

REVIEW
Stress, Exercise and Cardiovascular Disease

Prognostic value of exercise blood pressure: role of fitness and exercise training

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Abstract

Abnormal blood pressure (BP) responses to exercise, both hypertensive and hypotensive, are strong independent predictors of cardiovascular morbidity and mortality across populations. Higher cardiorespiratory fitness is associated with lower submaximal exercise BP across populations. Better vascular function and modulation of the autonomic nervous system likely drive this relation. Despite higher absolute BP values, athletes demonstrate lower BP responses relative to metabolic demand. These observations highlight a critical limitation of using absolute thresholds and support indexing exercise BP to metabolic or external workload for improved risk stratification. Furthermore, submaximal exercise BP is a superior target for risk assessment and intervention, as it provides unique prognostic value, predicting adverse events independent of resting BP and other risk factors, and consistently improves with exercise training, unlike the inconsistent response of maximal exercise BP. This review synthesizes the evidence on the prognostic value of exercise BP, its physiological relation with cardiorespiratory fitness, and its response to traditional and emerging exercise interventions. Ultimately, we propose a conceptual model for clinical consideration, designed to guide the integration of exercise BP assessment into practice as a modifiable cardiovascular disease risk factor.

blood pressure; cardiorespiratory fitness; exercise pressor reflex; exercise training; hypertension

INTRODUCTION

The influence of nonmodifiable factors like genetics, age, biological sex, and race/ethnicity on the exercise pressor reflex (EPR) is well documented (1–6). Understanding how modifiable factors affect exercise blood pressure (BP) is critical, given its physiological and clinical importance. In 1989, individuals with exaggerated exercise BP were encouraged to perform aerobic exercise training, lose weight, and restrict salt and alcohol intake before participating in sports (7). However, there are no recent review articles addressing this topic. Therefore, we critically examined evidence regarding whether cardiorespiratory fitness (CRF) and different exercise training approaches affect exercise BP among adults. We acknowledge several other crucial modifiable health factors, such as body weight status and diet [e.g., American Heart Association Life's Essential 8 framework (8)]. However, space limitations preclude discussion of such items. This review synthesizes evidence indicating that submaximal exercise BP is a prognostic target uniquely modifiable by exercise training and the underlying mechanisms. We therefore propose that submaximal BP represents a primary clinical target, one that provides unique prognostic value independent of resting BP (Fig. 1).

THE EXERCISE PRESSOR REFLEX

During physical exercise, active skeletal muscle requires increased blood flow to support oxygen delivery and clear metabolic by-products. To meet blood flow demands, the body increases BP to increase perfusion pressure to target organs like skeletal muscle and cardiac tissue. This compensatory increase in blood flow and, ultimately, perfusion is the EPR. The afferent arm of the EPR includes feedback from thinly myelinated group III nerve fibers sensitive to mechanical distortion and unmyelinated group IV nerve fibers sensitive to metabolite (e.g., $[H^+]$) accumulation in working skeletal muscle (9–36). Other important contributors to exercise BP include effort-dependent feedforward central command from the motor cortex (21, 29, 37–56), arterial and cardiopulmonary baroreflexes (22, 37, 57–78), and chemoreflexes (79–83). After neural integration of these inputs (84–86), the brain increases efferent sympathetic outflow and decreases parasympathetic outflow to increase heart rate, cardiac contractility, cardiac output, and vascular resistance. This exercise BP response is intensity dependent, influenced by active muscle mass, absolute workload, and/or relative workload, thereby driving blood flow to active muscle (24, 27, 34, 51, 87–118). Regional differences in



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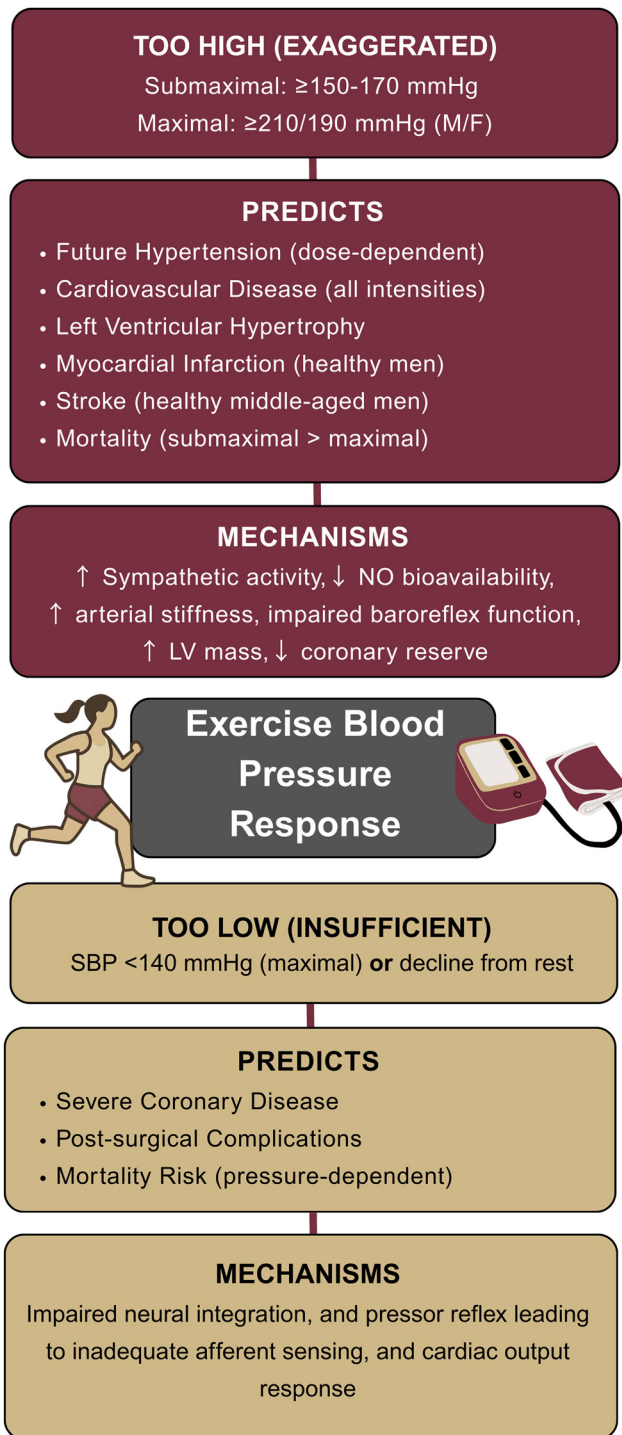


Figure 1. Prognostic value of exercise blood pressure. LV, left ventricular. NO, nitric oxide; SBP, systolic blood pressure.

sympathetic outflow (119, 120) and vasodilatory factors in active muscle (i.e., functional sympatholysis) influence local vasoconstriction (121–130), creating a balance that assists with oxygen extraction while maintaining BP (105). For an extended discussion on EPR mechanisms, we direct the reader to other reviews (5, 6, 14, 22, 35, 71, 105, 131–135).

