise on arterial stiffness seem to disappear within a few hours following exercise [32]. Thus, it is reasonable to assume that the effects of regular exercise on arterial stiffness, if any, are not due to a residual effect of the last bout of exercise but rather due to chronic adaptation of cardiovascular system as the measurements are typically performed >24 h after the last bout of aerobic exercise.

Middle aged and older men who performed endurance exercise on a regular basis demonstrate lower levels of aortic PWV and carotid AI than their sedentary peers [14]. We also reported that significant age-related increases in central arterial stiffness were absent in physically active women and that aerobic fitness was strongly and favorably associated with arterial stiffness [12]. These cross-sectional findings provide support for a role of regular aerobic exercise in the primary prevention of arterial stiffening that occurs with advancing age.

Unknown to most, the first intervention study to determine the influence of exercise training on arterial stiffness was conducted in Japan (This is in part because this paper was published only in Japanese) in 1983. A total of 80 healthy young men, who were new recruits for the Japanese self-defense or military school, were studied before and after 9 months of physical training incorporating a variety of exercise modes, including distance running, calisthenics, soccer, handball, judo, and swimming [33]. At the end of the training period, there was a small but significant reduction in aortic PWV, indicating a small reduction in arterial stiffness. We have also reported that a relatively brief period (3 months) of aerobic exercise can increase central arterial compliance in apparently healthy, middle aged and older adults [13, 28]. This improvement was not associated with changes in body weight, adiposity, blood pressure, or plasma cholesterol, indicating a direct effect of habitual exercise on arterial compliance.

Importantly, this small reduction in arterial stiffness was accomplished with an intensity (moderate) and type (walking) of physical activity that can be performed by most, if not all, healthy older adults [13, 28]. Interestingly, the beneficial effect of aerobic training involves only central elastic arteries whose elastic properties dampen fluctuations in pressure and flow [12, 13]. Additionally, the beneficial effects of regular aerobic exercise on arterial compliance are associated with a favorable influence on arterial blood pressure and arterial baroreflex sensitivity [20, 34, 35], indicating that the beneficial effect of regular exercise would extend to sequelae of arterial stiffening. Thus, the beneficial effects of habitual exercise lead not only to arterial destiffening but also to the attenuation of the adverse outcomes caused by arterial stiffening.

Most of the exercise training studies to date have focused on land-based exercises such as walking [13, 28] and cycling [32, 36]. Swimming is an attractive form of exercise as it is easily accessible, inexpensive, and isotonic [37]. Because of the buoyancy of water, compressive stress on joints is small, and orthopedic injury rate is low [38]. Due to cold temperature and increased thermoconductivity of surrounding water, heat-related illness is extremely low [39]. Thus, swimming can be an ideal form of exercise for those at elevated risks of vascular disease, including the elderly, and people with obesity and/or arthritis [37].

In the first cross-sectional study to address the effect of swim training on arterial elasticity [40], arterial compliance of middle-aged and older swimmers was compared with those of runners and sedentary controls. Central artery compliance was greater in swimmers than in age-matched sedentary controls, and the level of arterial compliance was not different between swimmers and runners, suggesting that high levels of regular swimming exercise may prevent arterial stiffening similar to land-based exercises. Subsequently, a swimming exercise intervention study involving previously sedentary middle aged and older adults was conducted. This follow-up intervention study allowed us to confirm the cross-sectional observations by demonstrating that regular swimming exercise produced a 21 % increase in arterial compliance and a 12 % reduction in the β-stiffness index, a measure of arterial stiffness that adjusts for the effect of alterations in distending pressure on arterial diameter, after 3 months of regular swimming exercise [41]. In summary, evidence from

both cross-sectional and interventional studies collectively indicates that regular swimming is beneficial in improving the elasticity of central arteries in middle-aged and older adults.

As discussed above, habitual aerobic exercise is an effective lifestyle intervention for preventing and reversing arterial stiffening for healthy adults. When prescribed to patients with essential hypertension, however, short-term (2–4 months) aerobic exercise interventions may not be as effective in reducing arterial stiffness as in healthy adults. For example, we reported that 3 months of aerobic exercise training composed of walking and jogging produced very small reductions in arterial stiffness in postmenopausal women with elevated systolic blood pressure [35]. Similarly, short-term aerobic exercise training was unable to reduce arterial stiffness in patients with isolated systolic hypertension [42] or in older patients with Stage I hypertension who had been on antihypertensive medications [43].

Currently, exercise intervention studies targeting patients with other diseases are very limited. In one of these studies, 8 weeks of aerobic exercise training did not change aortic PWV and carotid AI in patients with congestive heart failure [44]. Similarly, no changes in PWV were observed after 2 years of exercise training program in patients with type 2 diabetes mellitus [45]. Interestingly, Ikegami et al. [33] observed a trend for the magnitude of reductions in PWV with exercise training to be reduced in direct proportion to initial body fat levels, suggesting that the degree of destiffening effect of exercise may diminish as the CVD risks of participants increase. Clearly, future studies are warranted to investigate the potential efficacy of long-term (>1 year) aerobic exercise intervention on arterial stiffness in populations with CVD. For related discussions on the effects of aerobic exercise on other metabolic risk factors and vascular function, please see Chap. 5 and the Chapters in Part III.

Mechanisms Underlying Exercise-Induced Reductions in Arterial Stiffness

Considering these findings, the question that emerges is, *If habitual aerobic exercise reduces arterial stiffness*, *then what are the physiological mechanisms underlying its effects*? There are three primary elements of the arterial wall that determine its stiffness (Fig. 8.1). They are: (1) quantitative structural elements (e.g., amount/proportion of elastin and collagen); (2) qualitative structural elements (e.g., fracture/fragmentation of elastic lamellae and the cross-linking of collagen and advanced glycation—sometimes called nonenzymatic glycosylation end-products); and (3) functional elements (vasoconstrictor tone exerted by its smooth muscle cells). Any favorable influences of regular aerobic exercise should involve an attenuation or reversal of one or more of the physiological mechanisms contributing to arterial stiffening.

Structural elements, specifically decreased density of the arterial elastin with corresponding increases in collagen content in the arterial wall, play a major role in increases in arterial stiffness [7]. Because the elastin-collagen composition of the

arterial wall changes over a period of years, it is unlikely that this may be a physiological mechanism underlying reductions in arterial stiffness induced by short-term exercise intervention. In fact, using an animal experiment, we have demonstrated that the influence of regular exercise on arterial stiffness does not appear to be mediated by the quantitative changes in arterial wall elastin and collagen [9]. The results from gene microarray analyses are consistent with this finding since the gene expression of structural proteins (e.g., various types of collagens and procollagens) and enzymes that modulate structural proteins and the extracellular matrix (e.g., collagenase, matrix metalloproteinases) did not change significantly with exercise training in the rat aorta [46]. A recent animal study, however, reported that although total collagen content did not change with exercise training, some isoforms of collagen and calcifications were reduced [47]. Thus, we cannot exclude the possibility that qualitative structural elements, including the shift in collagen subtypes and alterations in collagen cross-linking, may play a role in the reductions in arterial stiffness resulting from regular exercise.

A more likely mechanism contributing to the improvements in the elastic properties of arteries with aerobic exercise is the reduction in vasoconstrictor tone exerted by the vascular smooth muscle cells. Because a number of different and interacting vasoactive molecules and peptides could respond to exercise training to influence the contractile states of the vascular smooth muscle cells, it is difficult to elucidate underlying mechanisms using traditional approaches (e.g., pharmacological blockade). In order to identify and confine relevant functional factors responsible for exercise training-induced decreases in arterial stiffness, we relied on the DNA microarray technique (i.e., multiplex lab-on-a-chip that assays large amounts of biological material suing high-throughput screening methods). Microarray provides a powerful and efficient tool by which to compare the differential expression of a large number of genes in a single reaction and enables a systematic analysis of responses of various gene expressions to exercise training. We found that genes associated with nitric oxide synthase (NOS) (along with prostaglandins and C-type natriuretic peptide) were differentially expressed in the aorta of exercise-trained rats [46]. Because the incidence of false positive findings is very high in the microarray analysis, the results were confirmed subsequently using real-time quantitative polymerase chain reaction and protein expressions [46].

Aside from the NO-mediated vasodilation, another important functional element that has been implicated in the pathogenesis of arterial stiffening is sympathetic adrenergic vasoconstrictor tone [48]. The sympathetic nervous system exerts a tonic restraint on the compliance of the common carotid artery, and removal of that restraint produces an immediate increase in its compliance [49]. We assessed the effects of systemic inhibition of α-adrenergic receptors and NOS on arterial compliance before and after 3 months of aerobic exercise training in middle-aged and older adults. Systemic, rather than local, administration of drugs was used in order to target the compliance of "central" (cardiothoracic) arteries, which makes the dominant contribution to the elastic reservoir function of the arterial system [50]. The effect of α-adrenergic receptor tone on the carotid artery significantly decreased following the aerobic exercise training intervention, as evidenced by a diminished

increase in arterial compliance from baseline to phentolamine (i.e., non-specific α-receptor blocker) administration. The NO-dependent vascular tone, however, did not change significantly after aerobic exercise training, as the magnitude of decrease in arterial compliance from the phentolamine administration to the combined phentolamine and L-NMMA (i.e., NOS blocker) administration was similar before and after exercise training [50].

