The Role of Exercise Blood Pressure in Hypertension: Measurement, Mechanisms, and Management

KATHARINE D. CURRIE[1], MARTIN G. SCHULTZ[2], PHILIP J. MILLAR[3], and LINDA S. PESCATELLO[4]

[1]Department of Kinesiology, Michigan State University, East Lansing, MI; [2]Menzies Institute for Medical Research, University of Tasmania, Hobart, AUSTRALIA; [3]Department of Human Health & Nutritional Sciences, University of Guelph, CANADA; and [4]Department of Kinesiology, University of Connecticut, Storrs, CT

ABSTRACT

CURRIE, K. D., M. G. SCHULTZ, P. J. MILLAR, and L. S. PESCATELLO. The Role of Exercise Blood Pressure in Hypertension: Measurement, Mechanisms, and Management. *Med. Sci. Sports Exerc.*, Vol. 57, No. 2, pp. 425–433, 2025. Hypertension affects one in three adults globally and is the leading modifiable risk factor for cardiovascular disease. Although blood pressure measurements at rest are fundamental to the detection and management of hypertension, abnormal blood pressure responses to exercise, namely, an exaggerated exercise blood pressure (EEBP), can provide additional independent information about current and future hypertension risk. This paper summarizes a symposium entitled, "The Role of Exercise Blood Pressure in Hypertension: Measurement, Mechanisms and Management" included at the 2023 American College of Sports Medicine annual meeting, which presented a timely discussion about the clinical utility of EEBP. Here we will summarize the evidence presented by the speakers including considerations for blood pressure measurement during exercise, an overview of EEBP thresholds and discussion about the value of EEBP during submaximal exercise for the identification and management of hypertension, a summary of the potential physiological mechanisms underpinning an EEBP, and a review of exercise prescription guidelines based on new and emerging evidence as they relate to the American College of Sports Medicine's exercise recommendations for hypertension. We conclude by highlighting areas for future research with the overarching goal of improving the measurement and management of hypertension. **Key Words:** EXAGGERATED EXERCISE BLOOD PRESSURE, EXERCISE TESTING, EXERCISE TRAINING, PERIPHERAL VASCULAR RESISTANCE, SUBMAXIMAL EXERCISE

High blood pressure (BP) or "hypertension" is often referred to as the "silent killer" because it typically has no symptoms and is the leading modifiable risk factor for cardiovascular disease, the leading cause of mortality worldwide (1). Although hypertension is estimated to affect one out of every three adults globally, almost half of those do not know they have the condition (1). Furthermore, of those people receiving treatment for hypertension, less than a quarter have BP under control (2). These alarming statistics highlight the importance of properly identifying individuals with hypertension and developing effective tools for its prevention, detection, and treatment.

Most of the world defines hypertension as a systolic BP ≥140 mm Hg and/or a diastolic BP ≥90 mm Hg (1,3). In 2017, the United States (American College of Cardiology and American Heart Association) lowered the systolic and diastolic BP thresholds to 130 and 80 mm Hg, respectively, due to a "pragmatic interpretation of BP-related cardiovascular disease risk and benefit of BP reduction in clinical trials" (4). The rationale was to mitigate the deleterious effects of hypertension by identifying and intervening sooner. For example, the TROPHY study demonstrated that 2 years of treatment with an angiotensin-receptor blocker in pre-hypertensive middle-aged adults reduced the risk of developing hypertension over a subsequent 2-yr follow-up (5). A limitation of this work is the continued reliance on resting BP measurements to identify at-risk individuals. An ideal risk factor should be able to delineate future hypertension risk before resting BP is elevated.

Cardiopulmonary exercise testing is common in clinical cardiovascular risk assessment and involves the measurement of exercise BP for safety reasons (6,7). Repeated observational studies and meta-analyses have shown that an exaggerated exercise BP (EEBP; also termed a hypertensive response to exercise, exercise-induced hypertension, or exercise hypertension) has the capacity to identify individuals with masked hypertension (8), as well as individuals with an elevated risk of developing hypertension and cardiovascular disease morbidity and

Address for correspondence: Katharine D. Currie, Ph.D., IM Sports Circle, 308 W Circle Drive, East Lansing, MI 48842; E-mail: curriek4@msu.edu.

0195-9131/25/5702-0425/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/MSS.0000000000003569

mortality later in life (9,10). Despite established evidence demonstrating the clinical utility of EEBP, it is not commonly used for prognostic or diagnostic purposes.

This purpose of this paper is to summarize the proceedings of a scientific symposium presented at the 2023 annual meeting of the American College of Sports Medicine (ACSM) in Denver, Colorado. The symposium was entitled "The Role of Exercise BP in Hypertension: Measurement, Mechanisms and Management" and featured presentations by Drs. Katharine Currie, Martin Schultz, Philip Millar, and Linda Pescatello. Here we will summarize the main topics of the symposium, which were to 1) review current BP guidelines and outline considerations when measuring BP during exercise testing, 2) discuss the value of EEBP during submaximal exercise for the identification and management of hypertension, 3) highlight potential physiological mechanisms underpinning an EEBP, and 4) discuss exercise prescription updates based on new and emerging evidence as they relate to the ACSM exercise recommendations for hypertension.

BP MEASUREMENT AT REST AND DURING EXERCISE

Clinical practice guidelines for the measurement of resting BP in the office have been established by the American College of Cardiology and American Heart Association (4) and are summarized hereinafter:

- The individual should prepare by avoiding exercise, caffeine, and smoking for 30 min and emptying their bladder before the measurement.
- Ensure proper cuff size and position on the upper arm. The cuff should be placed directly on the skin and the arm should be supported at heart level.
- The individual should sit for at least 5 min with their back supported and feet flat on the floor. There should be no talking during the measurement.
- BP should be assessed in both arms and the arm with the higher reading should be used at subsequent visits.
- Systolic BP should be identified as the appearance of the first Korotkoff sound (phase I), whereas diastolic BP is identified as the disappearance of all Korotkoff sounds (phase V). A minimum of two measures should be taken and averaged.
- Measurements can be performed manually by a trained individual, or with the use of an automatic oscillometric device; only validated automatic devices are recommended.

