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Review Article

Aerobic Exercise Only or in Combination with Resistance Exercise Provides a Significant Reduction in Blood Pressure: A Narrative Review

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Abstract
Hypertension is a major global health concern and a leading risk factor for cardiovascular disease. While pharmacological therapy remains central, lifestyle interventions, particularly Aerobic Exercise (AE), offer a cost-effective, safe, and sustainable strategy for reducing Blood Pressure (BP) and improving cardiovascular health. Evidence indicates AE consistently lowers Systolic Blood Pressure (SBP) more than Diastolic Blood Pressure (DBP), with clinically meaningful reductions in both. This review aimed to synthesize current evidence on the effects of AE, alone or combined with Resistance Training (RT) or dietary interventions, on BP in individuals with hypertension, elucidate underlying mechanisms, identify moderating factors, and evaluate safety considerations. A narrative review of English-language articles published from 2015 to 2025 was conducted via PubMed, including original and review studies, as well as selected textbooks. Keywords included “aerobic exercise”, “exercise”, “hypertension”, “blood pressure”, “coronary artery disease”, and “cardiovascular disease”. Eligible studies were synthesized into themes reflecting acute and chronic exercise responses, combination interventions, mechanistic pathways, influencing factors, and safety. Thirty-four publications (26 original articles, 6 reviews, 2 textbooks) were included. AE alone or combined with RT consistently reduced SBP, with smaller reductions in DBP, whereas the combination with a hypocaloric diet primarily enhanced cardiorespiratory fitness and body composition. Mechanisms include improved endothelial function, autonomic regulation, metabolic efficiency, and anti-inflammatory effects. Effect size was influenced by age, sex, Body Mass Index (BMI), medication use, exercise timing, and vascular stiffness. Safety data indicated high tolerability, minimal adverse events, and strong adherence. AE is a safe and effective non-pharmacological intervention for hypertension, producing clinically significant BP reductions, particularly in SBP. Combining AE with RT or dietary modification offers additional cardiometabolic benefits. These findings reinforce AE as a cornerstone of hypertension management and support its integration into routine clinical practice.

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Keywords: Blood Pressure, Cardiorespiratory Fitness, Coronary Artery Disease, Hypertension, Resistance Training.

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Introduction

Hypertension is a leading global health problem and a major risk factor for Cardiovascular Diseases (CVD), affecting more than one billion adults worldwide.[1] Its high prevalence, combined with the associated risk of stroke, myocardial infarction, heart failure, and chronic kidney disease, makes hypertension a central focus of preventive and therapeutic strategies in modern medicine. While pharmacological therapy remains a cornerstone of hypertension management, it is often accompanied by side effects and long-term adherence challenges. Therefore, current international guidelines strongly emphasize the integration of non-pharmacological interventions, particularly lifestyle modifications, alongside pharmacological treatment.[2-4]

Among lifestyle interventions, regular physical exercise has been widely recognized as a cost-effective and sustainable strategy for reducing Blood Pressure (BP) and improving cardiovascular health. Aerobic Exercise (AE), in particular, has been identified as one of the most effective modalities in the prevention and treatment of CVD, including Coronary Artery Disease (CAD), hypertension, and type 2 Diabetes Mellitus (DM).[5-7] Substantial evidence indicates that AE contributes to significant reductions in both Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP), with consistent improvements observed across diverse populations and age groups.[8] Epidemiological studies have indicated that a reduction of just 2 mmHg in SBP is associated with a 6% decrease in stroke mortality and a 4% decrease in CAD mortality. In contrast, a 5 mmHg reduction corresponds to approximately 14% and 9% lower risks of these conditions, respectively.[9]

Professional organizations have published exercise guidelines recommending AE at moderate intensity for at least 30 minutes per day, five days per week, or at higher intensity for at least 20 minutes per day, three times per week.[10] However, the magnitude of the antihypertensive response may vary depending on exercise intensity, frequency, and duration.[11] Moreover, factors such as baseline BP levels, comorbidities, and adherence also play important roles in determining the effectiveness of exercise.[10,12]

Given the clinical importance of AE, further discussion is warranted to clarify its role in the comprehensive management of hypertension. This narrative review aimed to provide an overview of AE in hypertensive patients, focusing on the acute and chronic responses of AE, evidence on the effects of AE alone or in combination with other exercises or interventions on BP, the mechanistic links between AE and BP reduction, and safety issues in the implementation of AE in hypertension management. Such an overview is essential to support clinicians in optimizing exercise prescriptions as part of an integrated strategy to combat hypertension and reduce the global burden of CVD.

Methods

This narrative review synthesized current evidence on the effects of AE, either alone or in combination with Resistance Training (RT), on BP in individuals with hypertension and CVD. A comprehensive literature search was conducted in PubMed, chosen for its wide biomedical coverage, using the following keywords: *"aerobic exercise"* or *"aerobic training"*, *"exercise"* or *"training"*, *"hypertension"* or *"hypertensive"*, and *"blood pressure"*. The search was limited to English-language articles published between 2015 and 2025. Both original and review articles were eligible, provided the full text was available to ensure data quality. In addition, textbooks and earlier seminal references were considered when particularly relevant to the topic. All retrieved publications were screened for relevance, and those meeting the inclusion criteria were synthesized narratively, with emerging themes identified.