We have also determined whether endothelin-1, a potent endothelium-derived vasoconstrictor peptide, is involved in the mechanisms underlying the increase in arterial compliance with aerobic exercise training [51]. Systemic endothelin-A/B receptor blockade was administered before and after 3 months of exercise training involving middle-aged and older adults. The increase in arterial compliance induced by regular exercise was associated with a corresponding reduction in plasma endothelin-1 concentration as well as the elimination of endothelin-1-mediated vascular tone [51]. These results suggest that aerobic exercise training-induced increases in arterial compliance are mediated, at least in part, through the removal of chronic restraint provided by vasoconstrictor tone and that multiple mechanisms are likely involved in the destiffening process.

Resistance Exercise Training

Prior to 1990, the resistance training modality was emphasized only as a means to develop muscular strength, power, and muscle mass [52, 53]. In recent years, however, statements on physical activity by various health organizations [54–58] have recommended resistance training as an essential part of physical activity preventive and rehabilitative programs. These recommendations are based primarily on the documented impact of resistance training on the attenuation of osteoporosis and sarcopenia (i.e., the age-related loss of muscle mass and strength) [59, 60] as well as on the evidence indicating associations between resistance training and metabolic risk factors [18]. Information concerning the impact of resistance training on vascular function in general, and arterial stiffness in particular, is limited but is emerging. For related discussions on the effects of resistance exercise on other metabolic risk factors and vascular function, please see Chaps. 2 and 6 and the Chapters in Part III as well.

Plasma norepinephrine levels are elevated after a bout of acute resistance exercise, giving rise to the possibility that sympathetic vasoconstrictor tone may also be elevated after resistance exercise [61]. In an attempt to tease out the chronic effects of resistance exercise from the acute effects, we determined the effect of one bout of acute resistance exercise on central arterial compliance [62]. We found that central arterial compliance was decreased immediately and 30 min after acute resistance exercise. These measures returned to baseline levels within 60 min following the bout of resistance exercise. These results suggest that changes in arterial stiffness, if any, that are observed 24–48 h after an exercise bout (typical waiting period for most exercise intervention studies) can be attributed to the chronic effects of resistance exercise training.

Based on a multitude of benefits that resistance training can elicit, it is reasonable to hypothesize that regular resistance exercise would be associated with reduced arterial stiffness. In the first cross-sectional study to address this hypothesis, Bertovic et al. [63] found that young men who performed resistance training on a regular basis demonstrated *lower* levels of systemic arterial compliance than their sedentary peers. We also found in a cross-sectional study that strength-trained middle aged men exhibited *decreased* levels of arterial compliance and that the age-associated reduction in arterial compliance was *greater* in the resistance-trained groups than in sedentary controls [64]. These findings from resistance training studies are in marked contrast to the beneficial effects of regular aerobic exercise that have been observed in the literature [20, 13, 28]. Given the well-known limitation of cross-sectional study designs and the conflicting results between aerobic and resistance training, interventional studies were needed to draw proper conclusions.

In the first intervention study to address this question, we found that several months of strenuous resistance training in young men induced a 20 % reduction in carotid arterial compliance [65]. Moreover, in order to isolate the effects of resistance training on arterial compliance as much as possible, a detraining program was implemented at the conclusion of the resistance training intervention. If the changes in arterial compliance were mediated by resistance training, such changes should return to the baseline level when the stimuli of daily resistance exercise were removed. Indeed during the detraining period, arterial compliance, which was reduced with resistance training, was reversed to the baseline values [65]. In support of these findings, the arterial stiffening effects of strenuous resistance training have since been observed in young women [66] and have been confirmed by a number of other studies [67–70].

Mechanisms Underlying the Strenuous Resistance Training-Induced Increases in Arterial Stiffness

Considering these findings, the question that emerges is, *What are the physiological mechanisms underlying the increase in arterial stiffness with strenuous resistance exercise training*? During resistance exercise bouts, arterial blood pressure increases to as high as 320/250 mmHg [71] and arterial walls are exposed to substantial amounts of distending pressures. There have been several case reports describing aortic dissection during heavy weight lifting exercises [72, 73]. It is possible that arterial stiffening may be caused by smooth muscle hypertrophy and synthesis of extracellular matrix stimulated by repeated elevations in local distending pressure [74] in order to strengthen the arterial wall against the risk of aortic rupture. Indeed, central arterial compliance was associated with arterial wall thickness in a group of resistance-trained adults [64]. Other potential mechanisms include the formation of collagen cross-linking and advanced glycation end products [75] and the increase in vasoconstrictor tone exerted by vasoactive molecules [8]. Although there are numerous vasoactive molecules that can affect

smooth muscle vasoconstrictor tone, endothelin-1 does not appear to play a role in arterial stiffening with resistance exercise training [76].