During exercise, most of the same recommendations apply with a few exceptions (6,11). The individual may be positioned on an exercise ergometer (e.g., treadmill or cycle ergometer) with the arm supported at heart level. Individuals should refrain from gripping the handrails of the ergometer as muscle contraction can increase BP (11,12). Presently, there is no guidance on which arm to measure exercise BP and limited evidence exists on whether inter-arm differences in BP at rest persist or appear during exercise (13). Although bilateral measurements during exercise would provide more information, they are generally not feasible, and thus, arm selection at the present time is up to the observer. Diastolic BP measurement requires the identification of the disappearance of the Korotkoff sounds, which can be challenging during higher exercise intensities when there is more movement and ambient noise from the ergometer and participant (14). Manual measurements (auscultation) are recommended over automated measurements, due to questionable accuracy of automated (oscillometric) devices (11). The SunTech Tango M2 exercise BP monitor (SunTech Medical Instruments, Morrisville, NC) is an automated auscultatory device that determines BP using Korotkoff sounds. It has been shown to have reasonable accuracy in comparison to intra-arterial BP during exercise (15) and showed concordance with manual BP measurements during the early stages of clinical exercise stress testing (16). It may not always be possible to measure BP during exercise (e.g., 6-min walk test), and thus, measurements should be performed immediately upon completion of the exercise test. However, it should be noted that BP can rapidly drop following the cessation of exercise, and thus, it is perhaps more appropriate to consider such a measurement and its interpretation as a "post-exercise BP."

BP RESPONSES DURING EXERCISE: NORMAL VERSUS EXAGGERATED

During graded aerobic exercise, it is normal for systolic and mean arterial BP to rise in proportion with exercise intensity, while diastolic BP remains relatively constant or decreases slightly (6,17,18). These changes occur due to an accompanying increase in cardiac output and fall in total peripheral resistance mediated through a combination of cardiac parasympathetic withdrawal, systemic sympathetic activation, endothelium-dependent vasodilation, and local metabolic vasodilation designed to competitively balance the metabolic need for increased skeletal muscle blood flow with the maintenance of systemic perfusion pressure (19). According to the ACSM, systolic BP should increase approximately 10 mm Hg per 1 metabolic equivalent of task (MET) (6). The ACSM and American Heart Association also define an EEBP response to peak/maximal intensity exercise as a systolic BP ≥190 mm Hg in women and 210 mm Hg in men, and/or a peak diastolic BP of ≥90 mm Hg or if diastolic BP increases >10 mm Hg above baseline during the exercise test (6). The systolic EEBP thresholds of 190 mm Hg in women and 210 mm Hg in men are consistent with values ≥90th percentile obtained in large samples of young healthy adults (20,21). The European Society of Cardiology uses a peak systolic BP ≥200 mm Hg in women or ≥220 mm Hg in men, and/or a peak diastolic BP ≥80 mm Hg in women and ≥85 mm Hg in men to identify an EEBP (3). Test termination is recommended in both sexes if systolic or diastolic BP exceed 250 and 115 mm Hg, respectively (6,7). Data in support of using an absolute systolic BP threshold to define an EEBP response to peak/maximal exercise are provided by earlier studies (20,22). However, it is important to emphasize that, although the current definitions of an EEBP represent distinct cutoffs, the risk is likely linear, with meta-analytic work showing each 10 mm Hg increase in peak

exercise systolic BP associated with a 19% increased risk of incident hypertension (23). In addition, several recent studies have defined submaximal EEBP thresholds as low as systolic BP values ≥150 mm Hg during stages 1 or 2 of a standard Bruce treadmill protocol (24) through to systolic BP values ≥170 mm Hg during stage 2 of a Bruce treadmill protocol (25). However, there remains no clear definition of EEBP recorded during submaximal exercise.

Although exercise testing guidelines provide an important framework to operate within, it is important to consider emerging evidence. Recent studies have reported a much lower increase in systolic BP per MET. In males 20–59 yr old, the greatest increase in systolic BP/MET was observed in the oldest cohort (50–59 yr) and was, on average, 8.3 ± 2.3 mm Hg·MET^{-1} (26). In a sample of 7298 males (mean age of 59 ± 11 yr), the median increase was 6.2 ± 4.7 mm Hg·MET^{-1} (27). Finally, Sabbahi et al. (28) demonstrated racial differences with Black men (42 ± 11 yr) demonstrating an average systolic BP increase of 10.8 ± 5.1 versus 8.2 ± 3.7 mm Hg·MET^{-1} in White men (43 ± 11 yr). Data from healthy (20–42 yr (26)) and athletic (21 ± 2 yr (29)) female participants have reported a systolic BP/MET slope of 5.0 ± 1.1 and 5.1 ± 1.6 mm Hg·MET^{-1}, respectively. One consideration of these data is that numerous studies have relied on estimating maximal oxygen consumption using predictive equations (26,27,29), which, depending on the equation used, can overestimate maximal METs leading to a lower systolic BP/MET slope (30).

Considerations for Sex, Age, and Race

The BP responses to exercise will vary based on sex, age, and race. Sex differences in the systolic BP response to exercise have been documented; absolute systolic BP increases are smaller in females compared with males (20,21,31), whereas systolic BP indexed to workload or METs have been shown to be similar between sexes (29,30) or higher in females (29,31). Peak systolic BPs are higher in older ages. In a sample of >10,000 apparently healthy adults, the 90th percentile for peak systolic BP obtained during a Bruce protocol in women and men was 190 and 210 mm Hg in 20- to 29-year-olds, and 220 and 234 mm Hg in 70- to 79-year-olds (21). The trajectory of increase in peak systolic BP with age also differs by sex. Data from the FRIEND registry showed that males had an increase in peak systolic BP with age until the fifth decade, with no noticeable change between the fifth and seventh decades, whereas females showed a continuous increase in peak systolic BP to the seventh (32). Racial differences in peak/maximal systolic BPs have also been reported (28), whereas other studies demonstrate no difference in submaximal (33) or peak/maximal systolic BP between races (20). It is important to acknowledge that these investigations are comparing adults who identify as Black or White, and therefore, a more comprehensive examination of the effects of race and ethnicity on exercise BPs is warranted.

Overall, this information is intended to highlight that a "normal" BP response to exercise should be interpreted with participant demographics in mind. Aside from sex-specific thresholds for EEBP, there are no recommendations on how to consider age or race when interpreting exercise BPs. In addition, for females, there is no recommendation on whether premenopausal females should be tested in a certain phase of the menstrual cycle. When menstrual cycle was not controlled for, Currie et al. (34) demonstrated that BP responses to a repeated graded exercise tests were unreliable in a small sample of participants. Given the lack of evidence on this topic, our recommendation is to document hormonal contraceptive use or the phase of the natural menstrual cycle that the premenopausal female is in during testing (when possible), and if repeated tests are required, to attempt to test within the same phase of the menstrual cycle or hormonal contraceptive use (e.g., during the same dosage of the active pill for oral contraceptive pill users). At this time, no recommendation can be made on whether the follicular or luteal phase of the menstrual cycle is preferred (35). We recognize that these recommendations may be challenging to adhere to in the clinical setting or during the perimenopause transition when cycles may be inconsistent.