Exercise BP is tightly regulated: experimentally reducing cardiac output or altering vascular resistance triggers

compensatory adjustments that maintain BP (136, 137). The clinical relevance of exaggerated exercise BP varies based on exercise modality (e.g., dynamic vs. isometric), intensity (e.g., submaximal vs. maximal), and the predicted outcome (e.g., hypertension vs. all-cause mortality). However, the clinical relevance of insufficient BP during exercise is much less appreciated. This discussion explores the prognostic relevance of abnormal exercise BP, encompassing both hypotensive and hypertensive responses.

INSUFFICIENT BLOOD PRESSURE DURING EXERCISE

Definition and General Mechanisms of Insufficient Blood Pressure during Exercise

A systolic BP (SBP) below 140 mmHg is considered inappropriately low (i.e., insufficient) during maximal exercise (138). Declines in SBP from rest to exercise (i.e., exercise-induced hypotension) are an absolute indication for stopping exercise (138). Insufficient increases or declines in BP during exercise are uncommon among adults without disease (139) but are observed among patients with coronary or noncoronary heart disease (139, 140). A hypotensive response to exercise has been linked to postsurgical cardiac complications (141), ischemia, and abnormal cardiac wall motion, suggesting it as a marker for severe coronary artery disease (142). Mechanistically, inadequate afferent sensing, integration, and/or effector responses (e.g., increases in cardiac output) likely underpin the absent pressor response needed to increase perfusion to active skeletal muscle.

Prognostic Value of Insufficient Blood Pressure during Exercise

A lower or insufficient SBP during submaximal or peak exercise is associated with a higher future mortality risk among people free from cardiovascular disease (143, 144), a general population of men (145), and United States veterans (146). This risk extends to other populations including patients after acute myocardial infarction (MI) (147–152), patients with coronary heart disease selected for cardiac catheterization (153), men referred to cardiac rehabilitation (154), patients who completed cardiac rehabilitation (155), patients surviving acute coronary syndrome (156), patients with hypertrophic cardiomyopathy (157), patients hospitalized with a suspected or confirmed acute ischemic event (158), and patients with heart failure (53, 159–161). The magnitude of risk varies by population, with post-MI patients showing particularly strong associations [hazard ratio (HR): 5.1 for cardiovascular death] (153) and heart failure patients demonstrating an 11% mortality reduction per 5-mmHg increase in peak exercise SBP (161). These relations appear pressure dependent, such that the lowest peak exercise SBP is associated with the highest risk regardless of exercise mode or clinical condition (162). However, cardiorespiratory fitness substantially modifies this risk: in unfit individuals, low SBP reserve (≤ 52 mmHg) confers nearly twofold-increased mortality (HR: 1.97), whereas fit individuals show no significant association (HR: 1.06) (146). Medical therapy influences but does not eliminate prognostic value, as peak SBP remains predictive in beta-blocker-treated patients (144,

161). The abnormal response occurring at moderate versus maximal intensity also affects risk magnitude (HRs: 2.59 vs. 1.80) (162), likely reflecting the severity of the underlying pathophysiology.

■ EXAGGERATED BLOOD PRESSURE DURING EXERCISE

Definition and General Mechanisms of Exaggerated Blood Pressure during Exercise

Various thresholds have been proposed for exaggerated BP responses during exercise (7, 138, 163, 164). Isolated systolic exercise hypertension is characterized by normal resting BP and BP that exceeds 200/90 mmHg during exercise at any intensity (165–167). The American Heart Association defines an exaggerated SBP response as SBP > 210 mmHg (men) or >190 mmHg (women), coupled with a diastolic BP (DBP) increase of >10 mmHg above resting or an absolute DBP of ≥ 90 mmHg at any intensity (168). These values coincide with the ≥ 90 th percentile among healthy adults (169, 170). Common cutoffs for exaggerated BP during maximal exercise include $\geq 210/105$ mmHg (men), $\geq 190/105$ mmHg (women), and $\geq 220/110$ mmHg (athletes) (168). For submaximal cycling, thresholds vary by age: 200/100 mmHg if <50 yr and 215/105 mmHg if ≥ 50 yr (171, 172). However, this understanding is complicated by fundamental methodological challenges and mixed evidence on the reproducibility of exercise BP. Obtaining accurate measurements during dynamic exercise can be difficult, as movement and noise artifacts can significantly disrupt readings, particularly for standard oscillometric devices. This limitation drives poor to moderate reproducibility (173, 174) and, combined with inconsistent terminology and thresholds, reduces clinical utility (175, 176), although evidence-based practical guidance does exist (177–179). For normative exercise BP, readers are directed to other sources (163, 180–182). Nonetheless, SBP/metabolic equivalent (MET) and SBP/workload ratios have gained interest because they account for exercise capacity and intensity (183–188). It is crucial to distinguish between the SBP/MET slope [the rate of BP rise relative to metabolic equivalents (i.e., metabolic demand), calculated as $\Delta\text{SBP}/\Delta\text{METs}$] and the SBP/workload ratio (typically peak SBP divided by peak watts). The SBP/MET slope indexes BP to the relative oxygen uptake rate ($\dot{V}\text{O}_2$), which inherently normalizes for total body mass and abolishes sex differences often seen with absolute workload measures (187, 189). In contrast, SBP/watt ratios remain markedly influenced by absolute power output (189).

This contextualization is essential for higher exercise BP among athletes (175, 183, 188), between sexes (184, 187), and in first responders (190), which does not increase cardiovascular disease (CVD) risk in the context of higher workloads attained (191). However, methodological precision is critical; calculating slopes using estimated METs rather than directly measured $\dot{V}\text{O}_2$ significantly reduces sensitivity for identifying high-risk phenotypes (192). Consequently, generating cardiorespiratory fitness (CRF)-indexed BP normative data is crucial (193, 194).

Compelling evidence for this approach comes from recent investigations in middle-aged adults with left ventricular

hypertrophy (LVH). These studies demonstrated that despite achieving identical absolute maximal oxygen uptake rate ($\dot{V}\text{O}_{2\text{max}}$) values, adults with LVH exhibited higher maximal exercise SBP but showed no significant differences in SBP/ $\dot{V}\text{O}_2$ slopes compared with healthy control subjects (195). These findings suggest that apparent exaggerated BP responses in high-risk populations may reflect baseline hemodynamic alterations rather than pathological exercise pressor reflex sensitivity, thereby underscoring the need for workload-contextualized exercise BP assessment.

Mechanisms of exaggerated response.

As illustrated in Fig. 2, exaggerated exercise BP is driven by a shift toward sympathetic dominance. This phenotype is initiated by afferent sensitization, where ischemia and metabolite accumulation amplify the “ischemia loop” via specific metaboreceptors (e.g., TRPV1, ASIC3), overriding normal baroreflex buffering (76, 196).

This neural drive is compounded by vascular and humoral dysregulation. Key drivers include elevated renin-angiotensin activity and endothelial dysfunction, characterized by the accumulation of asymmetric dimethylarginine (ADMA), a potent nitric oxide synthase inhibitor derived from protein turnover (197). Together with structural maladaptations like arterial stiffness, these mechanisms create a feedforward cycle of vasoconstriction and cardiac strain (Fig. 2).

Structural maladaptations include arterial stiffness (185, 198), left ventricular hypertrophy (often independent of resting BP) (199, 200), left atrial enlargement (201), reduced global longitudinal strain (202), and compromised coronary flow reserve (203). Acutely, runners with exercise-induced hypertension show elevated cardiac injury biomarkers post exercise (204).

Prognostic Value of Exaggerated Blood Pressure during Exercise

Exaggerated BP responses during submaximal or maximal exercise contribute to target organ damage and are prognostic for future cardiovascular morbidity and cardiovascular/all-cause mortality risk (141, 167, 176, 205–211). However, some evidence contradicts this association for healthy adults (212). For instance, the rate pressure product (heart rate $\times$ SBP), an index of myocardial oxygen uptake rate, underscores the direct relation between SBP and higher cardiac work (i.e., stress) (213, 214). That said, it is critical to remember the context of the BP responses for the individual and workload (215).

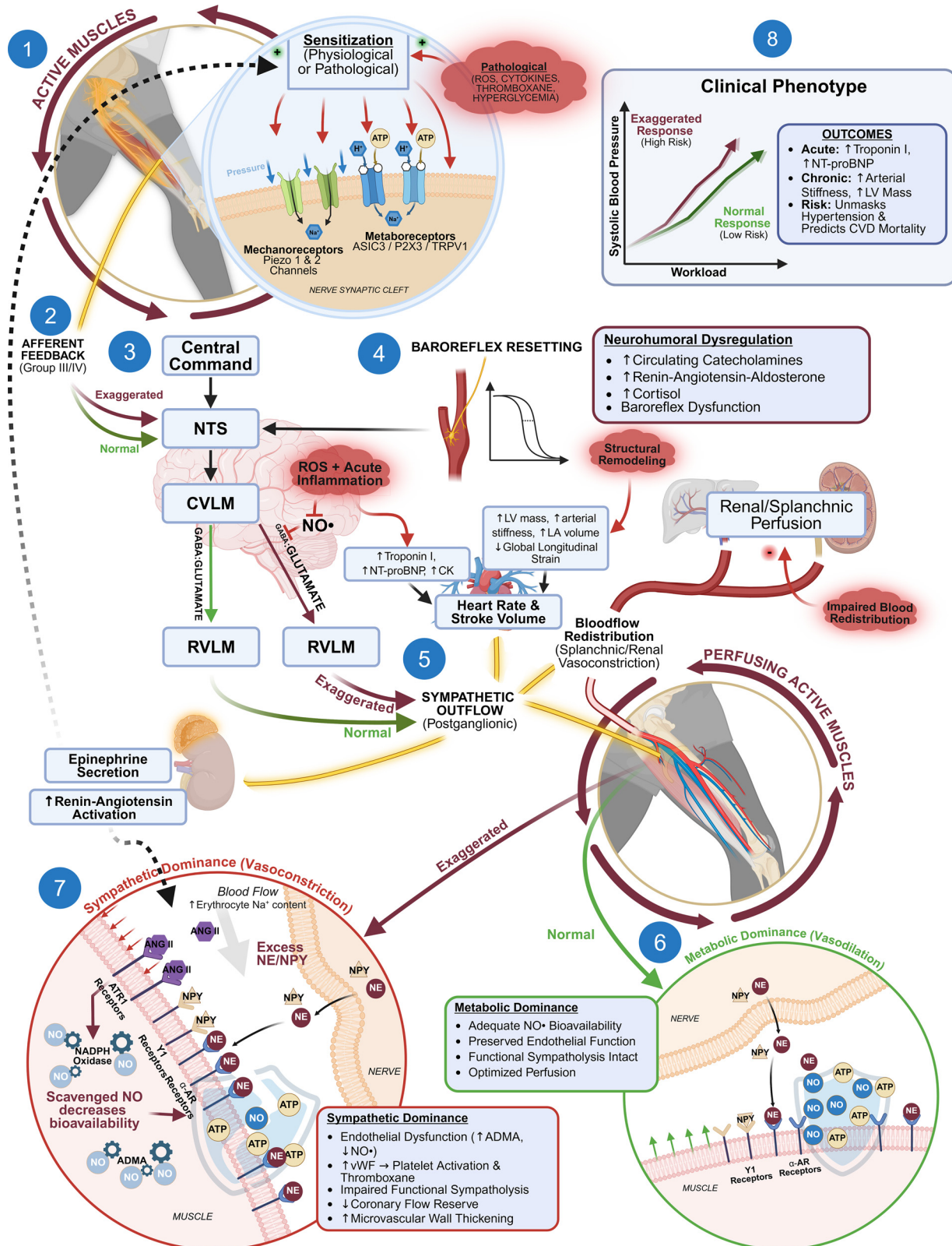
Morbidity.

Exaggerated BP during maximal exercise was not associated with risk for greater aortic dilation (216). A higher peak exercise BP was associated with an increased risk of heart failure among the general population (217). Similarly, higher rates of rise in SBP during exercise (218), as well as higher submaximal (219) and maximal (218) exercise SBP, are associated with a higher future risk of myocardial infarction among men without overt disease. In stark contrast, this relation appears to reverse in the case of established disease. Among those with coronary artery disease, a higher peak exercise BP was associated with a lower risk of both heart failure and myocardial infarction (220). This strongly suggests that the

utility of exercise BP for predicting the risk of developing heart failure may depend on disease status.

Higher submaximal exercise BP is associated with LVH in some (221, 222), but not all (223), past reports. Higher peak

exercise SBP is associated with LVH in most (199, 222, 224–226), but not all (200, 227), past reports. Among the many guidelines for exaggerated exercise BP, only the American Heart Association guidelines linked exaggerated exercise BP



with LVH in athletic and nonathletic adults (194). Recent evidence, however, suggests that adults with established LVH may exhibit apparently exaggerated exercise BP responses, reflecting primarily elevated resting BP rather than pathological exercise pressor reflex sensitivity (195). Furthermore, higher exercise SBP was inversely associated with left ventricular (LV) mass indexed to body surface area among resistance-trained young women (228). Thus, exaggerated exercise BP is likely linked to future LVH, but findings have been equivocal.

A higher rate of rise in SBP during exercise (229) and SBP during maximal exercise (229, 230) is associated with an increased risk of stroke among healthy middle-aged men. Subsequent research, however, indicates that exaggerated exercise SBP is associated with an increased future stroke risk, specifically among adults with a history of cardiovascular disease, but not among those without (231). Conversely, for patients with known or suspected coronary artery disease, exaggerated exercise BP was either inversely associated (220) or showed no association (232) with stroke events. Therefore, the association between exaggerated exercise BP and stroke risk remains unclear.

Exaggerated BP responses during low-intensity (100, 233), moderate- to vigorous-intensity (234–236), vigorous-intensity (237), or peak (238–240) exercise are associated with an increased future risk of cardiovascular disease, often even after adjustment for resting BP. Notably, some data suggest that higher DBP, and not SBP, during submaximal exercise is associated with a greater incidence of CVD (237, 241). Collectively, the evidence suggests a positive association between exercise BP and future CVD risk.

Higher BP during isometric handgrip exercise is associated with an increased future risk for hypertension (242–245). Exaggerated exercise BP during submaximal dynamic exercise predicts an increased future hypertension risk in most (171, 241, 243, 246–252), but not all (243, 253), studies, sometimes even independently of traditional risk factors [e.g., body mass index (BMI), waist circumference] (254). Exaggerated SBP during submaximal exercise (255) may reflect masked hypertension, which would only otherwise be captured with ambulatory BP monitoring, a method that can be more burdensome to collect (256–259). Exaggerated SBP during maximal or peak dynamic exercise predicts an increased future hypertension risk in a dose-dependent manner (239) in most (165, 208, 238–240, 248, 251, 252, 260–266), but not all (267), studies. This is also true when peak SBP is indexed to peak

workload (239, 248) and among highly trained athletes (268). Taken together, the majority of evidence suggests that higher BP during isometric, submaximal dynamic, and maximal/peak dynamic exercise predicts an increased future risk for developing hypertension.

Mortality.

Exaggerated BP during submaximal exercise is associated with an increased future mortality risk among healthy adults and those with hypertension (219, 223, 230, 236, 269–272). Interestingly, increases in submaximal exercise SBP values over 7 yr were associated with higher mortality, highlighting the importance of tracking exercise BP over time. Notably, most epidemiological data linking exaggerated BP during submaximal exercise with poor health outcomes are derived from a single cohort of ~2,000 healthy middle-aged men who completed exercise testing in the early 1970s (219, 230, 236, 269–271). Therefore, more work using diverse cohorts should corroborate these findings.

Exaggerated SBP during peak exercise is associated with an increased risk of cardiovascular or all-cause mortality in some (273, 274), but not all (220, 275–277), studies. Part of the discrepancy may stem from disease status and confounding variables (e.g., smoking) in statistical analyses (278). More recent research (5, 239, 279) advocates for considering absolute workload through workload-indexed SBP (183). A higher SBP/MET slope is associated with an increased mortality risk and demonstrates superior predictive value compared to SBP alone (280), a finding further supported by reduced risk with smaller, yet adequate, BP changes relative to maximal workload (190). However, other work suggests that CRF does not attenuate the relation between SBP and higher mortality (273). Mechanistically, exercising systemic vascular resistance may be a primary mechanism underlying the association between exercise BP and mortality (281), a hypothesis partly supported by data from the Framingham Heart Study linking higher exercise DBP, but not SBP, with greater all-cause mortality (241).

Collectively, the prognostic evidence presented for both morbidity and mortality, although complex, points toward a central conclusion. The association between exaggerated exercise BP and future risk appears most robust and consistent for hypertension and general cardiovascular disease. The link with mortality is also most frequently cited at submaximal intensities, although this evidence rests on a narrow cohort.

Figure 2. Exercise pressor reflex dysfunction: The figure illustrates physiological (green pathways) and pathological (red pathways) exercise pressor reflex function. 1) Active muscles: active skeletal muscle initiates the exercise pressor reflex. 2) Afferent feedback and sensitization: group III/IV afferents (mechanoreceptors and metaboreceptors) become active. Pathological factors sensitize these receptors, and specific channels, such as Piezo 1/2, transient receptor potential vanilloid 1 (TRPV1), and acid-sensing ion channel 3 (ASIC3), modulate the response. 3) Central command provides feedforward signals to the nucleus tractus solitarius (NTS). 4) Baroreflex resetting: neurohumoral dysregulation leads to baroreflex dysfunction, impairing its ability to buffer the metaboreflex. 5) Central integration: signals are processed in the brain stem [NTS, caudal ventrolateral medulla (CVLM), rostral ventrolateral medulla (RVLM)]. Reactive oxygen species (ROS) and reduced nitric oxide (NO) bioavailability within the NTS contribute to exaggerated sympathetic drive. Sympathetic outflow: increased postganglionic sympathetic activity raises heart rate/stroke volume and causes vasoconstriction in nonactive tissues. 6) Functional sympatholysis occurs where vasodilation attenuates sympathetic vasoconstriction to optimize muscle tissue perfusion. 7) In pathological states (e.g., heart failure), exaggerated sympathetic vasoconstriction and endothelial dysfunction impair muscle tissue perfusion. 8) The resulting exaggerated systolic blood pressure response is associated with a significantly increased risk of cardiovascular events and mortality. ADMA, asymmetric dimethylarginine; AR, adrenergic receptor; ATP, adenosine triphosphate; ATR1, ANG II type 1 receptor; CK, creatine kinase; CVD, cardiovascular disease; GABA, gamma-aminobutyric acid; LA, left atrial; LV, left ventricular; NE, norepinephrine; NPY, neuropeptide Y; NT-proBNP, NH₂-terminal pro-B-type natriuretic peptide; P2X3, purinergic receptor P2X3; Piezo 1 and 2, mechanically activated ion channels; vWF, von Willebrand factor. Figure created with a licensed version of BioRender.com.

In contrast, the evidence for specific outcomes like LVH and stroke, as well as for peak-exercise mortality, is equivocal and confounded by factors like preexisting disease. This pattern of discrepancy is not a contradiction; rather, it strongly supports this review's central thesis that submaximal exercise BP is the superior and more reliable prognostic marker. The inconsistent findings for maximal exercise BP, therefore, do not invalidate the "strong predictor" status of exercise BP but instead underscore the critical importance of measurement timing for clinical risk stratification.

CARDIORESPIRATORY FITNESS

$\dot{V}O_{2\max}$ serves as the reference measure of CRF, but clinical populations often achieve only a symptom-limited peak ($\dot{V}O_{2\text{peak}}$). CRF demonstrates strong protective effects against CVD (146, 282). Genetic factors account for 72% of the individual variability in $\dot{V}O_{2\max}$ for children and young adults (283, 284). This review refers to CRF when exercise capacity (e.g., peak workload) or estimated CRF is reported unless a distinction, such as between measured and estimated CRF, is crucial for explaining divergent findings among studies. This section discusses associations between CRF and exercise BP.

Cardiorespiratory Fitness and Blood Pressure during Submaximal Exercise

Higher CRF is associated with lower SBP during submaximal exercise across populations (183, 222, 234, 285–288) and even weakly predicted lower submaximal exercise SBP 7 yr later (173). Higher CRF is also associated with a slower rate of rise in both SBP and DBP during submaximal exercise among adults with untreated hypertension. Importantly, CRF significantly predicts DBP responses independently of changes in circulating catecholamines (285). Some data suggest a J-shaped relation between CRF and submaximal exercise SBP among young males (289). However, the authors did not statistically adjust for body mass index (BMI), a key limitation as BMI is also associated with exercise BP (290). Overall, the data indicate that higher CRF is linked with lower submaximal exercise SBP.

Cardiorespiratory Fitness and Blood Pressure during Peak Exercise

The relation between CRF and peak exercise BP demonstrates complex, population-specific patterns. About 28–43% of competitive athletes exhibit an exaggerated BP response (201), with greater SBP responses observed in endurance athletes (291). Higher CRF is associated with higher peak exercise SBP in young and middle-aged males (145, 286, 292). Conversely, CRF is not associated with peak exercise SBP among predominantly female or older adult cohorts (183, 285, 286). Notably, CRF is associated with lower peak exercise SBP among male and female adults with prehypertension (222). SBP has been indexed to CRF or exercise workload to enhance understanding of these distinct relations, as CRF is unrelated to the SBP/peak workload slope among athletes (279). This approach is further supported by findings demonstrating preserved SBP/ $\dot{V}O_2$ slopes in clinical populations despite elevated absolute exercise BP values (195).

Furthermore, recent evidence suggests that the SBP/MET slope offers a sex-independent index of vascular function, unlike absolute SBP/watt ratios that remain influenced by body mass (187, 189).

During peak exercise, professional male athletes exhibited a lower SBP/MET and SBP/watt slope than a nonathlete control group (183). Endurance athletes have considerably lower SBP/ $\dot{V}O_2$ slopes, but not the SBP/watt slope, compared with predicted normative values (279). The lower SBP for a given metabolic demand may partly explain why this group's higher absolute SBP does not necessarily increase cardiovascular disease risk (191). Adjusting for metabolic demand (i.e., SBP/MET slope) abolishes sex differences among competitive athletes (189). Therefore, indexing SBP to CRF or exercise workload is crucial for understanding the distinction between physiological and pathophysiological exercise BP responses (Fig. 3).

EXERCISE TRAINING

Physical activity is any movement that increases energy expenditure above the resting state. Genetic factors account for ~45% of physical activity heritability in adulthood (293). Classic experimental work demonstrates that 3 wk of bed rest produces cardiovascular deconditioning (e.g., cardiac and skeletal muscle atrophy) more severe than 40 yr of aging (294, 295). People with sedentary habits exhibit a heightened cardiovascular response during exercise, which may partially explain their higher risk for acute myocardial infarction (296).

Exercise is planned, structured, and repetitive physical activity to improve or maintain fitness (297). Central command (298) and baroreflex resetting (299) establish the hemodynamic response to exercise, producing graded, intensity-dependent increases in heart rate and BP (300). The mechanisms underlying exercise-induced changes in exercise BP are multifaceted. Exercise imposes repeated dynamic hemodynamic forces on the arterial system (shear stress, pulsatile pressure, and wall strain), driving endothelial and structural adaptations, which manifest as improved arterial function, vascular remodeling, and enhanced microvascular health (301). Ischemic conditions during exercise increase muscle sympathetic outflow (300). Thus, the specific exercise stimulus (e.g., prolonged high BP, ischemic vs. nonischemic) influences the modality-specific adaptations from exercise training that may differentially affect exercise BP.

Aerobic Exercise Training

Aerobic exercise training (AET) is the most extensively studied exercise modality. AET reduces resting SBP by ~4 mmHg and DBP by ~3 mmHg in healthy adults (302). These reductions are also observed among older adults (303), Black males (304), sedentary adults (305–308), patients with chronic kidney disease (309), men during fixed-workload cycling (205), patients with peripheral arterial disease (310), and normotensive individuals with exaggerated BP responses (254). However, these reductions are not observed after 12 wk of AET among sedentary middle-aged female adults (311), 10 wk of AET among sedentary adults with hypertension (312), 6 wk of AET among young sedentary males (313), or 3

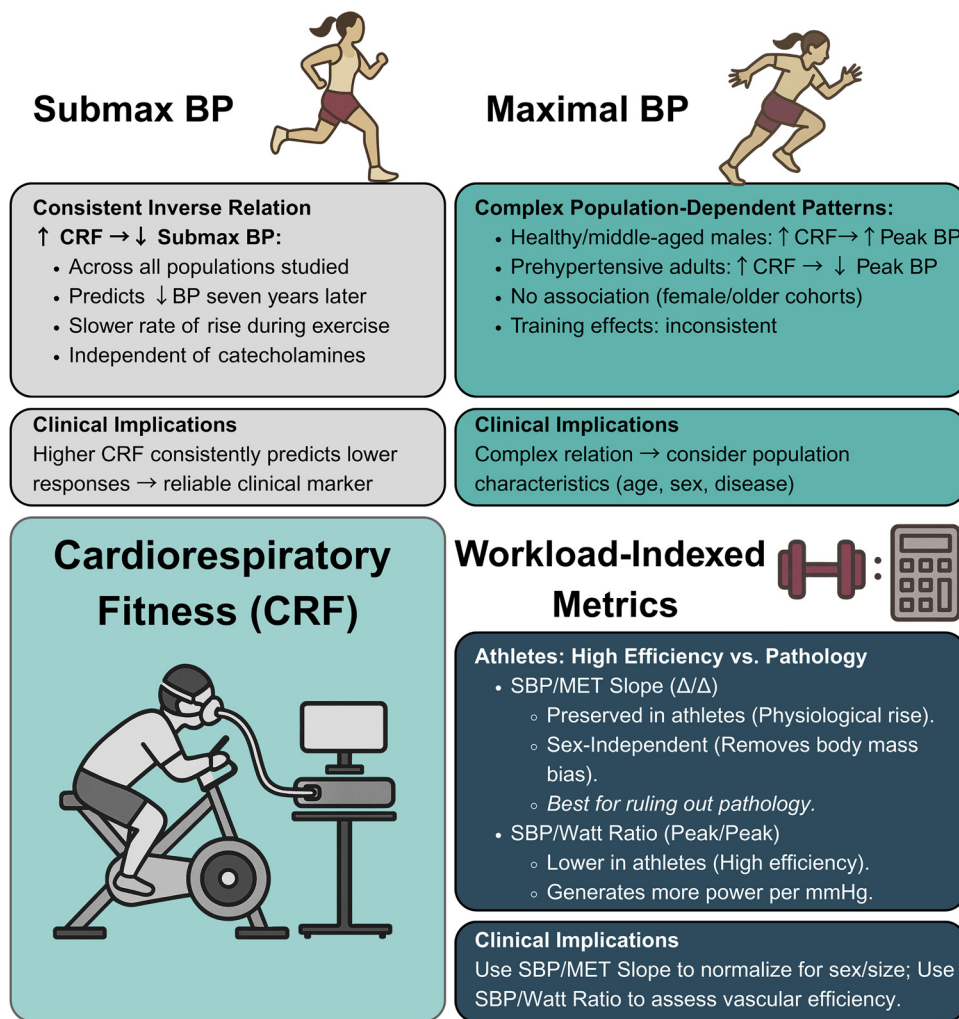


Figure 3. Cardiorespiratory fitness and exercise blood pressure. BP, blood pressure; CRF, cardiorespiratory fitness; MET, metabolic equivalent; SBP, systolic blood pressure; Submax, submaximal; $\dot{V}O_2$, oxygen uptake rate.

wk of AET among adults with hypertension (314). AET intensity did not affect exercise BP reduction among adults >55 yr of age (315, 316). Patients with peripheral arterial disease (310, 317, 318) and postmenopausal females with obesity (319) likewise achieve lower submaximal exercise BP with AET. These benefits likely stem from increased arterial compliance, enhanced endothelial function with higher nitric oxide availability, improved mitochondrial function, greater muscle oxidative capacity, enhanced vascular function, and reduced total peripheral resistance (4, 320–323). These adaptations are localized, with unilateral training attenuating exercise BP responses in the trained limb (324–327). Additional systemic benefits include improved sympathetic modulation (328), enhanced arterial baroreflex sensitivity through vagal mechanisms (329), and favorable shifts in redox balance (330, 331). Thus, AET provides modest but consistent reductions in exercise BP in many, though not all, populations.

Resistance Exercise Training

Acute BP responses during resistance exercise training (RET) are modulated by training status, exercise intensity, and muscle groups engaged. RET induces abrupt BP changes, which are significantly modulated by breathing

patterns. Recent evidence suggests that a brief breath hold during high-intensity exertion [$>80\%$ maximal voluntary contraction (MVC)] acts as a “brief Valsalva” that is often unavoidable for spinal stability (332). This maneuver generates a spike in intrathoracic pressure that is transmitted directly to the arterial tree, driving systolic pressures as high as 300 mmHg (333). Mechanistically, however, this high intrathoracic pressure simultaneously compresses the heart, thereby attenuating the transmural pressure (wall stress) despite the massive systemic rise (332, 334, 335). Although this may protect the left ventricle in healthy athletes, avoiding sustained Valsalva maneuvers remains critical in clinical populations to mitigate the absolute vascular load. During maximal effort lifts, BP reaches up to 480/350 mmHg because of increased intramuscular pressure, mechanical compression of blood vessels, and sympathetic activation (97). Acutely, RET using lower loads and higher repetitions acutely increases cardiovascular reactivity more than heavier loads with fewer repetitions among postmenopausal hypertensive women (336). Greater active skeletal mass (337) and higher loads during RET produce dose-dependent increases in BP (96, 338, 339). Concentric muscle actions produce greater BP increases than eccentric muscle actions (97) because eccentric movements involve lower motor unit

recruitment (97, 111) and metabolic demand (340), leading to reduced sympathetic activation (54, 341) and vascular compression (342, 343). Lower body exercise elicits higher brachial and aortic SBP; upper body exercise reduces diastolic BP (344). Interestingly, fast-twitch-dominant muscle exercise elicits higher BP than slow-twitch-dominant muscles (345).

Chronic training adaptations alter these acute responses. In young men, RET decreases BP responses for a given submaximal absolute workload while increasing BP response during maximal exercise (346). Individuals trained in bodybuilding exhibit lower exercise BP compared with novice lifters (347). These adaptations are primarily localized, with unilateral training attenuating exercise BP responses in the trained limb (324–327, 348). However, some systemic adaptations exist, such as those occurring via central command (349) and the chemoreflex (327). Traditional and time-efficient RET attenuates exercise BP among patients in cardiac rehabilitation (350), patients with heart failure during peak treadmill exercise (351), and patients with intermittent claudication during submaximal exercise SBP (outperforming BP reductions from a walking intervention) (352). Mechanistically, muscle metaboreflex activation during heavy RET increases muscle sympathetic nerve activity, vascular conductance, and BP during baroreflex unloading (69). Overall, RET provides reductions in exercise BP, although the mechanisms differ significantly from aerobic training (Fig. 4).

Combined Aerobic and Resistance Exercise Training

Reductions in resting BP with combined AET and RET are ~2 mmHg, which is smaller than the 3- to 4-mmHg reductions observed with AET alone. Nonetheless, 12 wk of combined exercise training (CET) reduced exercise SBP among young men with obesity and prehypertension (353). Likewise, CET for 6 mo reduced submaximal exercise BP in older adults (354) and in adults with well-healed severe burn injuries (355). Among the older adults (354), the exercise BP benefits were weakly associated with increases in $\dot{V}O_{2peak}$, and body mass reductions after training were associated with greater reductions in exercise BP (354). However, 12 wk of AET or RET, but not CET, lowered submaximal exercise DBP in older adults (356). In summary, CET seems more likely to reduce exercise BP in clinical populations than in adults without chronic disease.

Novel Exercise Modalities

Novel exercise modalities offer unique hemodynamic profiles and potential therapeutic benefits (Fig. 5).

Blood flow restriction training.

Blood flow restriction exercise training (BET) combines low-intensity exercise and external pressure to restrict venous return and partially occlude arterial inflow, thereby enhancing muscle metaboreflex stimulation. Resting blood flow occlusion estimates reasonably predict exercise occlusion (357). Despite low mechanical loads, this modality produces high metabolic stress and augments exercise BP in most studies (339, 358–367), though not all (368–373). This response is likely attributable to metaboreflex stimulation (98, 374, 375) and pain (376). BET induces load-dependent (377, 378) and time-dependent (379) cardiovascular responses, with higher flow restriction cuff pressures limiting venous return, and

subsequent stroke volume and cardiac output (339, 380). Acute traditional RET performed until failure, however, causes greater BP during exercise than acute BET (381).

Five weeks of endurance BET attenuated BP responses during unilateral exercise in the trained, but not untrained, limb among young male adults, suggesting localized peripheral adaptations (382). One month of dynamic BET attenuated BP responses during handgrip exercise among young male adults (383). This effect, however, was not attributable to reduced metaboreflex activity. Thus, more work is needed in populations other than young male adults, as well as with a greater understanding of the underlying mechanisms.

Isometric exercise training.

Isometric exercise training (IET) elicits superior SBP lowering at rest (–8 mmHg) compared with AET, RET, and CET (–4 to –5 mmHg for all) (333, 384). Traditional RET and IET produce comparable acute cardiovascular responses when matched for workload; however, IET can increase total peripheral resistance, whereas RET either does not change or reduces total peripheral resistance (385). Interestingly, BP is highest at the weakest joint angles but does not differ between joint angles at equivalent relative intensities (386). Absolute handgrip strength influences BP responses to isometric handgrip exercise, with observed sex differences diminishing after adjusting for muscle strength (87, 101, 387). Six weeks of unilateral handgrip training reduced sympathetic nerve activity responses during isometric handgrip in the trained arm without altering heart rate or BP, suggesting diminished metaboreflex stimulation (348). A small study reported no difference in sympathetic or cardiovascular (including BP) responses to acute handgrip exercise after 5 wk of IET (388). Thus, IET is promising for reducing resting BP, but these effects do not appear to translate into exercise, given the currently limited literature available.

High-intensity and sprint interval training.

High-intensity interval training (HIIT) and sprint interval training (SIT) offer time-efficient alternatives to traditional AET. Meta-analytic evidence supports HIIT as comparable to AET for resting SBP lowering among the general population (384) and as the most effective modality for resting SBP lowering in high-risk cardiovascular populations (389). SIT, defined as an “all-out” maximal, low-volume protocol (384), produces significant reductions in resting SBP (5 mmHg) and DBP (3 mmHg). Notably, the SBP reductions attributed to HIIT appear primarily driven by SIT rather than aerobic interval training (384). However, evidence regarding its effect on exercise BP is lacking. One study reported that HIIT reduced exercise SBP during the high-intensity, but not low-intensity, portions of exercise among patients with percutaneous coronary intervention after myocardial infarction who were completing cardiac rehabilitation (390). Therefore, further research is necessary in this area, given the promising effects observed at rest and SIT-induced improvements in peripheral arterial stiffness and flow-mediated dilation comparable to traditional endurance training (301).

Inspiratory muscle strength training.

High-resistance (75% of peak inspiratory pressure) inspiratory muscle strength training (IMST) reduces resting SBP by



AEROBIC TRAINING

BP Response: ↓ 4/3 mmHg (SBP/DBP)

- Most consistent across all populations and intensities
- Benefits both submaximal & maximal exercise BP
- Intensity-independent effects in adults >55 years
- Localized to trained limbs

Mechanisms: Vascular & Metabolic Adaptations

- ↑ Arterial compliance and endothelial function
- ↓ Total peripheral resistance during exercise
- ↑ Mitochondrial function
- Improved sympathetic modulation & ↑ baroreflex sensitivity

Evidence Ranking: #1 (Meta-analysis supported)

Clinical Note: Most consistent and evidence-based choice for exercise BP reduction across all populations



RESISTANCE TRAINING

BP Response: Complex, Population-Dependent

- ↓ Submaximal BP responses in trained individuals
- ↑ Maximal BP during acute training sessions
- Clinical populations: ↓ SBP response +28%→+11%
- Trained individuals: ↓ BP responses vs beginners
- Upper vs lower body regional differences

Mechanisms: Neural & Mechanical Adaptations

- Acute: ↑ Intramuscular pressure, ↑ mechanical vessel compression
- Chronic: ↓ Muscle metaboreflex sensitivity
- Localized neural adaptations in trained limbs
- Training-induced neural efficiency improvements

Evidence Ranking: #2 (Population-dependent effectiveness)

Clinical Note: Most beneficial in clinical populations and experienced trainees



COMBINED TRAINING

BP Response: Limited Benefits

- ↓ DBP 2 mmHg only (no SBP reduction)
- Less effective than individual training modalities
- Delayed cardiac autonomic recovery
- Heart rate remains elevated longer

Mechanisms: Competing Adaptations

- Mixed vascular and neural adaptations
- Potential interference between aerobic and resistance adaptations
- Slower cardiac vagal reactivation • Similar post-exercise hypotensive effects

Evidence Ranking: #3 (Least effective for exercise BP)

Clinical Note: Consider individual modalities for greater exercise BP benefits



ISOMETRIC TRAINING

Exercise BP Response: Moderate Exercise BP Benefits

- ↓ Exercise BP: 27-28% DBP responses in trained limb only
- ↓ MBP during ischemic exercise (109→98 mmHg)
- ↓ Sympathetic responses during exercise (111%→38%)
- Primarily benefits resting BP (↓ 10/7 mmHg)

Mechanisms: Chemoreflex & Neural Adaptations

- ↓ Muscle chemoreflex stimulation
- ↓ Sympathetic nerve activity responses
- ↑ Pressor response threshold
- Enhanced metabolic efficiency in trained muscle

Evidence Ranking: #4 (Localized benefits, superior for resting BP)

Clinical Note: Consider other modalities for exercise BP reduction - best for resting BP

Figure 4. Traditional exercise training and exercise blood pressure (BP). DBP, diastolic blood pressure; MBP, mean blood pressure; SBP, systolic blood pressure.

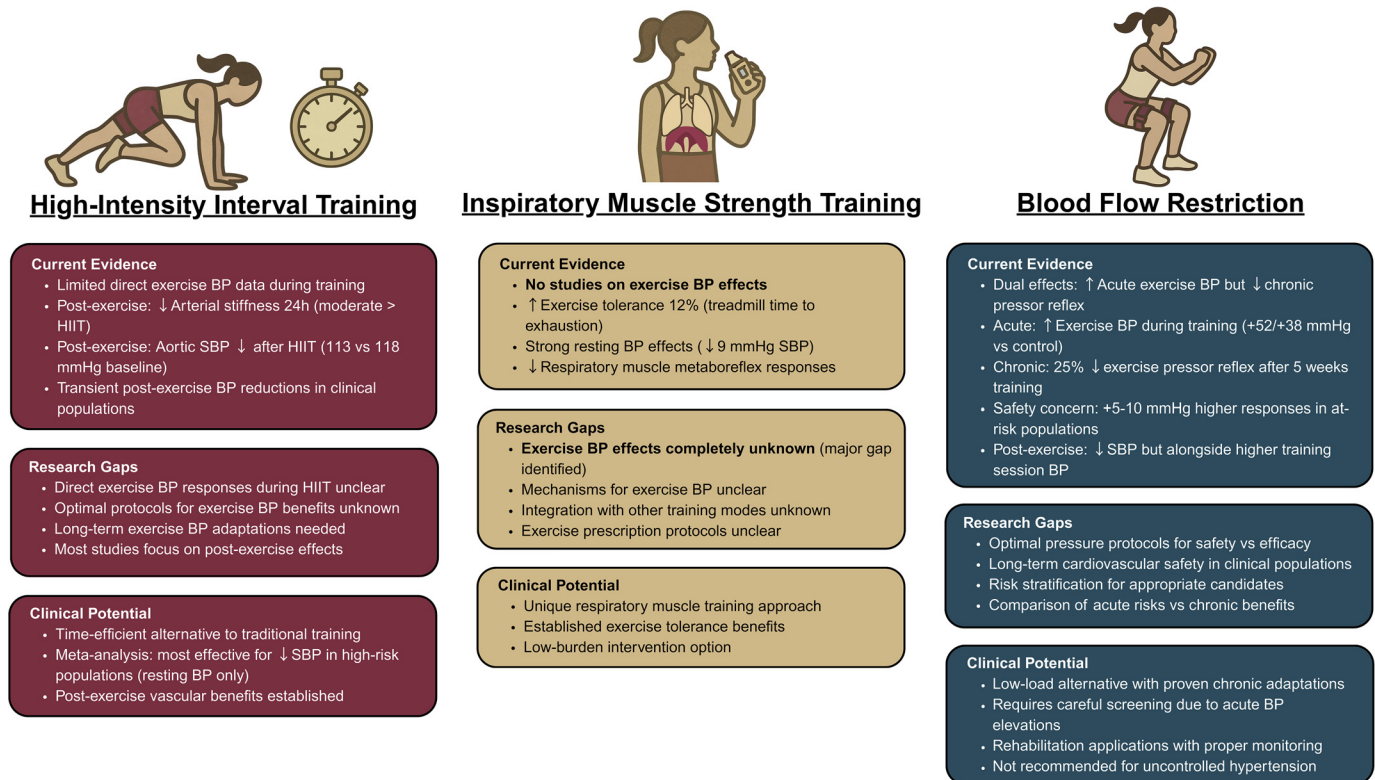


Figure 5. Novel exercise training modalities and exercise blood pressure (BP). HIIT, high-intensity interval training; SBP, systolic blood pressure.

9 mmHg, independent of health status (391). A single session of high-resistance IMST acutely reduces limb blood flow and shear rate during exercise (392). Four weeks of low-resistance (30% of peak inspiratory pressure) IMST improved hemodynamic responses during respiratory muscle and forearm exercise among patients with heart failure (393). However, the effect of high-resistance IMST on exercise BP has not yet been investigated, highlighting a significant research gap.

CONCLUSIONS

Knowledge Gaps and Future Research Directions

Individual variability and autonomic responses.

A fundamental challenge in exercise BP research is the pronounced interindividual variability in physiological responses. This variability in muscle sympathetic nerve activity during exercise, however, lacks a strong association with BP regulation, particularly during low- to moderate-intensity exercise (394). A critical gap in the literature is whether interindividual muscle sympathetic nerve activity variability (which can be more easily assessed by using circulating norepinephrine as a surrogate marker) indicates any future clinical risk, a question that should guide future research in this field.

The interindividual variability in muscle sympathetic nerve activity response patterns during static handgrip contributes to differences in autonomic cardiovascular regulation, with positive responders exhibiting greater increases in muscle sympathetic nerve activity yet similar BP elevations across groups (395). This variability may partly stem from

the polymodal nature of some group III and IV afferents, which are susceptible both to mechanical distortion and to metabolite accumulation (25). Future work should consider using established methodological approaches (passive limb movement, handgrip exercise, postexercise circulatory occlusion) to systematically delineate mechanoreceptor and metaboreceptor contributions [considering limb location and polymodal afferents (396)], thereby enhancing understanding of individual variability in exercise pressor reflex responses.

Reflex interactions and integration.

The interactions among reflexes, including the mechanoreflex, metaboreflex, chemoreflex, baroreflex, and psychologically related reflexes (such as panic and breathlessness), are still not fully understood. A comprehensive study of this intriguing topic requires consideration of these interactions rather than just isolated reflexes. Future research should focus on delineating the precise role of EPR-related receptors and neurotransmitters in humans and understanding how GABAergic mechanisms influence the resetting of baroreflex responses during exercise (14). Baroreflex-EPR dynamics warrant particular investigation (57), as does the role of the metaboreflex, specifically the P2X3 signaling pathway, given its key role in EPR sensitivity (397, 398). Activation of the muscle metaboreflex during heavy exercise modulates arterial baroreflex function by augmenting muscle sympathetic nerve activity. However, these complex interactions remain incompletely understood (69). For instance, postexercise circulatory occlusion during systemic hypercapnia sustains ventilation, whereas isolated local muscle hypercapnia does

not (327). This interaction highlights that muscle afferent input alone may be insufficient; however, in combination with central chemoreceptor stimulation, it can amplify reflex responses.

Perspectives and Significance

Maximal exercise BP: limited modifiability and prognostic value.

Maximal exercise BP is primarily determined by age (tending to be higher with advancing age), sex (higher in men), and maximal exercise workload capacity (170). Training interventions have demonstrated inconsistent effects on peak exercise BP. Some studies indicate that higher CRF is associated with higher peak exercise SBP in young and middle-aged males (286, 292). However, other research in more diverse cohorts has found no association (183), and a reduction in peak exercise SBP has only been observed in specific populations, such as prehypertensive adults (222). This is, however, not overly concerning, as epidemiological data in this area are often less definitive and frequently confounded by a lack of consistent correction for CRF or exercise workload.

Submaximal exercise BP: a modifiable risk factor.

AET and RET consistently provide modest reductions in submaximal exercise BP across many populations, thereby limiting unnecessary hemodynamic load. The extent to which higher inherent CRF or AET-induced increases in CRF yield similar benefits in lowering submaximal exercise BP remains unclear. AET, however, offers numerous benefits beyond its effect on exercise BP and is a potential strategy for reducing submaximal exercise BP (222, 234). For instance, higher CRF even predicts lower submaximal exercise SBP 7 yr later (173). Equally relevant, higher CRF is associated with slower rates of rise in BP during submaximal exercise among adults with hypertension (285). Indeed, higher habitual physical activity provides similar protection (113), but it is most apparent among younger females (399). RET is another strategy to reduce submaximal exercise BP (352). Importantly, data suggest that adaptations are localized (324, 327, 348). Therefore, whole body RET is recommended to maximize systemic benefits. Overall, these findings suggest that submaximal exercise BP should be the primary target for cardiovascular risk reduction. Future research should investigate optimal exercise modalities, intensities, and durations to maximize improvements in different populations (331).

The critical importance of workload context.

Risk assessment protocols should emphasize workload-indexed BP responses to provide meaningful clinical context for exercise testing results. Therefore, standardized submaximal exercise BP should become a routine component of cardiovascular risk assessment in clinical practice. Professional athletes demonstrate lower SBP/MET and SBP/watt slopes during peak exercise (183), and endurance athletes show considerably lower SBP/ $\dot{V}O_2$ slopes (279). Similarly, adults with LVH demonstrate preserved SBP/ $\dot{V}O_2$ slopes despite higher absolute exercise SBP values. This highlights the need to contextualize metabolic and/or mechanical demand across diverse clinical populations (195). Importantly, higher SBP/MET slopes are associated with mortality risk and

outperform absolute SBP values alone (280), emphasizing the importance of fitness-indexed BP normative data (193, 194).

Clinical implementation pathway.

There is strong rationale for submaximal exercise BP as a clinically relevant risk factor. For example, a meta-analysis of 46,314 adults found that submaximal, but not maximal, exaggerated exercise BP responses were associated with a higher risk for adverse cardiovascular events and mortality, independent of resting BP and other cardiovascular risk factors (5). Given the substantial evidence demonstrating the superior prognostic value of submaximal exercise BP responses, there is a compelling need for submaximal exercise BP to be adopted as a more consistent prognostic measure in clinical practice worldwide (5). Despite established evidence, exaggerated exercise BP remains underutilized for risk stratification, a missed opportunity given its lower burden than ambulatory monitoring.

Clinical guidelines have yet to affirm the independent CVD risk of exaggerated exercise BP (206). Nevertheless, clinical guidelines have not been updated to affirm the independent cardiovascular disease risk associated with exaggerated exercise BP, despite recommendations for measuring BP during exercise to detect uncontrolled high BP (5). The clinical pathway should include exercise systolic BP values of ≥ 150 –170 mmHg during submaximal exercise of moderate intensity as useful markers of exaggerated exercise BP. Values ≥ 170 mmHg warrant immediate clinical follow-up (5). Guidelines recommend standardizing graded exercise test protocols, with measurements taken at rest, after each stage, and upon cessation, using motion-tolerant automated devices whenever possible (400), which are designed to overcome the significant measurement challenges posed by noise and patient movement. This marks a paradigm shift toward recognizing submaximal exercise BP as a vital sign that provides unique prognostic information beyond resting measurements.

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We used the institutionally licensed Grammarly Pro software while writing this article. The software checks for grammar and typographical errors and provides suggestions to enhance clarity. Where appropriate, we edited the text to improve clarity when Grammarly flagged it as unclear.

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AUTHOR CONTRIBUTIONS

J.W.H. prepared figures; J.W.H. and J.C.W. drafted manuscript; J.W.H., C.D., P.S., and J.C.W. edited and revised manuscript; J.W.H., C.D., P.S., and J.C.W. approved final version of manuscript.

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