The aforementioned studies were conducted using strenuous weight training regimens in relatively young healthy subjects with high baseline arterial compliance. Whether or not moderate intensity strength training would further reduce the already low arterial compliance of middle-aged and older adults is a clinically important question. Older individuals are at greater risk for developing CVD as well as for experiencing functional disability associated with sarcopenia [59, 60], and resistance training is being strongly recommended as a preventive intervention for functional capacity with advancing age [54, 55, 57, 60, 77, 78]. As such, it is important to understand the interaction between age and resistance training for the key cardiovascular function of arterial compliance.

To do so, we recruited previously sedentary middle aged and older adults and prescribed a resistance exercise program that was consistent with the recommended guidelines established by the American Heart Association (AHA) [54]. We found that there was no significant decrease in central arterial compliance with strength training in middle-aged and older adults with low baseline arterial compliance [79]. In another study involving healthy postmenopausal women, 18 weeks of a moderate resistance training program did not change AI [80]. Moreover, 12 weeks of leg resistance training did not change aortic PWV in older men though maximal muscular power was increased by 16 % [76]. Collectively, these results suggest that older adults can gain the benefits of moderate resistance training without experiencing arterial stiffening.

Concurrent Training or Cross-Training

Arguably, one of the most effective way to maximize benefits from both aerobic exercise and resistance exercise appears to be the simultaneous performance of both training (i.e., concurrent training or cross-training) [81, 82]. Theoretically, the opposing effects of aerobic and resistance training exercise on central arterial compliance should negate the adverse effects of resistance training on arterial compliance if aerobic exercise training effects equals or exceeds the resistance training effects. This hybrid approach is consistent with the latest exercise recommendation that more inclusive practices of aerobic, resistance, and flexibility exercise training should be recommended as an approach to enhance both overall fitness and health [58].

As an initial approach to address this, we performed a cross-sectional study involving rowers. Rowing is unique because its training encompasses both endurance and strength training components. Rowers require large muscle strength for the acceleration of the boat at the race start and a high endurance capacity to maintain this speed during the race [83]. Likewise, rowers perform a combination of endurance and strength training during their usual training regimen as demonstrated by their large maximal aerobic capacity and muscle strength [83–85]. In order to minimize the weaknesses of the cross-sectional study design and to isolate the influence of rowing as much as possible, rowing and sedentary control groups were

carefully matched for age, body composition, blood lipids, plasma glucose, blood pressure, and dietary sodium intake [86]. Additionally, to isolate the effect of rowing, we excluded individuals for whom rowing was not their primary form of exercise. We demonstrated that central arterial compliance was higher and ß-stiffness index was lower in habitual rowers than in age-matched sedentary controls [86].

The results of subsequent interventional studies are consistent with this cross-sectional study. Concurrently-performed endurance training minimized arterial stiffening that was accompanied by high-intensity resistance training [67]. Additionally, there was a tendency for arterial compliance to increase with combined endurance and resistance training. Other groups have since confirmed these findings [87]. In a study involving healthy postmenopausal women, 3 months of combined circuit weight training and endurance training reduced PWV [87]. From the standpoint of exercise adherence and compliance, this type of concurrent training is highly beneficial as it is more enjoyable and breaks the boredom that often results from long-term participation in a single exercise mode [81, 82]. Thus, stiffening of the large arteries may be avoided if endurance training is incorporated into an exercise program that has a strenuous strength training component. For related discussions on the benefits of concurrent training, please see Chaps. 3, 4, 6, and 13.

Clinical Implications and Importance

There are a number of ways that arterial stiffening can contribute to the increased incidence of CVD (Fig. 8.2). Hypertension is one of the most prevalent risk factors for CVD, and the majority of patients with hypertension are classified as having

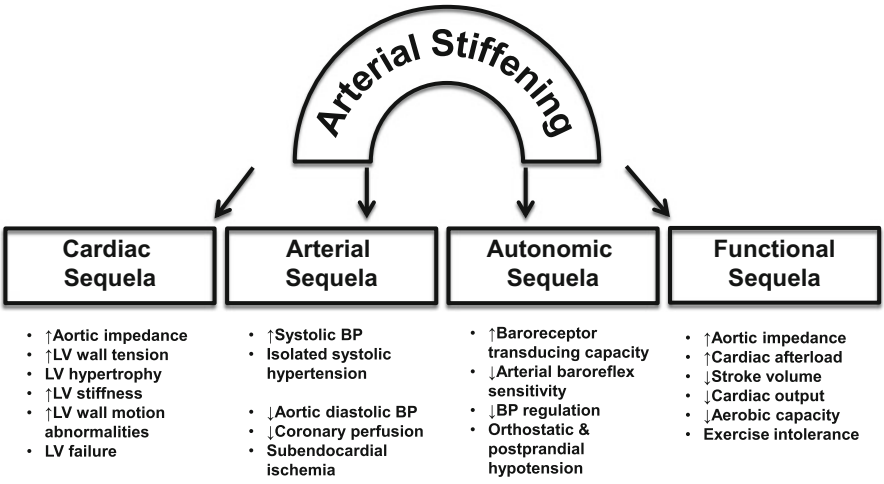

Fig. 8.2 Clinical and functional sequelae of arterial stiffening. *LV* left ventricle, *BP* blood pressure

essential hypertension without known causes of elevated high blood pressure. Most of these patients develop this health condition as they age as blood pressure, more specifically systolic blood pressure, increases progressively with advancing age [88]. Arterial stiffness is now thought to be a primary factor mediating the age-related increases in blood pressure [5]. By absorbing a proportion of the energy in systole and releasing it in diastole, the aorta and large arteries maintain coronary blood flow and avoid an increase in left ventricular afterload. Stiffening of central elastic arteries would reduce the buffering or cushioning effects translating the pulsatile effects of the arteries into arterioles and capillaries where there is very limited ability to cope with the pulsatile stress. Through the impairment of this buffering function, reductions in arterial compliance or increases in arterial stiffness contribute to elevations in systolic blood pressure, left ventricular hypertrophy, and coronary ischemia [4–6, 89]. Indeed, higher arterial stiffness is associated with a greater rate of mortality in patients with end-stage renal failure and essential hypertension [2, 3].

From a functional standpoint, arterial stiffness is significantly and inversely associated with maximal oxygen consumption, one of the most important determinants of exercise capacity as well as a CVD risk factor [12]. Associations between PWV and physical working capacity have also been reported [90]. Stiffening of central elastic arteries could increase aortic impedance and left ventricular afterload, thereby reducing stroke volume and systemic cardiac output, a critical determinant of maximal oxygen consumption [30, 91]. Indeed, the administration of calcium channel blockers that act to reduce arterial stiffness results in an improvement in aerobic exercise performance among older individuals [92]. Through this systemic hemodynamic mechanism, arterial stiffening can contribute to the exercise intolerance typically observed in older populations [93].

Conclusion

Regular aerobic exercise can reduce arterial stiffness in healthy middle aged and older adults and attenuate age-related increases in arterial stiffness. Importantly, this can be accomplished with an intensity (moderate) and type (e.g., walking and swimming) of physical activity that can be performed by most, if not all, adults. The beneficial effects of regular aerobic exercise on arterial stiffness are associated with a favorable influence on arterial blood pressure and arterial baroreflex sensitivity. However, regular aerobic exercise may not be effective in reducing arterial stiffness in patients with existing clinical conditions. In contrast to the effects of aerobic exercise, an intervention incorporating strenuous resistance training increases, rather than decreases, arterial stiffness in young adults. However, the arterial stiffening effect appears to be absent when older adults with already increased arterial stiffness perform moderate intensity resistance exercise programs. Simultaneously performed endurance and resistance training or concurrent training can elicit beneficial adaptations without inducing arterial stiffening effects. Thus, the effects of exercise training on the elastic properties of arteries depend on exercise modes and populations.

Key Points and Resources

- Regularly-performed aerobic exercise is effective in preventing and reversing arterial stiffening that occur with advancing age.
- Arterial stiffness increases after strenuous resistance training in young men but not in older adults with already increased levels of arterial stiffness.
- Concurrently-performed aerobic exercise effectively prevents the arterial stiffening effects of strenuous resistance training.
- Association for Research into Arterial Structure and Physiology http://www.arterysociety.org/
- North America Artery Society http://naartery.org/
- Seals DR, Desouza CA, Donato AJ, Tanaka H (2008) Habitual exercise and arterial aging. J Appl Physiol 105: 1323–1332 [20].

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