SUBMAXIMAL EXERCISE BP IN THE DETECTION AND MANAGEMENT OF UNCONTROLLED HIGH BP

There is now substantial evidence that outlines the clinical usefulness of assessing exercise BP during submaximal exercise of a low-to-moderate intensity (36). In a meta-analysis of 46,314 adults, a submaximal, but not maximal, EEBP response was associated with cardiovascular events and mortality independent of resting BP and other cardiovascular risk factors (10). These findings are supported by recent data (n = 11,143) indicating that exercise systolic BP values ≥170 mm Hg recorded at stage 2 of a Bruce treadmill protocol were independently associated with a 33% adjusted increased risk of cardiovascular events and mortality (25), whereas exercise BP recorded during submaximal cycling (100 W) displays a linear relationship with the risk of coronary artery disease (37). Much of the increased cardiovascular risk related to EEBP when recorded at a submaximal exercise intensity may be attributed to residual risk associated with the presence of undetected or uncontrolled high BP (and its associated risk factors). For example, in addition to associations found with an EEBP recorded at peak/maximal exercise, an EEBP during submaximal exercise is associated with the future development of hypertension in those with normal office resting BP at baseline (25,38), Moore et al. (39) also recently described (by meta-analysis of cross-sectional studies) a submaximal EEBP to be associated with the presence of many hypertension-related cardiovascular disease risk factors, including arterial structure and function, lipid, metabolic, inflammatory, and kidney function markers. The prevalence of a submaximal EEBP in those with type 2 diabetes, a condition characterized by increased cardiovascular risk related to poor BP control (40), is greater than those without (41), with several studies also indicating submaximal EEBP to be associated with adverse cardiac remodeling (including a heightened prevalence of left ventricular hypertrophy, a telling clinical marker of organ damage related to high BP) (39,42).

It is important to note that most studies that have assessed the relationship between EEBP and hypertension-related cardiovascular risk have assessed BP via office BP alone. However, out-of-office methods (such as home or ambulatory BP) are required to determine BP control and rule out the presence of masked uncontrolled or white coat hypertension. Twenty-four-hour ambulatory BP is the gold standard method to assess BP control because it provides a snapshot of BP load over a longer duration and during activities of daily life (3,4). Submaximal exercise of a light-to-moderate intensity reflects the intensity of daily life ambulatory activities (e.g., walking, light housework) (6,43), and therefore, measurement of BP during submaximal exercise is likely a truer reflection of the daily burden of BP than that of BP measured at rest or indeed during peak/maximal intensity exercise. To this end, Schultz et al. (24) reported exercise systolic BP values ≥150 mm Hg recorded during stages 1 or 2 of a standard Bruce treadmill protocol (light-to-moderate intensity) to be associated with the presence of masked hypertension confirmed by ambulatory BP monitoring. Others have also shown EEBP recorded during submaximal exercise of a low-to-moderate intensity to identify or rule out the presence of masked hypertension (8,44,45). These are important findings because masked hypertension is associated with 1.5 times the risk of cardiovascular disease (compared with those with normal BP) and can remain undetected with office BP measure alone (46). Collectively, this evidence points toward a submaximal EEBP as a useful marker of uncontrolled high BP and related cardiovascular risk that has gone undetected with traditional office BP measurements.

From a methodological and physiological perspective, there are several advantages to the measurement of BP during submaximal exercise of low-to-moderate intensity (moderate defined as approximately 46%–63% maximal oxygen consumption or 12–13 on a 20 point rating of perceived exertion scale (6)). As mentioned, it is comparatively easy to measure (compared with during peak/maximal intensity exercise) due to less noise and movement artifact (14). Of particular importance, the assessment of exercise BP during submaximal exercise allows the influence of cardiorespiratory fitness on the response to be considered. The interplay between cardiorespiratory fitness, exercise BP, and exercise workload was recently described by Schultz et al. (47), in relation to clinical interpretation of an EEBP. Although systolic BP shares a relatively linear relationship with workload (31), those with comparatively lower cardiorespiratory fitness likely have a steeper rise in systolic BP, with EEBP (at peak/maximal intensity) occurring early on during incremental exercise and at a lower relative workload. This BP response may be considered a "pathological" BP response to exercise and is likely indicative of the hypertension-related cardiovascular risk. On the other hand, with higher cardiorespiratory fitness, the rate of increase in systolic BP is likely to be lower, with EEBP at peak/maximal intensity occurring only after a greater duration of exercise and at a higher relative workload. This BP response may be considered a "physiological" BP response and is likely to occur in the absence of cardiovascular dysfunction (48,49). Understanding cardiorespiratory fitness is therefore of vital importance to differentiate a pathological BP response to exercise from one that is merely a physiological consequence of higher cardiorespiratory fitness. If exercise BP is only assessed during peak/maximal exercise intensity, where fixed workloads for BP measurement are not possible, the influence of cardiorespiratory fitness is obscured, precluding correct clinical interpretation of an EEBP (47). External workloads can, however, be fixed during submaximal exercise, and thus, for any given fixed workload during submaximal exercise, the influence of cardiorespiratory fitness on exercise BP will be clear and allow for correct clinical interpretation of EEBP (47).

There are many clinical contexts in which BP is measured during exercise. One of the most frequent is by exercise professionals (such as exercise physiologists) working within the (allied) health care sector. Because aerobic exercise testing is frequently undertaken to assess functional/aerobic capacity in this setting and with BP measurement, an important safety requirement (6,11), such professionals are optimally placed to

- use exercise BP to identify people with uncontrolled high BP;
- if EEBP is recorded, make referral to other health care providers (e.g., general practitioners, primary care physicians) for further assessment of BP;
- provide education about high BP and how to self-manage/monitor the condition (e.g., via home BP measurement); and
- undertake exercise and lifestyle interventions to lower BP and cardiovascular risk.

To help facilitate this process, Schultz and colleagues (50) recently published recommendations for measuring exercise BP within exercise-based professions, outlining a "pathway" for the identification and management of hypertension. The key components of this clinical pathway include recommendations as follows:

- Any person completing an exercise test should have BP measured during exercise to aid in the detection of uncontrolled high BP.
- BP should be measured using best-practice technique during fixed-workload, submaximal exercise of any modality that elicits a moderate intensity.
- The preference is for manual BP measurement, undertaken during exercise (rather than on completion).
- A useful definition of EEBP (based on published evidence indicating associations with cardiovascular disease risk markers) is exercise systolic BP in the range of ≥150–170 mm Hg recorded during submaximal exercise of a moderate intensity.
- If EEBP is recorded (exercise systolic BP ≥170 mm Hg), uncontrolled high BP should be considered and prompt
  - o correspondence with a general practitioner/primary care physician encouraging follow-up testing to ascertain BP status;
  - o guidance for the patient to complete home BP measurement and encouragement to report these findings to their treating physician; and

- ongoing exercise and lifestyle intervention that is targeted to lower high BP and improve overall cardiovascular disease risk.

These recommendations are currently being reviewed for professional and societal endorsement in an Australian exercise physiology context as one avenue to improve BP control rates. However, there is international relevance to this endeavor, with calls for greater involvement of the allied health professions (e.g., physical therapists, exercise physiologists) to measure and manage high BP (51), which are echoed in the Surgeon General's call to improve BP control rates in the United States (52).

MECHANISMS CONTRIBUTING TO AN EEBP

Given the association between EEBP and cardiovascular risk, an understanding of the physiological mechanisms underpinning this response may help to identify potential therapeutic targets. As mentioned previously, BP represents a controlled variable of the cardiovascular system, modified by changes in cardiac output and total peripheral vascular resistance. Unfortunately, few studies have assessed the relative contributions of changes in cardiac output and peripheral vascular resistance during exercise in those with an EEBP. In a cohort of young males, cardiac output index was not different between those with and without an EEBP, whereas the peripheral vascular index tended to be higher in those with an EEBP (53). In agreement, middle-aged females demonstrate higher peripheral vascular resistance at rest and during exercise in those with the highest tertile of exercise BP responses, whereas peak cardiac output index and stroke volume index during exercise were lower (54). These results suggest that an impairment in the ability to lower peripheral vascular resistance during dynamic exercise may underlie the EEBP phenotype. It is important to acknowledge that in both studies reporting absolute differences in peripheral vascular resistance, the relative changes in peripheral vascular resistance during exercise were similar, in contrast to differing exercise BP responses between groups (53,54). This aligns with data from submaximal exercise tests in which the changes in cardiac index and peripheral vascular resistance index were similar in a cohort of middle-aged athletes with and without an EEBP (48). Given the large proportion of cardiac output directed toward active skeletal muscle during maximal exercise (19), and the impact of baseline cardiac output on the contribution of changes in regional peripheral resistance on BP (55), future studies may require regional assessments of vascular resistance (or better yet, vascular conductance) in active muscle beds to better elucidate the respective relative contributions to mediating an EEBP. Furthermore, the balance in contribution of cardiac output and peripheral resistance to EEBP may also differ between individuals based on cardiorespiratory fitness.

The focus on attenuated reductions in peripheral vascular resistance during exercise has led to several investigations into the role of impaired endothelium-dependent vasodilation, elevated arterial stiffness, and/or heightened neurohumoral activation. The strongest evidence supports alterations in vascular function, with studies reporting that an EEBP is associated with attenuated brachial artery flow-mediated vasodilation (56,57), higher carotid-femoral pulse wave velocity (54), lower aortic distensibility and increased stiffness (58), and greater carotid artery intima-media thickness (57). Adults with an EEBP also possess augmented serum levels of asymmetric dimethylarginine, an endogenous inhibitor of nitric oxide synthesis (59). In line with a contributory role of reduced endothelium-dependent vasodilation and reduced nitric oxide bioavailability, those with EEBP demonstrated attenuated forearm blood flow responses to intra-arterial infusions of acetylcholine and nitric oxide synthase antagonist N^G-monomethyl-L-arginine, but not sodium nitroprusside or norepinephrine (56). Furthermore, resting brachial artery flow-mediated dilation has been reported to correlate negatively with exercise systolic BP responses (60,61). One important consideration of potential mechanisms is that those with and without an EEBP can have similar resting BP, highlighting the importance of studying potential mechanisms during exercise. For example, those with an EEBP had similar serum levels of nitrite/nitrate, an estimate of nitric oxide availability, and cyclic GMP, a critical intracellular messenger in mediating smooth muscle relaxation, as controls at rest, but lower cyclic GMP concentrations at peak exercise (56). The potent vasoconstrictor angiotensin II has also been implicated to be involved in mediating an EEBP. In cohorts of middle-aged adults, those with an EEBP had an augmented rise in angiotensin II during peak exercise, without any differences in plasma catecholamines (58,62). In support, 2 wk of treatment with losartan, an angiotensin receptor blocker, did not change resting BP but reduced peak systolic BP and increased exercise duration in patients with diastolic dysfunction and an EEBP (63). In contrast, it has been reported that plasma norepinephrine is higher during exercise in those with an EEBP (64). Whether this reflects increased central sympathetic drive or reduced norepinephrine reuptake is not known. Direct measurements of muscle sympathetic nerve activity at rest using microneurography have found no difference in adults with metabolic syndrome with and without an EEBP (65). Whether muscle sympathetic nerve activity differs during exercise remains unclear.

An attenuated cardiac baroreflex sensitivity has also been reported in those with an EEBP (65,66). However, this measure reflects reflex cardiac parasympathetic modulation of heart rate (67) and baroreflex-mediated changes in exercise BP are driven primarily by changes in total vascular conductance not cardiac output (68). How changes in cardiac baroreflex sensitivity are causally linked to an EEBP requires further clarification. To date, no studies have assessed the role of sympathetic baroreflex sensitivity in those with an EEBP.

EXERCISE AND HYPERTENSION: A PRESCRIPTION UPDATE

The proceedings of our scientific symposium to this point have focused on the acute BP response to submaximal and maximal exercise and the associated physiological mechanisms underpinning an EEBP in individuals likely to develop or who

have hypertension. Here, we will discuss exercise training in terms of the primary and secondary prevention of hypertension, as well as the antihypertensive effects of exercise (both in the short-term following an acute exercise bout, and the chronic adaptations that occur with exercise training). Indeed, habitual exercise/physical activity has been shown to prevent the development of hypertension in an inverse dose response relationship with the volume of physical activity performed, with each 10 MET-hours per week of physical activity in those with normal BP reducing the risk of developing hypertension by 6% (69). Exercise training interventions of varying type, duration, and modality have been shown to reduce BP, on average, 5–8 mm Hg in individuals with hypertension, 2 to 4 mm Hg in those with prehypertension, and 1 to 2 mm Hg in those with normal BP (70). Thus, the magnitude of the BP reduction is a direct function of resting BP, with the greatest BP reductions occurring in those with hypertension, followed by those with prehypertension and normal BP. For these reasons, exercise is recommended worldwide as first-line lifestyle therapy to prevent and treat hypertension (71–75).

An exercise prescription is an individualized physical activity program framed by the frequency (how often?), intensity (how hard?), time (how long?), and type (what kind?) or the FITT principle (6). Based on evidence from a meta-review of systematic reviews and meta-analyses performed by Physical Activity Guidelines for Americans Advisory Committee (74) and the ACSM Pronouncement that followed (70,76), the current ACSM FITT exercise prescription for hypertension is engaging in aerobic or dynamic resistance exercise alone or combined on most, preferably all, days of the week for at least 20 to 30 min·d^{-1} to total at least 90 to 150 min·wk^{-1} of continuous or accumulated exercise of any duration and intensity. Moderate-intensity exercise should be emphasized, although depending on clinical presentation and personal preference, light and vigorous intensity may also be encouraged. Neuromotor and flexibility may be added, although at the time that these ACSM recommendations were published, the evidence was much more compelling for aerobic and dynamic resistance than neuromotor exercise. Of note, these ACSM recommendations no longer place emphasis solely on aerobic exercise and encourage more exercise options that can be done in less time than previous ACSM recommendations.

Since the release of the ACSM exercise prescription recommendations for hypertension, new evidence has emerged on the use of exercise to prevent and treat hypertension. This proceeding now presents highlights from two reviews by Alves et al. (77,78) addressing the long-held notion that medications work better than exercise as antihypertensive therapy (52) and expansion of the guidelines to include more exercise options. Naci and colleagues (79) first compared the antihypertensive effects of exercise and medications on systolic BP among 39,742 healthy adults with normal BP, prehypertension, and hypertension. They found that aerobic and dynamic resistance exercise alone or combined and antihypertensive medications reduced systolic BP by ~9 mm Hg among adults with hypertension. Noone and colleagues (80) then compared the antihypertensive effects of exercise and medications among 32,404 adults with hypertension and found that the evidence was insufficient to make the conclusion that the antihypertensive effects of medications were better than exercise.

Following the work of Naci et al. (79) and Noone et al. (80), Pescatello and colleagues (81) conducted a scoping review among 28,810 adults with hypertension that contained a network meta-analysis of 12 randomized control trials that isolated and directly compared the antihypertensive benefits of exercise and medication combined to exercise or medication alone, and a meta-review of 13 meta-analyses of randomized controlled trials and controlled trials that performed medication moderator analyses. They concluded exercise and medication alone or combined were effective antihypertensive therapy, and when combined, they elicited BP reductions of 6–14 mm Hg greater than medication alone. In addition, exercise alone elicited greater BP reductions than medications alone by 5–9 mm Hg, although the evidence was more compelling for systolic than diastolic BP and alternative (e.g., Qigong, Tai Chi, and Yoga) than traditional types of exercise (i.e., aerobic and dynamic resistance alone or combined). Collectively, these findings show exercise measures up to medication as antihypertensive therapy. In fact, exercise may elicit greater BP reductions than medications alone, especially for systolic BP and alternative types of exercise, and when combined, their antihypertensive effects are greater than medication alone in the treatment of hypertension.

Three meta-analyses, two by Wu et al. (82,83) and one by Hanssen et al. (73), which was a consensus document by the European Association of Preventive Cardiology and the European Society of Cardiology, and a network meta-analysis by Edwards et al. (84), now provide evidence the exercise prescription and hypertension recommendations can be expanded to include not only aerobic and dynamic resistance exercise alone or combined but also neuromotor exercise and isometric resistance exercise to prevent and treat hypertension. Of note, Hanssen and Pescatello (85) and Alves et al. (77,78) contend to recommend isometric resistance exercise as standalone antihypertensive therapy is premature currently due to the small size of this literature that contains conflicting evidence for individuals with hypertension. Rather isometric resistance exercise should be recommended for the primary prevention of hypertension and as secondary prevention only for individuals with hypertension who are not

TABLE 1. The updated exercise prescription for hypertension (based on data from Refs. (70,76)).

FITT Component	Exercise Prescription Description
Frequency	On most, preferably all, days of the week
Intensity	Emphasize moderate, depending on clinical presentation and preference, Light and vigorous intensity may also be encouraged
Time	≥20 to 30 min·d^{-1} to total ≥ 90 to 150+ min·wk^{-1} of continuous or accumulated exercise of any duration
Type	Emphasize aerobic[a] or dynamic resistance[b] or neuromotor[c] exercise alone or combined and include flexibility[d]

[a] Prolonged, rhythmic activities using large muscle groups (e.g., walking, cycling, swimming).
[b] Resistance machines, free weights, resistance bands, and/or functional body weight exercise.
[c] Exercise involving motor skills and/or functional body weight and flexibility exercise such as yoga, pilates, and tai chi.
[d] Static, dynamic, and/or proprioceptive neuromuscular facilitation.
FITT, Frequency, Intensity, Time, and Type of Exercise.

able or do not adhere to aerobic and dynamic resistance exercise. See Table 1 for the expanded exercise prescription recommendations based on this new evidence for individuals with hypertension that now include neuromotor exercise as an option, and consideration of isometric resistance exercise in instances where individuals with physical limitations cannot perform aerobic and/or dynamic resistance exercise.

SUMMARY: FUTURE DIRECTIONS

Since the establishment of exercise testing guidelines in the 1970s (86,87), the measurement and interpretation of exercise BPs have continued to evolve. Like the clinical practice guidelines for the measurement of BP during rest, exercise testing guidelines (6,7) would benefit from expanding upon their current recommendations. Some areas worthy of additional investigation and discussion include the use of manual versus automated measurements, inter-arm differences in BP during exercise, and the effect of menstrual cycle phase on exercise BPs and EEBP. As we have summarized, there is a growing body of evidence that now highlights the clinical usefulness of EEBP (measured during submaximal exercise) for the identification of uncontrolled high BP. Despite this, clinical guidelines have not been updated to affirm the independent (hypertension related) cardiovascular disease risk associated with EEBP. We therefore support an updated review of all contemporary evidence surrounding EEBP during submaximal exercise for consideration for inclusion within exercise testing and hypertension management guidelines. To complement, future research should aim to fill outstanding evidence gaps (including establishing a universal definition/s of EEBP recorded during submaximal exercise) and provide practical guidance and implementation strategies for health professionals to measure BP during exercise. This guidance should include education surrounding interpretation of an EEBP in relation to the optimization of care for people with hypertension. The mechanisms responsible for the EEBP phenotype remain to be fully resolved and likely involve several interacting vascular and neurohumoral pathways. Future studies are necessary to better examine the role of the sympathetic nervous system in mediating an EEBP. For example, a centrally acting angiotensin-converting enzyme inhibitor can attenuate muscle sympathetic nerve activity and BP responses during submaximal cycling exercise compared with a peripherally acting angiotensin-converting enzyme inhibitor. Whether pathological resetting of the sympathetic baroreflex occurs in those with an EEBP is not known. Finally, research on the types and intensities of physical activity/exercise needed to prevent and treat hypertension is in continual pursuit of establishing the optimal FITT prescription. Additional research on isometric exercise (73,82,83) and high-intensity interval training (88,89) is warranted given the promising but limited and at times conflicting evidence to date. In the end, continued efforts to improve the identification and management of hypertension are a necessary and important field of study.

P. J. M. is supported by a Natural Sciences and Engineering Research Council (NSERC) Discovery Grant, the Canadian Foundation for Innovation, and an Ontario Early Researcher Award. M. G. S. was supported by a National Heart Foundation of Australia Future Leader Fellowship (Ref.: 102553). L. S. P. is founder and sole proprietor of P3-EX LLC software that prescribes exercise. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. The results of the present study do not constitute endorsement by the American College of Sports Medicine. Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

REFERENCES

1. World Health Organization. *Global Report on Hypertension: The Race Against a Silent Killer*. Geneva: World Health Organization; 2023. Available from: https://www.who.int/publications/i/item/9789240081062.
2. Centers for Disease Control and Prevention. Hypertension Cascade: Hypertension Prevalence, Treatment and Control Estimates Among U.S. Adults Aged 18 Years and Older Applying the Criteria from the American College of Cardiology and American Heart Association's 2017 Hypertension Guideline—NHANES 2017–2020. Atlanta, GA: May 12, 2023.
3. Mancia G, Kreutz R, Brunström M, et al. 2023 ESH guidelines for the management of arterial hypertension The task force for the management of arterial hypertension of the European Society of Hypertension: endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). *J Hypertens*. 2023;41(12):1874–2071.
4. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: executive summary: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. *Hypertension*. 2018;71(6):1269–324.
5. Julius S, Nesbitt SD, Egan BM, et al. Feasibility of treating prehypertension with an angiotensin-receptor blocker. *N Engl J Med*. 2006;354(16):1685–97.
6. Ligouri G, American College of Sports Medicine. *ACSM's Guidelines for Exercise Testing and Prescription*. 11th ed. Philadelphia: Wolters Kluwer; 2022.
7. Fletcher GF, Ades PA, Kligfield P, et al. Exercise standards for testing and training: a scientific statement from the American Heart Association. *Circulation*. 2013;128(8):873–934.
8. Schultz MG, Hare JL, Marwick TH, Stowasser M, Sharman JE. Masked hypertension is "unmasked" by low-intensity exercise blood pressure. *Blood Press*. 2011;20(5):284–9.
9. Keller K, Stelzer K, Ostad MA, Post F. Impact of exaggerated blood pressure response in normotensive individuals on future hypertension and prognosis: systematic review according to PRISMA guideline. *Adv Med Sci*. 2017;62(2):317–29.
10. Schultz MG, Otahal P, Cleland VJ, Blizzard L, Marwick TH, Sharman JE. Exercise-induced hypertension, cardiovascular events, and mortality in patients undergoing exercise stress testing: a systematic review and meta-analysis. *Am J Hypertens*. 2013;26(3):357–66.
11. Sharman JE, LaGerche A. Exercise blood pressure: clinical relevance and correct measurement. *J Hum Hypertens*. 2015;29(6):351–8.
12. Koletsos N, Dipla K, Triantafyllou A, et al. A brief submaximal isometric exercise test 'unmasks' systolic and diastolic masked hypertension. *J Hypertens*. 2019;37(4):710–9.
13. Holmstrup ME, Clarke MM, Conner CR, Jensen BT. An acute bout of aerobic exercise alters interarm systolic blood pressure difference. *Blood Press Monit*. 2017;22(2):68–71.
14. Lightfoot JT, Tuller B, Williams DF. Ambient noise interferes with auscultatory blood pressure measurement during exercise. *Med Sci Sports Exerc*. 1996;28(4):502–8.

15. Cameron JD, Stevenson I, Reed E, McGrath BP, Dart AM, Kingwell BA. Accuracy of automated auscultatory blood pressure measurement during supine exercise and treadmill stress electrocardiogram-testing. *Blood Press Monit*. 2004;9(5):269–75.
16. Moore MN, Picone DS, Callisaya ML, Srikanth V, Sharman JE, Schultz MG. Comparison of manual and automated auscultatory blood pressure during graded exercise among people with type 2 diabetes. *J Clin Hypertens (Greenwich)*. 2019;21(12):1872–8.
17. Fox SM 3rd, Naughton JP, Haskell WL. Physical activity and the prevention of coronary heart disease. *Ann Clin Res*. 1971;3(6):404–32.
18. Pollock ML, Foster C, Schmidt D, Hellman C, Linnerud AC, Ward A. Comparative analysis of physiologic responses to three different maximal graded exercise test protocols in healthy women. *Am Heart J*. 1982;103(3):363–73.
19. Joyner MJ, Casey DP. Regulation of increased blood flow (hyperemia) to muscles during exercise: a hierarchy of competing physiological needs. *Physiol Rev*. 2015;95(2):549–601.
20. Manolio TA, Burke GL, Savage PJ, Sidney S, Gardin JM, Oberman A. Exercise blood pressure response and 5-year risk of elevated blood pressure in a cohort of young adults: the CARDIA study. *Am J Hypertens*. 1994;7(3):234–41.
21. Daida H, Allison TG, Squires RW, Miller TD, Gau GT. Peak exercise blood pressure stratified by age and gender in apparently healthy subjects. *Mayo Clin Proc*. 1996;71(5):445–52.
22. Holmqvist L, Mortensen L, Kanckos C, Ljungman C, Mehlig K, Manhem K. Exercise blood pressure and the risk of future hypertension. *J Hum Hypertens*. 2012;26(12):691–5.
23. Carlen A, Lindow T, Cauwenberghs N, et al. Exercise systolic blood pressure response during cycle ergometry is associated with future hypertension in normotensive individuals. *Eur J Prev Cardiol*. 2024;31(9):1072–9.
24. Schultz MG, Picone DS, Nikolic SB, Williams AD, Sharman JE. Exaggerated blood pressure response to early stages of exercise stress testing and presence of hypertension. *J Sci Med Sport*. 2016; 19(12):1039–42.
25. Zafrir B, Aker A, Asaf Y, Saliba W. Blood pressure response during treadmill exercise testing and the risk for future cardiovascular events and new-onset hypertension. *J Hypertens*. 2022;40(1):143–52.
26. Currie KD, Floras JS, La Gerche A, Goodman JM. Exercise blood pressure guidelines: time to re-evaluate what is normal and exaggerated? *Sports Med*. 2018;48(8):1763–71.
27. Hedman K, Cauwenberghs N, Christle JW, Kuznetsova T, Haddad F, Myers J. Workload-indexed blood pressure response is superior to peak systolic blood pressure in predicting all-cause mortality. *Eur J Prev Cardiol*. 2020;27(9):978–87.
28. Sabbahi A, Arena R, Kaminsky LA, et al. Characterization of the blood pressure response during cycle ergometer cardiopulmonary exercise testing in black and white men: data from the Fitness Registry and Importance of Exercise: A National Database (FRIEND). *J Hum Hypertens*. 2021;35(8):685–95.
29. Bauer P, Kraushaar L, Dorr O, Nef H, Hamm CW, Most A. Sex differences in workload-indexed blood pressure response and vascular function among professional athletes and their utility for clinical exercise testing. *Eur J Appl Physiol*. 2021;121(7):1859–69.
30. Rycroft AD, Hilton SE, Khangura PK, Bommarito JC, Nardone M, Millar PJ. Workload-indexed blood pressure response to exercise: considerations for future studies estimating maximal oxygen uptake. *Eur J Prev Cardiol*. 2024;31(12):1553–5.
31. Hedman K, Lindow T, Elmberg V, Brudin L, Ekstrom M. Age- and gender-specific upper limits and reference equations for workload-indexed systolic blood pressure response during bicycle ergometry. *Eur J Prev Cardiol*. 2021;28(12):1360–9.
32. Sabbahi A, Arena R, Kaminsky LA, Myers J, Phillips SA. Peak blood pressure responses during maximum cardiopulmonary exercise testing: reference standards from FRIEND (Fitness Registry and the Importance of Exercise: A National Database). *Hypertension*. 2018;71(2):229–36.
33. Ekelund LG, Suchindran CM, Karon JM, McMahon RP, Tyroler HA. Black–White differences in exercise blood pressure. The Lipid Research Clinics Program Prevalence Study. *Circulation*. 1990;81(5):1568–74.
34. Currie KD, Soave KM, Slade JM. Reliability of blood pressure responses used to define an exaggerated blood pressure response to exercise in young healthy adults. *J Hum Hypertens*. 2023;37(1):56–61.
35. D'Souza AC, Wageh M, Williams JS, et al. Menstrual cycle hormones and oral contraceptives: a multimethod systems physiology-based review of their impact on key aspects of female physiology. *J Appl Physiol (1985)*. 2023;135(6):1284–99.
36. Schultz MG, La Gerche A, Sharman JE. Blood pressure response to exercise and cardiovascular disease. *Curr Hypertens Rep*. 2017;19(11):89.
37. Mariampillai JE, Engeseth K, Kjeldsen SE, et al. Exercise systolic blood pressure at moderate workload predicts cardiovascular disease and mortality through 35 years of follow-up in healthy, middle-aged men. *Blood Press*. 2017;26(4):229–36.
38. Schultz MG, Otahal P, Picone DS, Sharman JE. Clinical relevance of exaggerated exercise blood pressure. *J Am Coll Cardiol*. 2015; 66(16):1843–5.
39. Moore MN, Climie RE, Otahal P, Schultz MG. Exercise blood pressure and cardiovascular disease risk: a systematic review and meta-analysis of cross-sectional studies. *J Hypertens*. 2021;39(12):2395–402.
40. Franklin SS, Thijs L, Li Y, et al. Masked hypertension in diabetes mellitus: treatment implications for clinical practice. *Hypertension*. 2013;61(5):964–71.
41. Schultz MG, Otahal P, Kovacevic A-M, et al. Type-2 diabetes and the clinical importance of exaggerated exercise blood pressure. *Hypertension*. 2022;79(10):2346–54.
42. Kokkinos P, Pittaras A, Narayan P, Faselis C, Singh S, Manolis A. Exercise capacity and blood pressure associations with left ventricular mass in prehypertensive individuals. *Hypertension*. 2007;49(1):55–61.
43. Norton K, Norton L, Sadgrove D. Position statement on physical activity and exercise intensity terminology. *J Sci Med Sport*. 2010; 13(5):496–502.
44. Kayrak M, Bacaksiz A, Vatankulu MA, et al. Exaggerated blood pressure response to exercise—a new portent of masked hypertension. *Clin Exp Hypertens*. 2010;32(8):560–8.
45. Huang Z, Sharman JE, Fonseca R, et al. Masked hypertension and submaximal exercise blood pressure among adolescents from the Avon Longitudinal Study of Parents and Children (ALSPAC). *Scand J Med Sci Sports*. 2020;30(1):25–30.
46. Stergiou GS, Asayama K, Thijs L, et al. Prognosis of white-coat and masked hypertension: International Database of HOme blood pressure in relation to Cardiovascular Outcome. *Hypertension*. 2014; 63(4):675–82.
47. Schultz MG, La Gerche A, Sharman JE. Cardiorespiratory fitness, workload, and the blood pressure response to exercise testing. *Exerc Sport Sci Rev*. 2022;50(1):25–30.
48. Currie KD, Sasson Z, Goodman JM. Vascular–ventricular coupling during exercise is not affected by exaggerated blood pressures in endurance-trained athletes. *J Appl Physiol (1985)*. 2019; 127(3):753–9.
49. Currie KD, Sless RT, Notarius CF, Thomas SG, Goodman JM. Absence of resting cardiovascular dysfunction in middle-aged endurance-trained athletes with exaggerated exercise blood pressure responses. *J Hypertens*. 2017;35(8):1586–93.
50. Schultz MG, Currie KD, Hedman K, et al. The identification and management of high blood pressure using exercise blood pressure: current evidence and practical guidance. *Int J Environ Res Public Health*. 2022;19(5):2819.
51. Severin R, Sabbahi A, Albarrati A, Phillips SA, Arena S. Blood pressure screening by outpatient physical therapists: a call to action and clinical recommendations. *Phys Ther*. 2020;100(6):1008–19.
52. U.S. Department of Health and Human Services. *The Surgeon General's Call to Action to Control Hypertension*. Washington, DC: U. S. Department of Health and Human Services, Office of the Surgeon General; 2020.

53. Wilson MF, Sung BH, Pincomb GA, Lovallo WR. Exaggerated pressure response to exercise in men at risk for systemic hypertension. *Am J Cardiol*. 1990;66(7):731–6.
54. Sarma S, Howden E, Carrick-Ranson G, et al. Elevated exercise blood pressure in middle-aged women is associated with altered left ventricular and vascular stiffness. *J Appl Physiol (1985)*. 2020;128(5):1123–9.
55. O'Leary DS. Regional vascular resistance vs. conductance: which index for baroreflex responses? *Am J Physiol*. 1991;260(2 Pt 2):H632–7.
56. Chang HJ, Chung JH, Choi BJ, et al. Endothelial dysfunction and alteration of nitric oxide/cyclic GMP pathway in patients with exercise-induced hypertension. *Yonsei Med J*. 2003;44(6):1014–20.
57. Baycan OF, Çelik FB, Guvenç TS, et al. Coronary flow velocity reserve is reduced in patients with an exaggerated blood pressure response to exercise. *Hypertens Res*. 2022;45(10):1653–63.
58. Tzemos N, Lim PO, Mackenzie IS, MacDonald TM. Exaggerated exercise blood pressure response and future cardiovascular disease. *J Clin Hypertens (Greenwich)*. 2015;17(11):837–44.
59. Kayrak M, Bacaksiz A, Vatankulu MA, et al. Association between exaggerated blood pressure response to exercise and serum asymmetric dimethylarginine levels. *Circ J*. 2010;74(6):1135–41.
60. Thanassoulis G, Lyass A, Benjamin EJ, et al. Relations of exercise blood pressure response to cardiovascular risk factors and vascular function in the Framingham Heart Study. *Circulation*. 2012;125(23):2836–43.
61. Stewart KJ, Sung J, Silber HA, et al. Exaggerated exercise blood pressure is related to impaired endothelial vasodilator function. *Am J Hypertens*. 2004;17(4):314–20.
62. Shim CY, Ha JW, Park S, et al. Exaggerated blood pressure response to exercise is associated with augmented rise of angiotensin II during exercise. *J Am Coll Cardiol*. 2008;52(4):287–92.
63. Warner JG Jr., Metzger DC, Kitzman DW, Wesley DJ, Little WC. Losartan improves exercise tolerance in patients with diastolic dysfunction and a hypertensive response to exercise. *J Am Coll Cardiol*. 1999;33(6):1567–72.
64. Miyai N, Arita M, Morioka I, Takeda S, Miyashita K. Ambulatory blood pressure, sympathetic activity, and left ventricular structure and function in middle-aged normotensive men with exaggerated blood pressure response to exercise. *Med Sci Monit*. 2005;11(10):CR478–84.
65. Dutra-Marques AC, Rodrigues S, Cepeda FX, et al. Exaggerated exercise blood pressure as a marker of baroreflex dysfunction in normotensive metabolic syndrome patients. *Front Neurosci*. 2021;15:680195.
66. Sharman JE, Boutouyrie P, Perier MC, et al. Impaired baroreflex sensitivity, carotid stiffness, and exaggerated exercise blood pressure: a community-based analysis from the Paris Prospective Study III. *Eur Heart J*. 2018;39(7):599–606.
67. La Rovere MT, Pinna GD, Raczak G. Baroreflex sensitivity: measurement and clinical implications. *Ann Noninvasive Electrocardiol*. 2008;13(2):191–207.
68. Ogoh S, Fadel PJ, Nissen P, et al. Baroreflex-mediated changes in cardiac output and vascular conductance in response to alterations in carotid sinus pressure during exercise in humans. *J Physiol*. 2003;550(Pt 1):317–24.
69. Liu X, Zhang D, Liu Y, et al. Dose-response association between physical activity and incident hypertension: a systematic review and meta-analysis of cohort studies. *Hypertension*. 2017;69(5):813–20.
70. Pescatello LS, Buchner DM, Jakicic JM, et al. Physical activity to prevent and treat hypertension: a systematic review. *Med Sci Sports Exerc*. 2019;51(6):1314–23.
71. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH guidelines for the management of arterial hypertension. *Eur Heart J*. 2018;39(33):3021–104.
72. Martin SS, Aday AW, Almarzooq ZI, et al. 2024 Heart disease and stroke statistics: a report of US and global data from the American Heart Association. *Circulation*. 2024;149(8):e347–913.
73. Hanssen H, Boardman H, Deiseroth A, et al. Personalized exercise prescription in the prevention and treatment of arterial hypertension: a consensus document from the European Association of Preventive Cardiology (EAPC) and the ESC Council on Hypertension. *Eur J Prev Cardiol*. 2022;29(1):205–15.
74. U.S. Department of Health and Human Services. *Physical Activity Guidelines for Americans*. 2nd ed. Washington (DC): U.S. Department of Health and Human Services; 2018.
75. Charchar FJ, Prestes PR, Mills C, et al. Lifestyle management of hypertension: International Society of Hypertension position paper endorsed by the World Hypertension League and European Society of Hypertension. *J Hypertens*. 2024;42(1):23–49.
76. Pescatello LS, ACoS Medicine. What's new in the ACSM pronouncement on exercise and hypertension? In: *American College of Sports Medicine Blog*. Indianapolis (IN): American College of Sports Medicine; 2019.
77. Alves AJ, Wu Y, Lopes S, Ribeiro F, Pescatello LS. Exercise to treat hypertension: late breaking news on exercise prescriptions that FITT. *Curr Sports Med Rep*. 2022;21(8):280–8.
78. Alves AJ, Wu Y, Lopes S, Ribeiro F, Pescatello LS. Exercise for hypertension: new recommendations strategies for blood pressure control. *J Clin Exerc Physiol*. 2023;12(4):120–8.
79. Naci H, Salcher-Konrad M, Dias S, et al. How does exercise treatment compare with antihypertensive medications? A network meta-analysis of 391 randomised controlled trials assessing exercise and medication effects on systolic blood pressure. *Br J Sports Med*. 2019;53(14):859–69.
80. Noone C, Leahy J, Morrissey EC, et al. Comparative efficacy of exercise and anti-hypertensive pharmacological interventions in reducing blood pressure in people with hypertension: a network meta-analysis. *Eur J Prev Cardiol*. 2020;27(3):247–55.
81. Pescatello LS, Wu Y, Gao S, Livingston J, Sheppard BB, Chen M-H. Do the combined blood pressure effects of exercise and antihypertensive medications add up to the sum of their parts? A systematic meta-review. *BMJ Open Sport Exerc Med*. 2021;7(1):e000895.
82. Wu Y, Johnson BT, Acabchuk RL, et al. Yoga as antihypertensive lifestyle therapy: a systematic review and meta-analysis. *Mayo Clin Proc*. 2019;94(3):432–46.
83. Wu Y, Johnson BT, Chen S, Chen Y, Livingston J, Pescatello LS. Tai Ji Quan as antihypertensive lifestyle therapy: a systematic review and meta-analysis. *J Sport Health Sci*. 2021;10(2):211–21.
84. Edwards JJ, Deenmamode AHP, Griffiths M, et al. Exercise training and resting blood pressure: a large-scale pairwise and network meta-analysis of randomised controlled trials. *Br J Sports Med*. 2023;57(20):1317–26.
85. Hanssen H, Pescatello LS. Is isometric exercise training the best FIT for exercise prescription in the prevention and treatment of arterial hypertension? *Br J Sports Med*. 2024;58(4):231–2.
86. *ACSM's Guidelines for Graded Exercise Testing and Exercise Prescription*. Philadelphia: Lea & Febiger; 1975.
87. American Heart Association. Committee on Exercise. *Exercise Testing and Training of Apparently Healthy Individuals: A Handbook for Physicians*. The Association: Dallas; 1972.
88. Costa EC, Hay JL, Kehler DS, et al. Effects of high-intensity interval training versus moderate-intensity continuous training on blood pressure in adults with pre- to established hypertension: a systematic review and meta-analysis of randomized trials. *Sports Med*. 2018;48(9):2127–42.
89. Leal JM, Galliano LM, Del Vecchio FB. Effectiveness of high-intensity interval training versus moderate-intensity continuous training in hypertensive patients: a systematic review and meta-analysis. *Curr Hypertens Rep*. 2020;22(3):26.