Results

A total of 34 references were included in this review, consisting of 26 original articles, 6 review articles, and 2 standard textbooks. The evidence was synthesized into six main themes. First, studies addressing acute and chronic responses to AE demonstrated consistent distinctions between transient hemodynamic changes and long-term adaptations in cardiovascular and metabolic function. Second, evidence on the effects of AE alone and in combination with RT, derived from 11 original studies across diverse populations, showed a robust reduction in SBP, with smaller effects on DBP. Variability in outcomes was linked to exercise timing and training modality. Third, in examining AE combined with a hypocaloric diet, one original and one review article indicated that diet was the dominant factor in lowering blood pressure. At the same time, AE contributed mainly to improvements in Cardiorespiratory Fitness (CRF) and body composition. Fourth, mechanistic studies consistently highlighted improved endothelial function, autonomic regulation, metabolic efficiency, and anti-inflammatory effects as key pathways underlying the antihypertensive benefits of AE. Fifth, factors influencing the magnitude of BP reduction included age, sex, Body Mass Index (BMI), antihypertensive medication use, and vascular stiffness. These moderators were reported across both original and review articles. Finally, six trials demonstrated that AE, alone or in combination with RT, is safe and well-tolerated, with no serious cardiovascular events and high adherence rates. Overall, the evidence supports AE as an effective and safe intervention for lowering BP, with additional benefits when combined with RT or dietary modification.

Discussion

Acute and Chronic Responses to AE in Hypertension

AE, also known as cardiorespiratory endurance training, involves repetitive movements of large muscle groups, accompanied by cardiovascular and respiratory adaptations that support sustained physical activity without premature fatigue. This type of exercise is specifically designed to enhance CRF and highlight the essential role of oxygen in energy metabolism.[10,13]

AE acutely increases cardiac workload by elevating Stroke Volume (SV) and Heart Rate (HR), thereby augmenting Cardiac Output (CO). This is accompanied by a transient rise in Systemic Vascular Resistance (SVR), which increases Mean Arterial Pressure (MAP). In contrast, chronic regular exercise leads to reductions in resting BP.[13-14]

Regular AE induces a range of physiological adaptations across multiple systems. In terms of BP, resting BP generally remains unchanged in normotensive individuals, but a slight reduction is often observed in hypertensive individuals. Regarding blood volume and blood lipids, total blood volume increases following regular training. Muscle glycogen storage is enhanced on a biochemical level, supporting improved energy availability during exercise. However, the relative proportion of fast-twitch and slow-twitch fibers does not significantly change, although the cross-sectional area occupied by these fibers may be altered depending on training stimuli. From the energy system perspective, exercise training promotes several key adaptations. Anaerobic glycolysis capacity increases, along with muscle glycogen storage and enzyme activity. Maximal oxygen uptake (VO_2max) improves, reflecting enhanced aerobic capacity. In addition, the use of triglycerides as an energy source increases, along with an increased metabolic rate of both fats and carbohydrates.[13]

Exercise is recognized as a cornerstone in the prevention and management of hypertension. In general, regular physical activity has been shown to prevent the development of hypertension in a clear dose-response manner. Each additional 10 Metabolic Equivalent (MET)-hours per week of physical activity is associated with a 6% reduction in the risk of developing hypertension. In individuals with established hypertension, exercise programs produce an average BP reduction of 5–8 mmHg; in those with prehypertension, 2–4 mmHg; and in normotensive individuals, 1–2 mmHg. Thus, the higher the baseline BP, the greater the magnitude of BP reduction achieved through physical activity.[15]

Aerobic, Resistance, and Combined Exercises Prescription

The studies analyzed used a variety of AE methods, but most employed structured, measured moderate-intensity activities. Methods used included treadmill walking, cycling on a cycle ergometer, brisk walking outdoors, jogging, swimming, and video-based aerobic dance. Exercise intensity was generally defined using a percentage of heart rate, either maximum Heart Rate (HRmax) or Heart Rate Reserve (HRR), or aerobic capacity such as VO_2max/VO_2peak, with the most common intensity ranges being 50–75% HRR, 60–75% HRmax, or Rating of Perceived Exertion (RPE) 12–14 (Table 1). One study employed specific-intensity methods, such as adjusting stride frequency with a metronome or using VO_2-peak-based intervals in High-Intensity Interval Training (HIIT) protocols.[16-17] The majority of protocols were continuous moderate-intensity training, while a small number employed HIIT or variations such as dance aerobics. Exercise supervision varies from fully structured and supervised to independent practice without formal supervision.[18]

The exercise duration in these studies ranged from 8 to 16 weeks, with most programs lasting 12 weeks. The typical training frequency was 2–3 times/week, but some studies recommended up to 3–5 times/week. Each session lasted 20-60 minutes, and most studies employed a training structure consisting of a 5–10-minute warm-up, a 20–45-minute core exercise phase, and a 5–10-minute cool-down (Table 1). Some studies employed a measured progression in both duration and intensity, such as increasing duration from 20 to 40 minutes and increasing intensity from 50% to

70% of VO_2max or gradually increasing HRR from 40% to 70%.[19-20] However, some other protocols maintained moderate intensity without systematic progression. Overall, the AE protocols used show considerable variation but remain consistent with the basic principles of AE: moderate-intensity exercise, gradual duration, and regular frequency to achieve optimal cardiovascular adaptation.

In addition to AE, several studies provided detailed descriptions of the RT and their integration within combined aerobic–resistance exercise programs. The types of RT varied across trials but were generally structured, machine-based, and targeted major muscle groups. One study implemented one of the most comprehensive RT protocols, consisting of 12 multi-joint and single-joint machine exercises (e.g., chest press, seated row, leg press, quadriceps extension), performed initially as 2 sets of 18–20 maximal repetitions and progressed to 3 sets of 10–14 repetitions taken to volitional exhaustion, with all loads automatically monitored through the TechnoGym digital system.[20] Another study also employed machine-based RT involving 6 core movements (leg press, bench press, knee extension, biceps direct threading, knee flexion, and low row) using 4 sets of 8–12 repetitions at 60–80% 1-Repetition Maximum (RM).[21] Meanwhile, one study used 6 resistance exercises performed for 2 sets of 10 repetitions at 60% 1-RM, with 1-RM directly assessed via integrated machine ergometers, ensuring precision in load prescription.[22] Isometric RT represented a distinct modality, as demonstrated by a previous study that prescribed handgrip training at 30% Maximal Voluntary Contraction (MVC) for 2-minute bouts with 1-minute rest, performed 5 days per week using a calibrated digital device. Collectively, these findings demonstrate that RT interventions across studies used structured, measurable, and reproducible methods targeting strength adaptations relevant to cardiovascular health.[18]

Table 1. Summary of exercise protocols given in previous studies.

Author (Year)	Exercise Protocol
Ammar (2015).[23]	• Frequency: 3x/week for 12 weeks • Intensity: ○ Warm-up or cool-down: 40% MHR (220-age) ○ Main phase: Moderate intensity (60-75% of predicted MHR) ○ MHR Formula: 210 – Age • Time: 30 minutes/session (5 minutes warm-up or cool-down, 20 minutes main aerobic phase) • Type: Treadmill walking • Progression: No progression applied.
Belozo et al. (2018).[25]	• Frequency: 3x/week (non-consecutive days, 48h rest) for 8 weeks • Intensity: Moderate intensity (70-80% of predicted MHR) • Time: 30 minutes per session • Type: Continuous treadmill walking • Progression: No progression applied.
Brito et al. (2019).[31]	Morning Training (MT) • Frequency: 3x/week • Intensity: Moderate intensity (50-70% of predicted maximal heart rate, or RPE 12-14) • Time: 45 minutes per session (including 5 minutes warm-up and 5 minutes cool-down) • Type: continuous aerobic walking/jogging • Training time of day: Morning sessions: 07.00-09.00 AM • Progression: Intensity maintained within moderate HR/RPE range. No progression applied. Evening Training (ET) • Frequency: 3x/week • Intensity: Moderate intensity (50-70% MHR or RPE 12-14) • Time: 45 minutes per session • Type: Continuous aerobic walking/ jogging • Training time of day: Evening sessions: 06.00-08.00 PM • Progression: HR monitored to keep moderate intensity. No progression applied.

Caminiti et al. (2021).[22]	• Frequency: 3x/week • Intensity: Moderate intensity (RPE 13-14) • Time: 10 min warm-up or cool-down, 60 min aerobic treadmill walkin • Type: Treadmill walking • Progression: Intensity maintained at RPE 13-14. No structured progression applied.
Feairheller et al. (2014).[53]	• Frequency: 3x/week for 6 months • Intensity: Started at 50% Vo_2max • Time: Started at 20 min • Type: Supervised aerobic exercise • Progression: ○ Duration progressed from 20 to 40 min ○ Intensity progressed from 50% to 65% VO_2max
Gorostegi-Anduaga et al. (2018).[17]	MICT • Frequency: 2x/week for 16 weeks • Intensity: Moderate (65% VO_2peak) • Time: 45 min/session • Type: Treadmill or cycle ergometer • Progression: Intensity maintained High-Volume HIIT • Frequency: 2x/week • Intensity: ○ Intervals at 90% VO_2peak ○ Recovery at 65% VO_2peak • Time: 45min/session • Type: Treadmill + cycling • Progression: Progressive workload via HR-based adjustments, fixed interval structure Low-Volume HIIT • Frequency: 2x/week • Intensity: ○ Intervals at 90% VO_2peak ○ Recovery at 65% VO_2peak • Time: 20 min/session • