Exercise and Blood Pressure Control in Hypertension

8

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Abbreviations

1RM	One-repetition maximum
ACC	American College of Cardiology
ACSM	American College of Sports Medicine
AHA	American Heart Association
BP	Blood pressure
CO	Cardiac output
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
Ex R_x	Exercise prescription
FIT	Frequency, intensity, and time
FITT-VP	Frequency, intensity, time, type, volume, and progression
HIIT	High-intensity interval training
HR	Heart rate
IHG	Isometric handgrip
JNC 7	The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
JNC 8	The Eighth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
MAP	Mean arterial pressure
PEH	Postexercise hypotension
RPE	Rating of perceived exertion
RT	Resistance training
SBP	Systolic blood pressure
TPR	Total peripheral resistance
USA	United States
$\dot{V}O_2$ max	Maximal oxygen consumption
$\dot{V}O_2$ peak	Peak oxygen consumption
$\dot{V}O_2R$	Oxygen consumption reserve

Introduction

Hypertension Is a Major Public Health Problem

Hypertension is the most common, costly, and modifiable cardiovascular disease (CVD) risk factor in the United States (USA) and world [1–3]. Among 195 countries and territories, high systolic blood pressure (SBP) is the *leading* global risk factor and has accounted for 10.5 million deaths and 212.1 million years of life lost over the past two decades [3]. In the United States, the number of hypertension-related deaths increased 62% from the year 2000 to 2013 (i.e., from 245,220 to 396,675 deaths) [4]. The estimated direct and indirect cost of hypertension is

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$51.2 billion, and this figure is projected to increase to $274 billion by the year 2030 [1]. For these reasons, the Department of Health and Human Services established hypertension as a high-priority, leading health indicator within the Healthy People 2020 national objectives by aiming to (a) increase the proportion of adults with hypertension whose blood pressure (BP) is under control by 18% and (b) reduce the proportion of adults diagnosed with hypertension by 10% by the year 2020 [5]. Similarly, the World Health Organization has established a series of key targets to reduce the risk of premature death from CVD and stroke by 25% by the year 2025 ("25 by 25") [6]. One of the key targets, and arguably the most impactful, is reducing the global prevalence of SBP >140 mmHg by 25%.

Early diagnosis, including accurate and repetitive BP measurements using standard professional methodology and procedures, and effective antihypertensive therapeutic interventions are essential for meeting the Healthy People 2020 national objectives. Untreated and poorly controlled hypertension contributes to and accelerates pathological processes that lead to increased risk of CVD, shorter life expectancy free of CVD, more years lived with CVD, and increased of risk of mortality [2, 7–9]. Adoption of a healthy lifestyle is fundamental for the primary prevention, treatment, and control of hypertension. Given that the relationship between BP and CVD risk is linear, continuous, and consistent, early and aggressive lifestyle intervention is critical in order to prevent or delay the rapid, progressive rise in BP [2, 10, 11].

Definition and Key Concepts

Hypertension is a health condition defined by a transitory or sustained elevation of systemic arterial BP to a level likely to induce cardiovascular damage or result in other adverse health consequences [12]. Since the 2003 publication of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP (JNC 7) [13], several large-scale epidemiological studies and meta-analyses [9, 11, 14–18] have clearly and consistently shown increased cardiovascular risk at BP levels far below those used to define hypertension traditionally (i.e., resting SBP ≥140 mmHg or diastolic BP [DBP] ≥90 mmHg [13]). Starting at 115/75 mmHg, cardiovascular risk doubles for every 20 mmHg increase in SBP or 10 mmHg in DBP [9, 11]. Therefore, the American College of Cardiology (ACC) and the American Heart Association (AHA), with support from other professional societies, published the 2017 Guideline for the Prevention, Detection, Evaluation, and Management of High BP in Adults [10], a long-awaited update to the JNC 7 [13].

The most noteworthy change in the 2017 ACC/AHA guideline is the recommendation to use *lower* SBP and DBP thresholds to define hypertension among the general population (Table 8.1). These guidelines also recommend

Table 8.1 Blood pressure thresholds and classification scheme according to the 2017 American College of Cardiology/American Heart Association Guideline [10]

BP threshold (mmHg)			2017 ACC/AHA
Systolic BP		Diastolic BP	Classification scheme[a, b]
<120	and	<80	Normal BP (*normal BP*)
120–129	and	<80	Elevated BP (*prehypertension*)
130–139	or	80–89	Stage 1 hypertension (*prehypertension*)
140–159	or	90–99	Stage 2 hypertension (*stage 1 hypertension*)
≥160	or	≥ 100	Stage 2 hypertension (*stage 2 hypertension*)

BP blood pressure, *ACC/AHA* American College of Cardiology/American Heart Association
[a]Individuals with differing systolic BP and diastolic BP classifications should be designated to the higher BP category
[b]The BP classification scheme corresponding to the Seventh Report of the Joint National Committee (JNC 7) on Prevention, Detection, Evaluation, and Treatment of High BP [13] is provided in parentheses

incorporating the estimated 10-year risk of atherosclerotic CVD (determined by the ACC/AHA Pooled Cohort Equations [19]) *in addition* to resting BP to guide antihypertensive medication treatment decisions. Accordingly, hypertension is now defined as a resting SBP of 130 mmHg or greater, a resting DBP of 80 mmHg or greater, taking antihypertensive medication, being told by a physician or health professional on at least two occasions that one has high BP, or any combination of these criteria [10]. Using this definition, ≈103 million Americans (46%) [10, 20] and approximately 1.4 billion adults (31%) worldwide [21] have hypertension. Of note, this global estimate is based on the previous definition of hypertension (i.e., SBP or DBP of ≥140 mmHg or ≥90 mmHg, respectively [13]) and, thus, underestimates the global burden of hypertension as defined in the 2017 ACC/AHA guideline.

In addition to redefining hypertension, the 2017 ACC/AHA guideline eliminated the term "prehypertension" (SBP ranging from 120 to 139 mmHg and/or DBP ranging from 80 to 89 mmHg) introduced in the JNC 7 [13]. Instead, they include a BP category termed "elevated BP," defined as resting SBP ranging from 120 to 129 mmHg *and* DBP <80 mmHg [10]. There is substantial evidence from individual epidemiological studies and meta-analyses of these data that show a graded and progressive rise in cardiovascular risk as BP levels increase from normal BP, to elevated BP, to stage 1 hypertension [10]. Hence, elevated BP, like prehypertension, represents an opportunity for increased awareness and intervention so that individuals with this condition can delay or prevent incident hypertension through the adoption of healthy lifestyle interventions, such as participation in regular exercise [10, 11, 22–25].

In approximately 90% of cases, the etiology of hypertension is unknown, and it is called essential, idiopathic, or primary hypertension. Systemic hypertension with a known cause is referred to as secondary or inessential hypertension. Systemic hypertension primarily involves disorders and diseases of the renal, endocrine, or nervous systems, such as kidney disease, Cushing's syndrome, and Guillain-Barre Syndrome, respectively. Other causes of secondary hypertension are obstructive sleep apnea, tumors, or drug-induced.

Although essential hypertension and secondary hypertension are the major classifications of hypertension, several other descriptive terms are used to define various types of hypertension [10]:

- Isolated systolic hypertension is defined as SBP of 130 mmHg or greater and DBP of less than 90 mmHg.
- White coat hypertension is characterized by elevated BP readings when measured in the physician's office (or other clinical setting) but normal BP when measured outside of the physician's office using ambulatory or home BP monitoring.
- Masked hypertension is characterized by normal BP readings when measured in the physician's office (or other clinical setting) but elevated BP when measured outside of the physician's office using ambulatory or home BP monitoring. There are three subtypes of masked hypertension: morning, daytime (stress-induced), and nocturnal hypertension [26].
- Pulmonary hypertension is characterized by elevated pulmonary arterial pressure accompanied by dyspnea, fatigue, syncope, and/or substernal chest pain.
- Resistant hypertension is defined as the failure to achieve goal BP in patients who are adhering to maximum doses of an appropriate three-drug regimen that includes a diuretic. Resistant hypertension may be caused by improper BP assessments, volume overload (i.e., fluid retention, excess sodium intake), drug-induced (i.e., side effect of a prescription medication), lifestyle habits (e.g., diet, excessive alcohol intake), or other identifiable causes of hypertension (i.e., secondary hypertension) (for an expanded discussion on resistant hypertension and cardiorespiratory fitness, see Chap. 7).

- Malignant hypertension is defined by markedly elevated BP levels (i.e., SBP >200 mmHg and/or DBP >140 mmHg) due to papilledema, a condition of optic nerve swelling that is secondary to elevated intracranial pressure [27].

Pathophysiology

BP is a highly heterogeneous, quantitative trait that serves as a biomarker of hypertension. Many physiologic factors have been implicated in the pathogenesis and maintenance of elevated BP that include (but are not limited to) activation of the sympathetic nervous and renin-angiotensin-aldosterone systems, endothelial dysfunction, and vascular structural changes [28–31]. The regulation of BP is a sophisticated and multifaceted process as it is modulated by intermediary phenotypes associated with renal, hormonal, vascular, peripheral, and central adrenergic pathways. BP regulation is further complicated by the fact that these intermediary phenotypes are controlled by complex, sometimes redundant, interwoven mechanisms, including BP itself [32, 33]. According to Ohm's law, mean arterial pressure (MAP) is the product of cardiac output (CO) and total peripheral resistance (TPR): MAP = CO × TPR. Therefore, the pathogenic mechanisms leading to hypertension must increase TPR, CO, or both [34]. Once hypertension has been established, it is commonly characterized by elevated TPR and a lower or normal CO [34].

Essential hypertension, as detected by resting (brachial) BP levels, tends to develop gradually, emphasizing the importance of prevention and early diagnosis. Essential hypertension tends to cluster in families and represents a collection of genetically based diseases and syndromes with several underlying inherited biochemical abnormalities. Factors considered important in the genesis of essential hypertension include family history, genetic predispositions, salt sensitivity, imbalances in the major BP regulatory systems that favor vasoconstriction over vasodilation, and their interaction with environmental factors. Several lifestyle-related factors have been commonly implicated in the development of hypertension and include obesity, physical inactivity, and excessive salt intake. The relationship among these modifiable factors is complex, with several overlapping mechanisms of action. Indeed, even modest alterations in these lifestyle-related factors can elicit reductions in SBP ranging from 2 to 20 mmHg [10].

Early manifestations of essential hypertension such as autonomic dysfunction (diminished parasympathetic and increased sympathetic tone) are present far before noticeable elevations in BP occur [31] (see Davis et al. [35] and Grassi et al. [36] for detailed reviews on autonomic and hemodynamic disturbances that occur with hypertension). In the presence of other CVD risk factors (e.g., obesity and dyslipidemia), early markers of dysfunction are often exacerbated, and the pathophysiologic progression toward fully developed hypertension is accelerated [28, 37]. Thus, elevations in CO, normal TPR, and enhanced endothelium-dependent dilation characterize the early (developmental) stages of essential hypertension. As elevations in BP progress toward essential hypertension, it becomes better characterized by normal CO, elevated TPR, endothelial dysfunction, and left ventricular hypertrophy – alterations that reflect the adverse hemodynamic and vascular changes that arise from chronically elevated peripheral resistance [34].

Chronic elevations in peripheral resistance may be related to neurohumoral mechanisms (e.g., increases in circulating levels of epinephrine), myogenic autoregulation, structural changes in small arteries, or a combination of these factors [31]. Poiseuille's law states that resistance is *inversely* related to the vessel radius of the fourth power [34]; thus, functional or structural changes in arterial lumen size or radius are major determinants of TPR. Decreases in lumen size or radius will increase TPR, which in turn aids in the maintenance of persistent elevations in BP and, subsequently, hypertension (for detailed reviews see Safar et al. [38] and Laurent et al. [39]). Sustained elevations in pressure exert greater vessel wall stress that, overtime, induce alterations in the vessel properties and wall composition that favor mechanobiological responses

that attempt to reduce wall stress (i.e., vascular thickening and structural stiffening). However, these compensatory mechanisms eventually fail, and endothelial dysfunction ensues. Of note, changes in vascular structure and function have traditionally been viewed as either the *cause* or the *consequence* of elevated BP. More recent perspectives suggest that, based on the totality of available evidence, vascular stiffness is *both* a cause and a consequence of hypertension [40].

Hypertension, if untreated, contributes to and accelerates the pathological processes that lead to premature death from heart disease, stroke, and renal failure [41]. Hypertension damages the endothelium, which predisposes the individual to atherosclerosis and other vascular pathologies. Hypertension-induced vascular damage can lead to stroke and transient ischemic attacks as well as end-stage renal disease. Chronic elevations in BP increase afterload on the heart and thus the mechanical stress or workload of the heart. In response to these stressors (and other neurohormonal factors), the left ventricle may undergo distinct structural changes or *maladaptation* that include increased left ventricular wall thickness and wall mass and decreased chamber size. Collectively, these alterations characterize a condition known as concentric left ventricular hypertrophy [42–45]. Concentric left ventricular hypertrophy is an antecedent to chronic heart failure and is an independent risk factor for other types of CVD, such as coronary heart disease and stroke [42, 46, 47].

Epidemiological Evidence of the Scope of the Hypertension (Prevalence and Incidence)

Several definitions of hypertension exist in the literature, resulting in slight variations in hypertension prevalence and control rates [48, 49]. The importance of utilizing a clear and consistent definition of hypertension is essential to guiding diagnosis, treatment, control, and surveillance of hypertension. As such, the definition of hypertension and adult BP categories published in the 2017 ACC/AHA guideline will be the BP classification scheme incorporated in this chapter (Table 8.1) [10].

Hypertension affects ≈103 million (45.6%) adults living in the United States [10, 20], an additional 31 million newly diagnosed Americans when compared to prior estimates using the JNC 7 definition of hypertension (i.e., ≈72 million or 31.9% of US adults) [13, 20]. Despite a higher prevalence of hypertension in the United States under the new guideline, the majority of newly diagnosed adults can be treated with lifestyle modifications (e.g., diet and exercise) without concomitant pharmacological intervention. Like JNC 7 [13], the 2017 ACC/AHA guideline [10] recommends that pharmacological treatment be initiated among the general population when resting SBP or DBP levels exceed 140 mmHg or 90 mmHg, respectively, or among those with stage 1 hypertension (Table 8.1) with high CVD risk (i.e., patients with known CVD, diabetes mellitus, or chronic kidney disease, or estimated CVD risk >10%). Accordingly, there is a small increase (1.9%) in the proportion of US adults who are recommended pharmacological therapy under the 2017 ACC/AHA guideline compared to the JNC 7 (36.2% vs. 34.3%) [20].

Another ≈27 million US adults (≈12%) have elevated BP [20]. This means more than half (≈60%) of the adult population in the United States – approximately ≈130 million Americans – have high BP (i.e., elevated BP to established stage 1 or 2 hypertension). Individuals with elevated BP are at increased risk for developing hypertension, additional CVD risk factors, heart disease, and stroke [10, 37, 50]. The rate of progression from elevated BP to hypertension can occur rapidly (within a 5-year period) [1, 9, 51, 52] and is positively and strongly associated with age, baseline BP, and comorbidities [53, 54].

The prevalence of hypertension increases substantially with age, with 77% and 82% of US adults ≥65 and ≥75 yr of age, respectively, having hypertension compared to ≈36% of adults <55 yr of age [10, 55]. Among adults ≥50 yr of age, the lifetime risk of developing hypertension approaches 90% [1, 10, 53]. The prevalence of hypertension also varies by racial/ethnic populations and sexes living in the United States [1, 10]:

- Non-Hispanic whites 20 yr or older, 47% of men and 41% of women
- Non-Hispanic Asians 20 yr or older, 45% of men and 36% of women
- Non-Hispanic blacks 20 yr or older, 59% of men and 56% of women
- Hispanic Americans 20 yr or older, 44% of men and 42% of women

On average, blacks have the highest prevalence of hypertension of all other races [1, 10]. For adults 45 years of age without hypertension, the 40-year risk of developing hypertension was 93% for blacks, 92% for Hispanic, 86% for white, and 84% for Chinese adults [10]. Compared to whites, blacks develop hypertension earlier in life, their BP is higher over their lifetime, and among those with hypertension, blacks are more likely to have resistant hypertension than whites and Hispanics (19.0% vs. 13.5% and 11.2%, respectively) [1, 56]. Despite similar awareness and treatment rates as whites, blacks experience more severe hypertension and greater difficulty in achieving BP control due to reduced effectiveness of some antihypertensive medications, resulting in 1.5 times greater risk of heart failure, 1.8 times greater risk of fatal strokes, and 4.2 times greater risk of end-stage renal disease [1].

Hypertension is one of the most common primary diagnoses in the United States (≈34% of all office-based physician visits by adults in 2013 were related to hypertension care [57, 58]) and is the leading cause for medication prescriptions among adults >50 yr [59, 60]. Overall, population estimates of awareness, treatment, and control of hypertension in the United States have increased overtime, but the rate of improvement has plateaued in recent years [55, 61]. In contrast, the prevalence of hypertension has remained unchanged for more than a decade [61]. The most recent estimates show that among adults ≥20 yr of age with hypertension, 84.1% were aware of their condition, 76.0% were receiving treatment for their high BP, and 52% were properly controlled (BP control was higher, 69%, among those currently being treated) [1], which falls short of the Healthy People 2020 target of controlling 61.2% of all adults with hypertension [61]. A recent paper by Quindry and Franklin [62] nicely summarizes the independent and interrelated cardioprotective effects of exercise and pharmacotherapies prescribed to treat CVDs and related risk factors. The authors put forth the notion that in order to effectively reduce the prevalence and burden of CVD, contemporary therapies must include *both* adjunctive exercise and lifestyle interventions in addition to pharmacological agents. Indeed, if the prevalence and control of hypertension were improved by increasing the use of lifestyle-based antihypertensive therapies, such as exercise, as a primary or complementary treatment strategy, our society would experience substantial health and economic benefits.

Epidemiologic Association Between Cardiorespiratory Fitness and Hypertension

The preventive benefits of physical activity on all-cause mortality and the onset of CVD risk factors have been documented in many large-scale prospective studies and are summarized in several recent reviews [63–72]. These data clearly and consistently demonstrate that physical activity and cardiorespiratory fitness are inversely and independently associated with cardiovascular morbidity and mortality among both healthy and clinical populations (e.g., adults with CVD), independent of sex/gender and race/ethnicity. Accumulating evidence from prospective cohort studies show that, on average, every one metabolic equivalent increase in cardiorespiratory fitness is associated with a 10–25% reduction in mortality risk [66, 71–73], with the least fit individuals (i.e., <5 metabolic equivalents) experiencing the greatest reduction in risk.

The cardioprotective benefits of physical activity are expansive, but for the purposes of this chapter, we will focus our discussion on the relation between cardiorespiratory fitness and hypertension. Several recent studies have added to a growing body of evidence that shows baseline cardiorespiratory fitness is an important predictor of incident hypertension, such that higher levels of baseline physical activity and cardiorespira-

tory fitness are associated with a lower risk of incident hypertension among men and women [63, 72, 74–77]. More recent investigations have added to our understanding of this relationship by examining longitudinal patterns in cardiorespiratory fitness and incident hypertension [78–80]. Collectively, these studies support that improvements in cardiorespiratory fitness throughout one's lifetime are associated with the lowest risk of incident hypertension. Similarly, longitudinal patterns that result in higher levels of cardiorespiratory fitness being maintained later in life are also associated with lower risk of incident hypertension [79].

In addition to the cardioprotective effects of physical fitness, several studies also support that higher levels of muscular fitness (i.e., muscular strength and endurance) confer similar protective effects both *independently* and *jointly* with high levels of cardiorespiratory fitness. Moreover, higher levels of muscular fitness are associated with lower risk of developing CVD risk factors, including hypertension [81–83], CVDs, and all-cause mortality [84–89]. Among those with hypertension, higher levels of muscle strength were inversely associated with mortality, independent of cardiorespiratory fitness [85, 90]. Reductions in mortality risk were even greater among individuals with high levels of muscular strength *and* cardiorespiratory fitness. These findings underscore the importance of including both aerobic and resistance exercises in the Ex R_x for individual adults with hypertension [23, 24, 91].

To summarize, there is overwhelming evidence to support that cardiorespiratory fitness may be the single best predictor of cardiovascular morbidity and mortality among the general population and those with established hypertension. In fact, a recent Scientific Statement published by the AHA advocated that cardiorespiratory fitness be considered a clinical vital sign and that it be assessed as part of routine practice among healthy and clinical populations [72]. Increases in cardiorespiratory fitness and the maintenance of such improvements favorably modulate the age-related progressive increase in arterial stiffness [92], BP, and, ultimately, the development of hypertension [63, 66, 78, 79]. Among those with hypertension, higher levels of cardiorespiratory fitness are associated with reduced BP and left ventricular hypertrophy regression, which in turn may slow the progression of hypertension to more severe forms [63, 64]. In addition, when the types and quantity of physical activities and/or structured exercises required to improve cardiorespiratory and muscular fitness levels are executed appropriately, additional coexisting CVD risk factors (e.g., obesity, dyslipidemia) may experience favorable effects, resulting in even greater health benefits [63–65, 67, 68, 70, 72, 81, 83, 90, 93, 94].

The Antihypertensive Effects of Exercise

Participation in regular exercise is a key modifiable determinant of hypertension and is recognized as a cornerstone therapy for the primary prevention, treatment, and control of high BP [10, 11, 22–25]. Meta-analyses of randomized controlled, intervention trials have concluded that regular, aerobic exercise lowers resting BP 5–7 mmHg, while dynamic resistance exercise lowers resting BP 2–3 mmHg among individuals with hypertension [91, 95]. The magnitude of these BP reductions following aerobic and resistance exercise can lower CVD risk by 20–30% and 6–14%, respectively [9, 10, 13, 96]. Furthermore, aerobic exercise can lower BP by a magnitude that rivals those obtained with first-line antihypertensive medications [13, 96–98]. Exercising as little as 1 day per week is as effective (or even more so) than pharmacotherapy for reducing all-cause mortality among those with hypertension [99]. In addition, when lifestyle modifications are executed appropriately, coexisting CVD risk factors may also experience benefits, translating to an even greater reduction in overall cardiovascular risk. Indeed, a recent meta-analysis of major exercise and drug trials showed no statistically detectable difference between exercise and drug interventions for the secondary prevention of coronary heart disease and prediabetes, and physical activity interventions were more effective than drug interventions for the secondary prevention of stroke mortality [100].

For these reasons the 2017 ACC/AHA guideline [10], JNC 7 [13], 2014 Evidence-Based Guidelines for the Management of High Blood Pressure in Adults (JNC 8) [101], AHA/ACC 2013 Lifestyle Work Group [25], European Society of Hypertension and European Society of Cardiology [102], Canadian Hypertension Education Program [103], and the ACSM [23] universally endorse aerobic exercise for the primary prevention and treatment of hypertension. Although the recommended exercise prescription (Ex R_x) for individuals with hypertension in terms of the *f*requency (how often?), *i*ntensity (how hard?), *t*ime (how long?), *t*ype (what kind?), *v*olume (how much?), and *p*rogression (or FITT-VP) principle varies slightly across the various aforementioned organizational guidelines (Table 8.2), the general consensus is that adults with elevated BP to established hypertension should participate in 30–60 min/d of moderate-intensity aerobic exercise on most, if not all, days of the week to total 150 min/wk of exercise (or more) supplemented by moderate-intensity dynamic resistance training (RT) on 2–3 d/wk [91].

It should be noted that these recommendations are limited by methodological quality of the studies upon which the evidence is based [91, 104]. Major limitations in the current state of the literature include small sample sizes, assessing study populations with normal and elevated BP rather than hypertension, not accounting for major confounders to the BP response to exercise that include timing of the last bout of exercise and detraining effects, and lack of standard protocols for the assessment of BP and the exercise intervention. As a result of these limitations, the effectiveness of exercise as antihypertensive lifestyle therapy among individuals with hypertension has been underestimated [91, 95, 105]. Furthermore, large randomized clinical trials that examine both the acute and chronic BP lowering effects of exercise among diverse populations are needed before professional organizations can definitively determine the optimal Ex R_x for individuals with hypertension. New and emerging research are highlighted later in this chapter, as such studies may serve to fine-tune exercise prescription among individuals with hypertension.

Presently, the ACSM recommends the following *FITT-VP* Ex R_x for individuals with hypertension (Table 8.3) [24]:

- *F*requency: For aerobic exercise, on most, preferably all days of the week supplemented by resistance exercise 2–3 d/wk and flexibility exercise 2–3 d/wk.
- *I*ntensity: Moderate (i.e., 40–<60% oxygen consumption reserve [$\dot{V}O_2R$] or 11–14 on a scale of 6 [no exertion] to 20 [maximal exertion] level of physical exertion [106] or an intensity that causes noticeable increases in heart rate [HR] and breathing) for aerobic exercise, moderate-to-vigorous (60–80% of one-repetition maximum [1RM]) for resistance, and stretch to the point of feeling tightness or slight discomfort for flexibility.
- *T*ime: For aerobic exercise, a minimum of 30 min or up to 60 min/d for continuous or accumulated aerobic exercise. If intermittent, begin with a minimum of 10 min bouts.
- *T*ype: For aerobic exercise, emphasis should be placed on prolonged, rhythmic activities using large muscle groups such as walking, cycling, or swimming. Dynamic resistance exercise may supplement aerobic exercise and should consist of at least one set of 8–12 repetitions of 8–10 different exercises targeting the major muscle groups. For flexibility, hold each muscle 10–30 s for 2–4 repetitions per muscle group. Balance (neuromotor) exercise training 2–3 d/wk is also recommended as adjuvant exercise in individuals at high risk for fall (i.e., older adults) and is likely to benefit younger adults as well.
- *V*olume: To total at least 150 min/wk or 700–2000 kcal/wk of moderate-intensity aerobic exercise.
- *P*rogression: Progress gradually, avoiding large increases in any of the components of the FITT. Increase exercise duration over the first 4–6 wk and then increase frequency, intensity, and time (or some combination of these) to achieve the recommended volume of 700–2000 kcal/wk over the next 4–8 months. Progression may be individualized based on tolerance and preference in a conservative manner.

Table 8.2 The existing professional exercise recommendations among adults with hypertension

	Professional committee/organization						
FITT of the Ex R_x	JNC 8 [101] and AHA/ACC Lifestyle Work Group [25]	JNC 7 [13]	AHA Scientific Statement [161]	ACSM Position Stand [23]	ESH/ESC [102]	2016 CHEP [103]	2017 ACC/AHA Guideline [10]
*F*requency (how often?)	3–4 d/wk ≥12 wk	5–7 d/wk (most days)	5–7 d/wk (most days)	5–7 d/wk	5–7 d/wk	4–7 d/wk and habitual activity	5–7 d/wk (most days)
*I*ntensity (how hard?)	Moderate to vigorous[a]		Moderate to vigorous[a]	Moderate[a]	Moderate[a]	Moderate[a]	Moderate to vigorous[a]
*T*ime (how long?)	40 min/d	≥30 min/d	150 min/wk	30–60 min/d (single or multiple ≥ 10 min bouts)	≥30 min/d	30–60 min/d (accumulation)	90–150 min/wk
*T*ype (what kind?) *Primary*	Aerobic exercise	Aerobic exercise	Aerobic exercise	Aerobic exercise	Aerobic exercise	Aerobic exercise	Aerobic exercise
Evidence rating	High[b]/Grade B[b], COR IIa, LOE A[c]		COR I, LOE A[c]	LOE A[d] LOE B[d]	COR I, LOE A–B	LOE Grade D[e]	COR I, LOE A[c]
Adjuvant			RT	RT[a] 2–3 d•wk^{-1}	RT[a] 2–3 d/wk	RT, Isometric (Handgrip) RT	RT, Isometric RT 3 d/wk
Evidence rating			COR IIa, LOE B[c]	LOE B[f, d]		LOE Grade D[e]	COR I, LOE A[c]

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Shaded boxes indicate information was missing or not reported in original report. *ACC* American College of Cardiology, *ACSM* American College of Sports Medicine, *AE* aerobic exercise, *AHA* American Heart Association, *CHEP* Canadian Hypertension Education Program, *COR* class of recommendation, indicates the *strength* of the recommendation, *ESH* European Society of Hypertension, *ESC* European Society of Cardiology, *Ex* R_x exercise prescription, *FITT f*requency, *i*ntensity, *t*ime, and *t*ype, *LOE* level of evidence, rates the *quality* of scientific evidence, *JNC* 7 Seventh Report of the Joint National Committee, *JNC* 8 Eighth Report of the Joint National Committee, *RT* dynamic resistance training (unless specified otherwise), $\dot{V}O_2R$ oxygen consumption reserve

[a]Moderate intensity, 40–59% $\dot{V}O_2R$ or an intensity that causes noticeable increases in heart rate and breathing; 60–80% of 1RM for RT; vigorous (or high) intensity, ≥60% $\dot{V}O_2R$ or an intensity that causes substantial increases in heart rate and breathing

[b]Evidence statement (LOE), *AE lowers blood pressure (BP)*, high; evidence recommendation (COR), *FIT* (*f*requency, *i*ntensity, *t*ime) *of the Ex* R_x *to lower BP*, Grade B or moderate (COR IIa, LOE A)[c]

[c]AHA criteria to evaluate the COR and LOE [10, 161]

[d]Strength of evidence (COR): *immediate effects of AE* (i.e., *postexercise hypotension)*, Category B; *long-term effects of AE* (i.e., *chronic effects)*, Category A; *FIT of the Ex* R_x *to lower BP*, Category B

[e]Strength of evidence (LOE) ranges from Grade A to D *(strongest to weakest evidence)* [103]; *AE recommendations for individuals with normal BP to prevent hypertension or for individuals with hypertension to reduce BP*, Grade D; *for individuals with normal BP or stage 1 hypertension, resistance exercise* (i.e., *RT, isometric handgrip RT) does not adversely influence BP*, Grade D; *higher intensities of exercise are not more effective*, Grade D

[f]*Immediate effects of RT*, COR C[c]

Table 8.3 The current exercise prescription for adults with hypertension [23, 24]

FITT-VP principle of the Ex R_x	ACSM recommendations
*F*requency (how often?)	5–7 d/wk
*I*ntensity (how hard?)[a]	Moderate (40–<60% $\dot{V}O_2R$ or 12–13 on a scale of 6 [no exertion] to 20 [maximal exertion] level of physical exertion or an intensity that causes noticeable increases in heart rate and breathing)
*T*ime (how long?)	≥30–60 min/d; one continuous bout or multiple bouts of at least 10 min each
*T*ype (what kind?) *Primary*	Aerobic exercise; prolonged, rhythmic activities using large muscle groups (e.g., walking, cycling, swimming)
Adjuvant 1	Muscle strengthening *F:* 2–3 d/wk (non-consecutive) *I:* Moderate-to-vigorous-intensity (60–80% of 1RM); major muscle groups *T:* 8–10 exercises; 2–4 set of 8–12 repetitions
Adjuvant 2	Flexibility *F:* ≥2–3 d/wk *I:* Stretch to the point of feeling tightness or slight discomfort *T:* ≥10 min/d; ≥4 repetitions per muscle group; hold each static stretch for 10–30 s
Adjuvant 3[b]	Neuromotor *F:* ≥2–3 d/wk *I:* undetermined *T:* ≥20–30 min/d
*V*olume (how much?)[c]	≥150 min/wk or 700–2000 kcal/wk
*P*rogression	Progress gradually, avoiding large increases in any of the components of the Ex R_x; increase exercise duration over first 4–6 wk and then increase frequency, intensity, and time (or some combination of these) to achieve recommended quantity and quality of exercise over next 4–8 months

Note. ACSM American College of Sports Medicine, *FITT f*requency, *i*ntensity, *t*ime, *t*ype, *v*olume, and *p*rogression of exercise, *Ex* R_x exercise prescription; 1*RM* one-repetition maximum, $\dot{V}O_2R$ oxygen consumption reserve

[a]Vigorous-intensity aerobic exercise (i.e., ≥60% $\dot{V}O_2R$ or ≥14 on a scale of 6–20 [106]) may elicit greater and more extensive benefits and may be introduced after exercise preparticipation health screening and gradual progression

[b]Balance (neuromotor) training is recommended for older adults, individuals who are at substantial risk of falling, and is likely to benefit younger adults as well

[c]For greater and more extensive benefits, progress exercise volume to total 60 min/d and 300 min/wk of moderate intensity

The Relationship Between the Blood Pressure Effects of Acute (i.e., Postexercise Hypotension) and Chronic Exercise (i.e., Exercise Training)

Physiological responses to acute or short-term exercise translate into functional adaptations that occur during and persist for some time after an isolated exercise session, a phenomenon termed the last bout effect. It has been previously hypothesized that frequent repetition of these acute exercise sessions produces more permanent structural adaptations, forming the exercise training response. These persistent alterations in structure and function remain for as long as the training regimen is continued and then dissipate quickly, returning to pretraining values [107]. Several recently published studies support the notion that the reductions in BP experienced immediately following a single bout of exercise are similar in magnitude to those experienced after exercise training, an observation that suggests the BP benefits attributed to chronic exercise are largely the result of *postexercise hypotension* or PEH [22, 108–110]. PEH describes the immediate, short-term reductions in BP following acute exercise and persists for up to 24 h after the exercise bout [24].

The relationship between the BP response to acute and chronic exercise has yet to be fully elucidated; however, they appear to be related [22, 108–115]. Initial studies examining whether PEH

could be used to predict the BP response to aerobic exercise training reported moderate to large correlations between the acute and chronic SBP (r = 0.66–0.89) and DBP (r = 0.66–0.75) responses [110–112]. Since then, researchers have published similar observations for dynamic resistance (r = 0.47–0.74 for SBP, r = 0.45–0.80 for DBP) [110, 114, 115] and isometric resistance (SBP only: r = 0.0.58 for handgrip, r = ≈0.77 for leg) [109, 113] exercise ($P < 0.05$ for all). Collectively, these studies support the notion that the BP response to exercise training is largely a function of PEH.

However, it should be noted that 20–25% of individuals with hypertension do not experience reductions in BP to acute or chronic exercise [23]. Some individuals may even experience *increases* in BP as a result of exercise training [116], albeit this is far less common and not consistently reported [117]. Therefore, PEH has great potential to be used as a health screening tool to identify individuals with hypertension that will (likely) respond favorably to exercise as a nonpharmacologic, lifestyle-based therapy to control their high BP. Further research in a larger, more diverse sample of adults with hypertension is needed to substantiate this premise.

The next section will overview the effects of *acute* (i.e., immediate, short-term, or PEH) and *chronic* (i.e., long-term or training) aerobic, resistance (dynamic and isometric), and concurrent exercise on BP among individuals with hypertension. When appropriate, new and emerging research will be presented that has the potential to alter the way in which exercise may be prescribed to prevent, treat, and control hypertension.

Interventional Evidence of Aerobic (Endurance) Exercise and Blood Pressure Effects

Acute, Immediate, or Short-Term Effects, or Postexercise Hypotension

The BP reductions following acute exercise are immediate but short-term, persisting for up to 24 h after the exercise bout [24]. This response is termed postexercise hypotension or PEH and is an *expected* physiological response to exercise [22, 23, 91, 108, 118–120]. For this reason, individuals with hypertension are encouraged to exercise on most days of the week in order to benefit from the acute effects of aerobic exercise on BP [23, 24]. The BP response to acute aerobic exercise has been summarized in several reviews [108, 121–125], concluding that a single bout of aerobic exercise lowers resting SBP 6–11 mmHg and DBP 4–5 mmHg among adults with high BP and that the magnitude of PEH depends on several factors, including the characteristics of the sample and the intensity and duration of the aerobic exercise performed.

Despite the considerable range in the magnitude of PEH reported in these reviews, the general overall consensus supports that the magnitude of BP reductions resulting from acute aerobic exercise is most pronounced in individuals who stand to benefit the most (i.e., individuals with higher BP compared to normal BP) [23, 24, 124–127]. Short (10–15 min) and long (30–40 min) bouts of aerobic exercise performed continuously at a constant intensity (i.e., workload) or in intervals elicit PEH, independent of exercise intensity [91, 108]. However, there is accumulating evidence to suggest that BP benefits may be maximized when acute aerobic exercise is performed at more vigorous levels of physical exertion (i.e., the magnitude of the BP reductions is intensity-dependent) [91, 108]. New and emerging evidence regarding the relationship between aerobic exercise intensity and PEH will be discussed in the sections that follow.

A limited number of studies have directly compared aspects of the FIT of an acute aerobic exercise intervention on PEH among adults with hypertension [91, 104]. As a result, it remains unclear which factors, that is, aerobic exercise intensity, duration, the total work performed [128], or how the exercise is conducted (i.e., continuous, interval, intermittent or fractionized), are more influential in determining magnitude and duration of PEH. New and emerging evidence regarding the acute effects of aerobic exercise, and its application to the current Ex R_x for hypertension, will be discussed next.

Eicher and colleagues [129] published one of the first studies showing that the illustrated

magnitude of the BP reductions resulting from acute aerobic exercise occurs as a direct function of intensity such that the greater the intensity, the greater the BP reduction [91]. Briefly, Eicher et al. [129] examined the antihypertensive effects of three bouts of acute aerobic exercise performed at light (40% of VO_{2max}), moderate (60% of VO_{2max}), and vigorous (100% of VO_{2max}) intensity in men with elevated BP to stage 1 hypertension ($n = 45$). The authors found that BP decreased by 1.5/0.6 mmHg for every 10% increase in relative VO_{2max}, suggesting that more vigorous levels of physical exertion lowered BP to a greater extent than lower levels of physical exertion for individuals willing and able to tolerate more intense levels of exercise [129].

More recent investigations have focused on whether PEH is modulated by *how* the aerobic exercise is performed (i.e., single bout of continuous exercise vs. multiple bouts spread intermittently throughout the day). Bhammar et al. [130] compared the effects of fractionized aerobic exercise (3 × 10 min bouts) spread throughout the day (morning, midday, and afternoon) and a single bout of continuous exercise (1 × 30 min bout) performed at 60–65% of VO_{2peak} on ambulatory BP among 11 individuals with elevated BP. Bhammar et al. concluded that fractionized exercise was as effective as continuous exercise in eliciting PEH, reducing SBP 3–4 mmHg compared to control throughout the day until the following morning. Less is known about the antihypertensive effects of very short (<10 min) bouts of aerobic exercise. Miyashita et al. [131] compared the BP response to 30 min of running at 70% of VO_2max performed in either a single continuous bout (1 × 30 min) or multiple, very short bouts (10 × 3 min) among seven young men with elevated BP. Miyashita et al. reported reductions in SBP of 6 and 8 mmHg, respectively, that persisted for 24 h after the bout (Ps < 0.01). Taken together, these studies suggest that short (10 min) and very short (3 min) bouts of intermittent aerobic exercise interspersed throughout the day elicit PEH and that the antihypertensive effects of short and very short bouts of moderate-to-vigorous-intensity aerobic exercise are similar in magnitude and duration to those observed following a single bout of continuous aerobic exercise of the same intensity [130, 131].

In summary, a single, isolated session of aerobic exercise results in an immediate reduction in BP on the order of 5–7 mmHg among individuals with hypertension (i.e., PEH), with the greatest reductions occurring among those with the highest BP (i.e., upwards of 8–11 mmHg) [23, 91, 124–127]. PEH is a low-threshold phenomenon in terms of exercise duration as short (≈10 min) and now very short (<10 min) durations of exercise produce PEH [130, 132–134]. However, the minimum duration needed to produce the effect is dependent on the intensity of the exercise [131] and, at this time, remains to be determined. New and emerging research indicates that exercise intensity is an important determinant of PEH such that increasing levels of exertion lower BP in a dose-response pattern [91, 125, 129, 135] and that short bouts of exercise accumulated across a day can have the same beneficial impact on BP as one continuous bout of exercise [125, 130, 131].

Chronic, Training, or Long-Term Effects

Meta-analyses of studies investigating the antihypertensive effect of chronic aerobic exercise training among individuals with hypertension have concluded that dynamic aerobic or endurance exercise training reduces resting office and 24-h ambulatory BP 5–7 mmHg [136–139] and 3–4 mmHg [140, 141], respectively, among individuals with high BP. Of note, the participants in these meta-analyses were generally white and/or middle-aged, limiting the generalizability of the results to more diverse populations [95]. Nonetheless, one clear pattern that has emerged from these meta-analyses is that resting BP is lower due to aerobic exercise training and that the magnitude of the reduction is greatest for those with the highest BP.

Of the meta-analyses conducted to date, most have failed to provide insight into how population characteristics and/or the FIT of the exercise intervention moderate the BP effects of aerobic

(endurance) exercise training [95, 104, 108, 141], with the exception of two [98, 142]. Briefly, Whelton et al. [98] examined a large group (n = 2419) of racially/ethnically diverse patients (n = 1935 whites; n = 391 Asians; and n = 93 blacks) and reported BP reductions of 3/3 mmHg for whites, 6/7 mmHg for Asians, and 11/3 mmHg for blacks [98]. More recently, Cornelissen and Smart [142] examined the effect of aerobic exercise training lasting at least 4 wk in duration on resting BP and identified several moderators related to characteristics of the sample and the aerobic exercise intervention. Briefly, the authors found that samples consisting of all men experienced BP reductions that were greater in magnitude than those of all women (3–5 mmHg vs. 1 mmHg), concluding that sex may influence the BP response to exercise training. They also found that the magnitude of training-induced BP reductions was greater among studies that implemented exercise training programs lasting <24 wk than ≥24 wk (3–6 mmHg vs. 1–2 mmHg), involved aerobic exercise sessions lasting 30–45 min/session, and accumulated a weekly exercise volume of <210 min compared to a weekly volume ≥210 min. Last, aerobic exercise intensity appeared to alter the BP response to training such that BP reductions were greatest with moderate-to-vigorous-intensity aerobic exercise training compared to lower intensity aerobic exercise (4–5/2–3 mmHg vs. ≈1 mmHg) [142].

A growing body of new and emerging evidence suggests that high-intensity interval training (HIIT), characterized by brief periods of very high-intensity aerobic exercise (>90% of VO_2max) separated by recovery periods of lower-intensity exercise or rest [143], may be superior to continuous, moderate-intensity aerobic exercise in lowering BP. Indeed, a recent review found that HIIT resulted in greater BP benefit for individuals with hypertension than normal BP (8 mmHg vs. 3 mmHg, respectively) [91]. HIIT holds promise for some people with hypertension because it allows individuals to perform brief periods of vigorous-intensity exercise that would not be tolerable for longer periods of time. In addition, HIIT can also yield an equal amount of work (i.e., energy expenditure) compared to continuous, moderate-intensity exercise in a shorter amount of time [91, 143–145]. Additional investigations involving more diverse samples with hypertension are warranted.

In summary, the BP reductions experienced after aerobic exercise training appear to be equivalent in magnitude to those observed with PEH (i.e., 5–7 mmHg) [136, 137, 139], supporting the notion that BP reductions following chronic exercise or exercise training are largely a function of PEH. Furthermore, as with PEH, the magnitude of the BP reductions with exercise training appears to be greatest in those with the highest BP [136–139, 141], and exercise intensity appears to be an important moderator of the BP response to aerobic exercise training, with BP reductions occurring in a dose-response fashion [142]. HIIT shows promise as a viable alternative to the current ACSM Ex R_x recommendations for hypertension; however, further investigation is warranted among individuals with hypertension to more definitively determine the benefit-to-risk ratio of exercising at vigorous intensity among a population that is predisposed to heightened CVD risk [91, 143] (for an expanded discussion on the risks associated with exercise, see Chap. 25). Last, the FITT components of the aerobic exercise training intervention (i.e., the duration, intensity, and weekly volume of exercise) and population characteristics (i.e., sex, race/ethnicity) appear to moderate the BP response to aerobic exercise training and warrant confirmation in future randomized controlled trials [98, 142].

Interventional Evidence of Dynamic Resistance Exercise and Blood Pressure Effects

Dynamic resistance exercise involves both a lifting and lowering phase that occurs during each repetition against an external resistance, or load does not change. These phases of muscular activity correspond to the shortening and lengthening of the involved muscle group. It was previously thought that individuals with hypertension should avoid resistance exercise due to reports of marked elevations in BP while exercising [146] and

following the Valsalva maneuver [147]. Indeed, increases in BP as high as 480/350 mmHg have been recorded among bodybuilders during a single bout of heavy resistance exercise performed at or above 80% of 1RM until concentric failure [146]. However, such BP surges are known to return to initial values within 10 s of the last repetition of each set [146], and to the best of our knowledge, it remains to be answered whether brief elevations in BP of this magnitude are harmful. In fact, there is an established, but growing, body of literature that shows muscular strength is inversely associated with CVD and all-cause mortality [85, 90, 148, 149], incident hypertension, and prevalence of other adverse cardiometabolic health outcomes [81–83, 90].

Acute, Immediate, or Short-Term Effects, or Postexercise Hypotension

An increasing number of studies [150–155] as well as reviews and meta-analyses [122, 123, 125, 156] are reporting immediate reductions in BP following acute dynamic resistance exercise that persist for several hours after the bout or PEH. Furthermore, the magnitude of these reductions appears to be the greatest among those with the highest BP. Indeed, a recent meta-analysis [156] of 30 acute dynamic resistance exercise studies (81 interventions) involving 646 adults with normal BP to established hypertension concluded that dynamic resistance exercise elicited PEH to a greater extent among samples with hypertension ($n = 141$) compared to samples with normal BP ($n = 505$) (9/5–6 mmHg vs. 3/3 mmHg, respectively) ($P < 0.01$). This same meta-analysis also identified several moderator patterns related to the FIT of the dynamic resistance exercise intervention that will be discussed as new and emerging research below.

A limited number of studies have examined aspects of the FIT of an acute resistance exercise intervention on PEH, and when they have, the results have been mixed. For example, some studies have shown that high-intensity (80% of 1RM) acute resistance exercise results in greater BP reductions than light-moderate-intensity (50% of 1RM) resistance exercise (≈34/16 mmHg vs. ≈24/7 mmHg) [150, 151], while other studies have reported reductions of similar magnitude following light- (40% of 1RM) and high (80% of 1RM)-intensity acute resistance exercise (14/1–2 mmHg) [152].

More consistent evidence seems to support that the volume of resistance exercise (i.e., the number of exercises, repetitions, and sets of a given exercise) more so than the intensity moderates the magnitude of PEH. For example, Scher et al. [155] examined the effect of low versus high volume on PEH among 16 older adults with treated hypertension (130/76 mmHg). All subjects performed 1 set of 20 repetitions at light intensity (40% of 1RM) at each station in the 10-exercise circuit; however, the number of total circuits (laps) differed such that subjects completed two sessions that consisted of low (1 lap; 20 min) and high (2 laps; 40 min) volume. The authors reported that both low and high volumes of resistance exercise elicited PEH for 60 min in the laboratory compared to control (Ps < 0.05), but the magnitude of these reductions was greater after high- rather than low-volume resistance exercise (10/7 mmHg vs. 8/6 mmHg, respectively) ($P < 0.05$). Interestingly, only high-volume resistance exercise reduced awake and 24-h ambulatory SBP compared to control, and again, these reductions were greater than those observed after lower volumes of resistance exercise (Ps < 0.05).

Two recent meta-analyses seem to confirm these observations. Cassonatto et al. [156] reported that the magnitude of PEH was greater following a bout of resistance exercise that involved larger muscle groups (targeted with either single- or multi-joint movements) than smaller muscle groups (SBP only: 3–5 mmHg vs. 1–2 mmHg) ($P < 0.05$), and in a separate meta-analysis, Carpio-Rivera et al. [125] found that PEH magnitude was associated with the number of exercises performed in a given session ($r = -0.20$) and the number of sets performed per exercise ($r = -0.47$) (Ps ≤ 0.01) such that a greater number of resistance exercises and sets per exercise elicited greater reductions in BP.

To summarize, acute resistance exercise can lead to remarkable surges in BP while exercising;

however, whether this phenomenon is harmful to overall cardiovascular health is unknown [146]. In fact, most of the studies involving individuals with hypertension have reported immediate reductions in BP following a single bout of dynamic resistance exercise that appear to be clinically meaningful and comparable to the magnitude of PEH resulting from acute aerobic exercise among individuals with hypertension (i.e., ≈5–10 mmHg vs. 5–7 mmHg, upwards of 8–11 mmHg based on new and emerging research presented in this chapter) [150–155]. Similar to the BP benefits resulting from acute and chronic aerobic exercise, BP reductions following acute dynamic resistance exercise appear to be more pronounced in individuals who stand to benefit the most (i.e., those with higher BP compared to normal BP) [125, 156, 157]. At this time, it is unclear whether other patient characteristics or aspects of the FIT of the acute resistance exercise intervention influence PEH.

Chronic, Training, or Long-Term Effects

Until recently, the general consensus from randomized controlled trials and meta-analyses [95, 158] has been that dynamic RT lowers resting BP, but to a lesser degree than aerobic exercise training (i.e., 2–3 mmHg vs. 5–7 mmHg). A major limitation of earlier meta-analyses resided in the fact that the majority of included studies involved white and/or middle-aged adults with normal BP and elevated BP, limiting the generalizability of the results to more ethnically diverse populations and, importantly, to those with hypertension [95].

Recent investigations, and meta-analyses of these newly published studies, have shown that BP reductions following dynamic RT are similar to those resulting from aerobic exercise training. For example, Mota et al. [154] found that 16 wk of moderate-intensity (i.e., 70% of 1RM) dynamic RT reduced resting BP 14/4 mmHg among women with treated hypertension. Likewise, Moraes et al. [159] reported that 12 wk of moderate-intensity (60% of 1RM) dynamic RT reduced resting BP 16/12 mmHg among men with hypertension. Most recently, we meta-analyzed 64 controlled studies (71 interventions) involving middle-aged adults (n = 2344), the majority of who were white (57%) with elevated BP (126/76 mmHg) [158]. On average, we found that dynamic RT interventions of moderate intensity, performed 2–3 d/wk for 14 wk, elicited BP reductions of ≈3/2 mmHg. However, subsequent moderator analyses revealed dynamic RT elicited greater BP reductions among individuals with hypertension (6/5 mmHg) and elevated BP (3/3 mmHg) compared to normal BP (0/1 mmHg).

Of note, we found that the antihypertensive effects of dynamic RT were moderated by race/ethnicity, such that among nonwhite samples with hypertension, BP was reduced 14/10 mmHg, a magnitude that is approximately twice that reported following aerobic exercise training (i.e., 5–7 mmHg) [158]. These promising findings suggest that, for some populations, dynamic RT elicits BP reductions comparable to or greater than those achieved with aerobic exercise training, and for those patients (i.e., nonwhite samples with hypertension), dynamic resistance exercise may serve as a viable stand-alone antihypertensive lifestyle therapeutic option [158].

Presently, professional committees/organizations recommend that individuals with hypertension engage in moderate-intensity, *dynamic* resistance exercise 2–3 d/wk as a supplement to aerobic exercise training. Upon more careful scrutiny of the literature [91] and the new findings by MacDonald et al. [158], there is suggestive evidence that dynamic resistance exercise can be an alternative stand-alone exercise modality option for patients with hypertension. These findings are consistent with the new and emerging evidence regarding the acute effects of dynamic resistance exercise presented in the previous section. Additional randomized controlled, intervention trials are needed to determine if these novel findings prove to be true and to better understand what FIT features of the dynamic RT program would yield the greatest BP benefit.

In summary, it has long been thought that dynamic RT reduces BP ≈ 2–3 mmHg. However, new and emerging research has demonstrated that dynamic resistance exercise has an even more beneficial influence on BP among those

diagnosed with hypertension (i.e., ≈5–6 mmHg, upwards of ≈10–14 mmHg) [158–160]. Additionally, of the few meta-analyses that have explored the influence of population characteristics, they have shown that initial BP levels and race/ethnicity can influence the magnitude of reductions experienced after dynamic RT and warrant additional investigation [158]. Based on the available evidence, it is unclear whether other aspects of the FIT of the Ex R_x influence the magnitude of BP reductions following dynamic RT.

Interventional Evidence of Isometric Resistance Exercise and Blood Pressure Effects

Isometric resistance exercise involves sustained contraction against an immovable load or resistance with no (or minimal) change in length of the involved muscle group. To date, two different forms of isometric resistance exercise have been evaluated in terms of their effectiveness to lower BP: isometric handgrip (IHG) and isometric leg exercise. Currently, the ACSM and other professional committees/organizations (Table 8.2) do not provide guidelines on isometric resistance exercise for adults with hypertension due to the limited evidence supporting its effectiveness to lower high BP [23, 24]. In 2013, a Scientific Statement from the AHA featured IHG as a potentially effective adjunctive alternative therapy for lowering BP but stated there were inconsistent or inclusive data regarding its efficacy as antihypertensive therapy at the time [161]. Additional studies have been published since then, and two professional committees/organizations [10, 103] included IHG exercise as another potentially viable exercise-based therapeutic option for adults with hypertension in their most recent treatment guidelines (Table 8.2). Despite these recent endorsements, significant gaps in the current state of knowledge regarding the effects of isometric exercise on resting BP remain. Furthermore, there are fewer well-controlled isometric exercise interventions compared to aerobic and dynamic resistance exercise, especially among those involving adults with hypertension [142, 162, 163].

Acute, Immediate, or Short-Term Effects, or Postexercise Hypotension

The antihypertensive effects of isometric exercise among adults with hypertension have primarily been evaluated in terms of their long-term or chronic BP benefit [162, 164, 165]. A limited number of studies have evaluated PEH following a single bout of isometric exercise among adults with hypertension [166–170] and with mixed results.

Two studies [169, 170] observed PEH following a single bout of IHG exercise, reporting reductions in ambulatory SBP of 5.4 mmHg over 7 hours under conditions of daily living [169] and office SBP reductions ranging from 14.4 to 18.7 mmHg during 60 min of recovery in the laboratory [170]. In contrast, three studies did not observe PEH following a single bout of IHG exercise [166–168]. Reasons for the discrepancies observed among these studies are not completely clear but appear to be the result of differences in study design (e.g., ambulatory vs. laboratory assessment of PEH) and patient clinical characteristics (e.g., age, baseline BP, race/ethnicity, physical activity level, among others). Of note, the study of Ash et al. [166] is the only study to date to directly compare the magnitude and duration of PEH (laboratory/office BP and ambulatory BP over 19 h) after acute IHG exercise with acute aerobic exercise in the same group of adults with elevated BP to stage 1 hypertension (n = 27). Using a randomized controlled crossover design, Ash et al. [166] reported that aerobic exercise induced a clinically meaningful reduction in ambulatory BP compared to non-exercise control (4–6 mmHg) over awake hours, while IHG failed to elicit PEH. In addition to differences in experimental study design, Ash et al.'s [166] participants were sedentary, with low cardiorespiratory fitness, elevated to stage 1 hypertension, not receiving antihypertensive medication, and obese, and the majority (56%) were African-Americans – a major contrast to the study participants reporting favorable BP effects following IHG exercise (i.e., participants were physically active, with average cardiorespiratory fitness, normal BP, or normal BP levels controlled with antihypertensive

medication, of normal weight, and mostly Caucasian) [162, 164, 169, 170].

In summary, there is a lack of compelling evidence at this time to support that acute isometric resistance exercise, particularly IHG exercise, elicits PEH among adults with elevated to established hypertension.

Chronic, Training, or Long-Term Effects

As previously stated, the majority of isometric resistance exercise studies including adults with hypertension have examined the long-term or chronic BP effects [162, 164, 165, 171, 172]. Based on the findings from two recent meta-analyses [162, 164], IHG training consisting of four sets × 2 min unilateral IHG contractions at 30% MVC performed 3 d/wk for 8 wk or longer reduced resting BP 4–5/5–6 mmHg among adults with hypertension (n = 61), all of whom were on medication. Carlson et al. [164] and Inder et al. [162] also found that IHG training significantly reduced resting BP among adults with normal BP (n = 162 and n = 217). Interestingly, both meta-analyses reported that SBP was reduced to a greater extent among adults with normal BP compared to those with hypertension, while the opposite was true for DBP: 7.8/3.1 mmHg versus 4.3/5.5 mmHg [164] and 5.4/2.9 mmHg versus 4.5/5.5 mmHg [162], respectively. Both meta-analyses were unable to explain the larger reductions in SBP among the adults with normal BP compared to adults with hypertension, and the reverse pattern observed for DBP. Furthermore, the small sample of participants with hypertension were derived from the same three studies [173–175] in both meta-analyses. Their sample of adults with hypertension (n = 61) is much smaller in size than the samples of adults with hypertension in meta-analyses investigating aerobic [142], dynamic resistance [158] and combined aerobic and dynamic resistance [176] exercise training. For these reasons, any conclusions made about the antihypertensive benefits of isometric RT should be made with caution.

Despite earlier claims of superior BP-lowering effects resulting from isometric resistance exercise, not all investigations have come to a similar conclusion. Moreover, few studies have directly compared the BP response to isometric RT with aerobic or dynamic resistance exercise, modalities that are currently recognized as primary or adjuvant lifestyle therapy for adults with hypertension. Of interest are three recently published studies [166, 172, 177] that evaluated the long-term or chronic changes in resting and ambulatory BP induced by isometric RT compared to aerobic exercise training, which is universally endorsed for the primary prevention and treatment of hypertension. Collectively, these studies [166, 172, 177] reported that aerobic exercise training significantly reduced office (≈4–10/3–4 mmHg) [172, 177] and ambulatory BP over waking hours (≈5.5–8/4.4–5 mmHg) [166, 172] post- compared to pre-intervention, while IHG training did not.

To summarize, earlier studies and meta-analyses of these studies suggest that isometric resistance exercise may reduce resting BP on average 4–5/5–6 mmHg among adults with hypertension. However, the majority of isometric resistance exercise studies have included either young, healthy white men with normal BP or older, white men with well-controlled hypertension through pharmacological agents [163, 178]; women and racial/ethnic minorities have been consistently underrepresented in this literature. Although once promising, more recent research questions the effectiveness of isometric RT as antihypertensive therapy.

Interventional Evidence of Concurrent Exercise and Blood Pressure Effects

Concurrent exercise is defined as aerobic and resistance exercise being performed in close proximity to each other (i.e., in a single exercise session or within a couple hours of one another) [104, 179]. The ACSM does not currently provide any guidelines on concurrent exercise for adults with hypertension due to the limited number of studies that have investigated the acute and chronic effects of concurrent exercise on BP

among adults with hypertension. However, based on the new and emerging evidence that will be discussed in the sections that follow, it appears that concurrent exercise may be as effective as aerobic exercise as antihypertensive therapy among individuals with hypertension [91].

Acute, Immediate, or Short-Term Effects, or Postexercise Hypotension

In comparison to the aerobic and dynamic resistance exercise literature, fewer studies have investigated the acute effects of concurrent (*combined* aerobic and resistance) exercise among adults with high BP [104, 108, 179]. Collectively, these studies reported BP reductions ranging from 6 to 12 mmHg for SBP and 3 to 17 mmHg for DBP, respectively [179–184]. Based on the limited number of small studies conducted to date, it is likely premature to comment on the population or FIT characteristics that may modulate the duration and magnitude of PEH following acute concurrent exercise. At this time, however, no clear moderator patterns have emerged in relation to the intervention characteristics such as the concurrent exercise intensity, volume, or the order of the aerobic and resistance exercise components and the magnitude and duration of PEH.

For example, a single bout of concurrent exercise consisting of moderate-to-vigorous-intensity aerobic exercise and moderate-intensity resistance exercise (70–75% of 1RM) lowered BP 6–11/3–5 mmHg and remained lowered for 60–90 min following the bout [180, 182, 183]. Similarly, acute concurrent exercise consisting of moderate-to-vigorous-intensity aerobic exercise and low-intensity resistance exercise (40–50% of 1RM) lowered BP by 7–12/3–17 mmHg for 30–180 min following the bout [181, 184]. Tibana et al. [180] observed a similar effect when investigating the influence of moderate-intensity aerobic exercise (30 min at 70% of HR reserve) followed by either low- (1 set) or high (3 sets)-volume resistance exercise (8–12 repetitions for 6 exercises at 80% of 10RM workload) among 16 women with normal to high BP. They reported that SBP was reduced 7–9 mmHg compared to control for 90 min following both concurrent exercise sessions. Lastly, the order of the aerobic and resistance exercise components within the concurrent exercise session does not appear to influence PEH among adults with hypertension. Menêses et al. [184] found that aerobic exercise (30 min at 50–60% of HR reserve) performed before or after dynamic resistance exercise (3 sets of 10 repetitions for 7 exercises at 50% of 1RM) elicited PEH among 19 middle-aged women with controlled hypertension (130/68 mmHg), and the magnitude of these reductions was not different between conditions (7–8/3 mmHg).

In contrast to the concurrent exercise variables discussed thus far, *how* the concurrent exercise session is performed (i.e., single bout vs. multiple bouts interspersed throughout the day) does appear to modulate PEH. In a recent study, Azevêdo et al. [182] investigated the effects of concurrent exercise (3 sets of 10 repetitions for 4 exercises at 75% of 8RM workload followed by 20 min of moderate-to-vigorous-intensity aerobic exercise) performed in a single bout (in the morning *or* evening) compared to fractionized (spread throughout the day, in the morning, *and* in the evening) on PEH among 11 middle-aged women with hypertension. The authors found that a single session of concurrent exercise performed in the morning *or* evening, but not fractionized throughout the day, reduced SBP 10.7 mmHg and 6.3 mmHg compared to control. Furthermore, reductions in SBP were greater following a single bout of concurrent exercise performed in the morning compared to fractionized concurrent exercise (−10.7 mmHg vs. +3.3 mmHg, respectively).

To summarize, a single bout of concurrent exercise elicits PEH among middle-aged to older adults with high BP, and the magnitude of these reductions appears to be similar to those observed after aerobic exercise. Moreover, based on the limited number of small studies conducted to date, the currently available data does not suggest that PEH is modulated by aspects of the acute concurrent exercise intervention (i.e., exercise intensity, volume, or the order of the aerobic and

resistance exercise components). However, further investigation is warranted among larger samples of adults with hypertension to determine whether aspects of the FIT of the acute concurrent exercise intervention influence PEH. At this time, it is unclear whether patient characteristics influence PEH.

Chronic, Training, or Long-Term Effects

The results of several recent meta-analyses and concurrent exercise training studies suggest that concurrent exercise may be as effective as aerobic exercise as antihypertensive therapy among individuals with hypertension [91, 176]. Briefly, Hayashino et al. [185] performed a meta-analysis of 42 trials, 14 of which were concurrent exercise training trials involving middle-aged adults with type 2 diabetes mellitus and hypertension (36%). The authors reported BP reductions of 1.7/2.3 mmHg following aerobic training, 2.8/2.3 mmHg following dynamic RT, and 3.2/1.9 mmHg following concurrent exercise training, with no statistically significant differences among the three modalities [185]. Cornelissen and Smart [142] reported similar findings in their meta-analysis of 93 trials, such that BP was reduced 3.5/2.5 mmHg following aerobic exercise training (105 interventions), 1.8/3.2 mmHg following dynamic resistance exercise training (29 interventions), and 2.2 mmHg (SBP only) following concurrent exercise training (14 interventions). Once again, no differences were noted among the three modality groups.

Most recently, Corso et al. [176] pooled 68 trials (76 interventions) and examined the influence of concurrent exercise training on BP. The authors found, on average, concurrent exercise training performed 3 d/wk at moderate intensity (aerobic, 55% of VO_2max; resistance, 60% of 1RM), ≈60 min/d for 20 wk, significantly reduced BP by 3.2/2.5 mmHg. However, among trials of higher study quality that examined BP as the primary outcome, individuals with hypertension experienced BP reductions as great as 9.2/7.7 mmHg [176]. BP reductions of this magnitude are clinically meaningful and, if confirmed, could result in an expansion of the existing professional exercise recommendations for hypertension to include concurrent exercise training. Interestingly, as stated within the ACSM's current exercise recommendations [23], individuals with hypertension would almost *always* be engaging in concurrent exercise training (i.e., aerobic exercise supplemented by dynamic RT). Therefore, it is imperative that future trials better explore the combined influence of aerobic and dynamic resistance exercise as antihypertensive lifestyle therapy among individuals with hypertension to determine whether patient characteristics or aspects of the FIT of the Ex R_x influence the BP response to chronic concurrent exercise.

In summary, new and emerging research examining the influence of aerobic exercise training in combination with dynamic RT (i.e., concurrent exercise training) is promising as it appears that concurrent exercise may elicit chronic BP reductions similar in magnitude to those seen after aerobic exercise training [91, 176, 179]. However, future randomized controlled trials are needed before concurrent exercise training can be integrated in the Ex R_x for individuals with hypertension.

Mechanisms of Exercise-Induced Blood Pressure Changes

An isolated bout of aerobic, and now, based on new and emerging evidence, dynamic resistance and concurrent exercise, elicits immediate reductions in BP of 5–7 mmHg among individuals with hypertension that persist for up to 24 h or PEH [10, 24]. The precise mechanisms responsible for PEH are not clear. However, it is unlikely that PEH is the result of a single underlying mechanism given the complexity of BP regulation and the multifactorial pathogenesis of hypertension. Rather, PEH represents a collection of complex hemodynamic adjustments that occur in response to and in recovery from exercise, and these adjustments are driven by several highly coordinated and controlled mechanisms [29,

186]. Arterial pressure is determined by CO and TPR; thus, the mechanisms that mediate acute and chronic exercise-induced BP reductions can be further discussed in terms of their individual determinants: HR and stroke volume (CO) and the degree of vasoconstriction or vasodilation of individual vascular beds (TPR).

PEH following acute aerobic exercise is characterized by a reduction in vascular resistance resulting from peripheral vasodilation. This sustained postexercise vasodilation is mediated by combined central neural mechanisms (arterial baroreflex resetting) and local vasodilatory mechanisms [186]. PEH following acute dynamic resistance exercise, in contrast, is characterized by an attenuation in CO resulting from reduced stroke volume and increase in vascular resistance [186]. The underlying mechanisms of PEH, hemodynamic adjustments (e.g., regional vascular changes), and autonomic contributions (e.g., baroreflex resetting) are better documented following aerobic than resistance exercise. However, differences in the mechanisms that underlie PEH may be related to changes in cardiac sympathetic activation and/or arterial baroreflex sensitivity, as well as local vasodilatory controls.

BP reductions observed with exercise training are primarily the result of reductions in TPR. Although exercise training can alter HR and stroke volume, the net effect on resting CO is minimal among healthy populations. Peripheral resistance is mediated by neurohumoral and structural adaptations such as increased vasodilatory factors (e.g., nitric oxide), decreased vasoconstrictor factors (e.g., norepinephrine), increased vessel diameter, and/or increased vessel distensibility [29, 34] (for an expanded discussion on exercise and the endothelium, see Chap. 3). Therefore, reductions in TPR after acute exercise appear to be predominately the result of exercise-induced alterations involving the sympathetic nervous and renin-angiotensin systems and their influence on vascular, renal, and baroreceptor function [29, 118–120]. Frequent repetition of acute exercise bouts produces more permanent structural adaptations and persistent alterations in function, leading to long-term reductions in BP (i.e., the exercise training response).

Clinical Implications and New Advances in Exercise Prescription for Optimizing Blood Pressure Benefits

The FITT Ex R_x recommendations that follow are based upon the new and emerging findings discussed in this chapter as they related to the current consensus of knowledge regarding the effects of acute and chronic aerobic, dynamic resistance, and concurrent exercise on hypertension as presented in this chapter. The modified Ex R_x we propose below is summarized in Table 8.4:

*F*requency:

- Aerobic exercise should be performed on most, preferably all, days of the week (i.e., 5–7 d/wk) in combination with dynamic resistance exercise 2–3 d/wk on non-consecutive days.
- A combination of aerobic and dynamic resistance exercise can be performed on separate days (i.e., *combined* aerobic and resistance exercise) or during the same exercise session (i.e., *concurrent* exercise).
- Aerobic and resistance exercise should be supplemented by flexibility exercise 2–3 d/wk.

This recommendation is made due to the well-established immediate and sustained BP lowering effects of acute aerobic exercise or PEH [22, 23, 91, 108, 118–120, 124–126, 129, 130], and the new and emerging evidence that supports acute dynamic resistance [150–153, 155–157] and concurrent [179–184] exercise can elicit PEH to a similar magnitude as aerobic exercise among adults with hypertension. Put simply, BP is lower on days when individuals with hypertension exercise than the days they do not exercise.

Table 8.4 The modified exercise prescription for adults with hypertension based on new and emerging evidence [91, 104]

FITT-VP principle of the Ex R_x	Modified ACSM recommendations
*F*requency (how often?)	Aerobic exercise: 5–7 d/wk Dynamic resistance exercise: ≥2–3 d/wk (non-consecutive) A combination of aerobic and dynamic resistance exercise can be performed on separate days (i.e., *combined* aerobic and resistance exercise) or during the same exercise session (i.e., *concurrent* exercise) *What's new*: The addition of dynamic resistance as another viable stand-alone antihypertensive therapeutic option
*I*ntensity (How hard?)[a]	Aerobic exercise: moderate-to-vigorous (40–≥60% of $\dot{V}O_2R$ or 12–≥14 on a scale of 6 [no exertion] to 20 [maximal exertion] level of physical exertion or an intensity that causes noticeable increases in heart rate and breathing) Dynamic resistance exercise: moderate (60–80% of 1RM or 12–14 on a scale of 6 [no exertion] to 20 [maximal exertion] level of physical exertion) Concurrent exercise: a combination of moderate-to-vigorous-intensity aerobic exercise and moderate-intensity resistance exercise as described above *What's new*: The expansion of the recommendation for aerobic exercise intensity to include higher levels of physical exertion (i.e., vigorous-intensity aerobic exercise)
*T*ime (how long?)	Aerobic exercise: ≥30–60 min/d; one continuous bout or multiple bouts of ≥10 min (moderate intensity) or ≥ 3–10 min (vigorous intensity, i.e., ≥60% of $\dot{V}O_2R$) each Dynamic resistance exercise: 8–10 exercises; 1–4 sets of 8–12 repetitions Concurrent exercise: ≥20–30 min/d of aerobic exercise and dynamic resistance exercise consisting of 4–8 exercises; 1–3 sets of 8–12 repetitions *What's new*: The inclusion of multiple "very short bouts" (i.e., 3–10 min), performed at vigorous intensity, that can be interspersed throughout the day Specific recommendations regarding the aerobic and resistance exercises when performed concurrently in a single exercise session
*T*ype (what kind?) *Primary*	Aerobic exercise: prolonged, rhythmic activities using large muscle groups (e.g., walking, cycling, swimming) performed continuously at a constant intensity or with repeated bouts alternating between high and low (≥70% vs. <40% of $\dot{V}O_2R$) intensity (i.e., *interval exercise*) Dynamic resistance exercise: a combination of multi- and single-joint exercises targeting the major muscle groups of the upper and lower body using traditional or circuit resistance training Concurrent exercise: a combination of the aerobic and resistance exercises described above performed in any order (i.e., aerobic exercise can be performed before *or* after resistance exercise) *What's new*: The recommendation for the type of aerobic exercise has been expanded to include interval exercise Specific recommendations regarding the order of aerobic and resistance exercises when performed concurrently in a single exercise session
Adjuvant 1	Flexibility *F:* ≥2–3 d/wk *I:* Stretch to the point of feeling tightness or slight discomfort *T:* ≥10 min/d; ≥4 repetitions per muscle group; hold each static stretch for 10–30 s

(continued)

Table 8.4 (continued)

FITT-VP principle of the Ex R_x	Modified ACSM recommendations
Adjuvant 2[b]	Neuromotor *F:* ≥2–3 d/wk *I:* undetermined *T:* ≥20–30 min/d
*V*olume (how much?)[c]	≥150 min/wk or 700–2000 kcal/wk of *total* aerobic and resistance exercise
*P*rogression	Progress gradually, avoiding large increases in any of the components of the Ex R_x; increase exercise duration over first 4–6 wk and then increase frequency, intensity, and time (or some combination of these) to achieve recommended quantity and quality of exercise over next 4–8 months

ACSM American College of Sports Medicine, *FITT f*requency, *i*ntensity, *t*ime, *t*ype, *v*olume, and *p*rogression of exercise; *Ex* R_x exercise prescription, 1*RM* one-repetition maximum, $\dot{V}O_2R$ oxygen consumption reserve

[a]Vigorous-intensity aerobic exercise (i.e., ≥60% of $\dot{V}O_2R$ or ≥ 14 on a scale of 6–20 [106]) appears to elicit greater and more extensive benefits than lower levels of physical exertion for individuals who are willing and able to tolerate more intense levels of exercise and may be introduced after exercise preparticipation health screening and gradual progression

[b]Balance (neuromotor) training is recommended for older adults, individuals who are at substantial risk of falling, and is likely to benefit younger adults as well

[c]For greater and more extensive benefits, progress exercise volume to total 60 min/d and 300 min/wk of moderate-to-vigorous intensity

Also, individuals with hypertension are often overweight to obese and have additional CVD risk factors (i.e., insulin resistance, dyslipidemia, the metabolic syndrome) [28, 37, 50]. Therefore, large amounts of caloric expenditure should be emphasized [24] while maintaining lean mass, muscular strength, and function [108].

*I*ntensity:

- Aerobic exercise: Moderate-to-vigorous intensity (i.e., 40–≥60% $\dot{V}O_2R$; 12–≥14 rating of perceived physical exertion on the Borg 6–20 scale [106]) or an intensity that causes noticeable increases in HR and breathing
- Dynamic resistance exercise: moderate-intensity (60–80% of 1RM; 12–14 rating of perceived physical exertion on the Borg 6–20 scale [106])

Due to the growing evidence that greater BP reductions can be achieved with greater levels of physical exertion [22, 91, 125, 129, 135], the aerobic exercise intensity recommendation has been expanded to include vigorous intensity if the patient or client is willing and able to tolerate higher levels of physical exertion. For dynamic resistance and concurrent exercise, it appears that moderate-intensity resistance exercise is efficacious for reducing BP among adults with hypertension [150–152, 155, 157, 159].

*T*ime:

- Aerobic exercise: 30–60 min/d of continuous or intermittent (i.e., fractionized) exercise. If intermittent, bouts should be ≥10 min (moderate-intensity exercise) or ≥3–10 min (vigorous-intensity exercise, i.e., >60–80% $\dot{V}O_2R$) in duration depending on the level of physical exertion and accumulate to total 30–60 min/d.
- Dynamic resistance exercise: should consist of at least 1 set of 8–12 repetitions for 8–10 exercises targeting the major muscle groups.
- Concurrent exercise: ≥20–30 min/d of continuous aerobic exercise and dynamic resistance exercise consisting of at least 1 set of 8–12 repetitions for 4–8 exercises targeting the major muscle groups.

This recommendation is consistent with existing evidence that PEH is a low-threshold phenomenon regarding the time (duration) of the acute aerobic exercise bout but has been expanded to consider the interaction between time and intensity. When several short bouts of aerobic exercise are interspersed throughout the day,

PEH offers a viable therapeutic lifestyle option for BP control among individuals with high BP [125, 130, 131]. Bouts of at least 10 min are recommended for moderate-intensity aerobic exercise, while bouts of <10 min (i.e., ≥3–10 min) may be recommended for more vigorous-intensity aerobic exercise. At this time, there is no compelling evidence to support that intermittent or fractionized resistance or concurrent exercise [182] can offer the same PEH benefit reported with a single, continuous bout of resistance or concurrent exercise, respectively.

*T*ype:

- For aerobic exercise, emphasis should be placed on prolonged, rhythmic activities using large muscle groups such as walking, cycling, or swimming. Aerobic activities can be performed continuously at a constant intensity or in repeated bouts alternating between high and low (≥70% vs. <40% $\dot{V}O_2R$) intensity (i.e., *interval exercise*).
- For dynamic resistance exercise, emphasis should be placed on multi- and single-joint exercises that target the major muscle groups of the upper and lower body. Resistance exercises can be performed using a conventional or circuit (i.e., lighter weights, higher repetitions, with minimal rest between exercises) RT protocol.
- For concurrent exercise, a combination of the aerobic and resistance exercises described above can be performed in the same exercise session, in any order (i.e., aerobic exercise can be performed before *or* after resistance exercise).
- For older adults or individuals who are at substantial risk of falling, neuromotor (balance) training 2–3 d/wk is also recommended as adjuvant exercise.

Aerobic exercise training has consistently been shown to lower BP among adults with hypertension, and now, new and emerging research supports that dynamic resistance and concurrent exercise training can elicit comparable BP reductions. Therefore, we have expanded this recommendation to include dynamic resistance exercise and concurrent exercise as viable stand-alone antihypertensive therapy that should be performed in addition to aerobic exercise. Consistent with our expanded recommendation for aerobic exercise intensity, we have also modified this recommendation to include interval aerobic exercise. BP reductions following aerobic interval training (e.g., HIIT) are similar to or exceed those observed with continuous, constant-intensity aerobic exercise [91, 132, 135, 143]. Furthermore, interval exercise allows adults with hypertension to experience the health and BP benefits associated with higher levels of physical exertion that would not be tolerable with longer duration exercise [91, 143–145]. The recommendation for dynamic resistance exercise has been expanded with specific information regarding the RT protocol. Existing as well as new and emerging evidence has shown that adults with hypertension experience similar BP benefit with conventional and circuit resistance exercise [108, 158]. Finally, there is no strong evidence to support that the order of the aerobic and resistance exercise performed in a single concurrent exercise session influences PEH or the BP reductions that occur with training.

Special Considerations

- Consideration should be given to the level of BP control, recent changes in antihypertensive drug therapy, medication-related adverse effects, the presence of target organ disease and other comorbidities, and age. Adjustments to the Ex R_x should be made accordingly. In general, progression should be gradual, avoiding large increases in any of the FITT components of the exercise prescription, especially intensity.
- An exaggerated BP response to relatively low exercise intensities and at HR levels <85% of the age-predicted maximum HR is likely to occur in some individuals, even after resting BP is controlled with antihypertensive medication (<130 and <80 mmHg). In some cases, an exercise test may be beneficial to establish the exercise HR corresponding to the exaggerated BP in these individuals.

- Exercise is contraindicated if resting SBP exceeds 200 mmHg or DBP exceeds 110 mmHg.
- When exercising, it is prudent to maintain a SBP less than 220 mmHg and/or DBP less than 105 mmHg.
- Although vigorous-intensity aerobic exercise is not necessarily contraindicated in patients with hypertension, moderate-intensity aerobic exercise is generally recommended to optimize the benefit-to-risk ratio.
- Individuals with hypertension are often overweight or obese. The Ex R_x should focus on increasing caloric expenditure coupled with reducing caloric intake to facilitate weight reduction and minimize weight gain (see Chap. 11).
- Inhaling and breath holding while engaging in the actual lifting of a weight (i.e., Valsalva maneuver) can result in extremely high BP responses, dizziness, and even fainting. Thus, such practice should be avoided during resistance exercise.
- Individuals taking antihypertensive medications should be monitored during and after exercise for potential adverse interactions with exercise (see Table A.1 in the ACSM's Guidelines for Exercise Testing and Prescription [24] for a comprehensive summary of the effect of antihypertensive medications at rest and in response to exercise).
- β-Blockers and diuretics may adversely affect thermoregulatory function or increase the predisposition to hypoglycemia in certain individuals. Individuals taking β-blockers and diuretics should be well informed about signs and symptoms of heat intolerance and/or hypoglycemia and should be educated on how to make prudent modifications in their exercise routine to prevent adverse events.
- β-Blockers may also attenuate the HR response to exercise, while α-blockers, calcium channel blockers, and vasodilators may lead to sudden excessive reductions in postexercise BP. Therefore, an adequate cooldown may be especially important for patients taking these medications.
- Given that the BP lowering effects of exercise are immediate (i.e., a physiological response termed PEH), individuals should be educated about these immediate BP benefits to possibly enhance adherence.

Gaps in the Literature and Future Research Needs in the Exercise Prescription for Hypertension

It should be noted that these recommendations are limited by methodological quality of the studies upon which the evidence is based [91, 95]. Major limitations in the current state of the literature include small sample sizes, assessing study populations with normal BP rather than hypertension, not accounting for major confounders to the BP response to exercise that include timing of the last bout of exercise and detraining effects, and lack of standard protocols for the assessment of BP and the exercise intervention. As a result of these limitations, the effectiveness of exercise as antihypertensive lifestyle therapy among individuals with hypertension has been underestimated [91, 95, 105]. Furthermore, large randomized clinical trials that examine both the acute and chronic BP lowering effects of exercise among diverse populations are needed before professional organizations can definitively determine the optimal Ex R_x for individuals with hypertension.

Key Points

- Hypertension is the most common, modifiable, and costly CVD risk factor.
- Adults with hypertension are encouraged to engage in 30 min/d (or more) of moderate-intensity aerobic exercise on most, if not all, days of the week in addition to moderate-intensity dynamic resistance exercise 2–3 d/wk to total 150 min/wk of total exercise (or more) to prevent and control high BP (Table 8.4).
- The antihypertensive effects of acute and chronic aerobic exercise are a low-duration

phenomenon with intermittent durations appearing as effective as continuous durations in lowering BP.

- Exercise intensity is an important moderator of the antihypertensive effects of acute and chronic aerobic exercise with individuals with the highest BP experiencing the greatest BP benefit. Furthermore, vigorous-intensity exercise appears to elicit greater and more extensive benefits than lower levels of physical exertion for individuals who are willing and able to tolerate more intense levels of exercise and may be introduced after exercise preparticipation health screening and gradual progression (Table 8.4).
- Acute resistance exercise may result in marked elevations in BP while exercising; however, these BP surges appear to immediately decrease back to levels below that of baseline; the magnitude of the BP reductions is clinically meaningful and is most pronounced in individuals who stand to benefit the most (i.e., those with higher BP compared to normal BP).
- It has long been thought that dynamic RT reduces BP ≈ 2–3 mmHg. However, new and emerging research has demonstrated that dynamic RT has an even more beneficial influence on BP among those diagnosed with hypertension, reductions that are comparable to or greater than those achieved with aerobic exercise training. Based on this new and emerging research, we recommend that adults with hypertension perform dynamic RT *in addition to* aerobic exercise as stand-alone antihypertensive lifestyle therapy (Table 8.4).
- New and emerging research also indicates that aerobic and resistance exercise performed *concurrently* reduces BP to levels similar to that of aerobic exercise training. Future research is needed to confirm these findings.
- Despite the volume of literature on exercise and hypertension, there remains a critical need to identify patient and FITT exercise interventions' characteristics that influence the BP response to acute and chronic exercise so that exercise can be more precisely prescribed as antihypertensive therapy.

Conclusions

Hypertension is one of the most important CVD risk factors due to its high prevalence and significant medical costs [1, 2]. Indeed, hypertension affects approximately 46% of adults in the United States (≈103 million) [10, 20] and more than 31% of adults worldwide (>1.4 billion) [21]. Both the 2017 ACC/AHA guideline [10] and the ACSM [23] recommend aerobic exercise supplemented by dynamic resistance exercise as initial lifestyle therapy for individuals with hypertension because it lowers BP 5–7 mmHg and 2–3 mmHg, respectively, among those with hypertension. BP reductions of this magnitude can decrease the risk of developing CVD by 20–30% and rival those obtained with first-line antihypertensive medications [9, 10, 13, 96] as well as with other types of lifestyle therapy. New and emerging research has shown dynamic resistance and concurrent exercise to elicit BP reductions comparable to or greater than those achieved with aerobic exercise training and may serve as viable stand-alone antihypertensive therapeutic options for some patients. We have expanded the current exercise recommendations for hypertension in Table 8.4 to include this, as well as the other new and emerging research discussed in this chapter. Future research that addresses the existing research gaps is needed to confirm these findings.

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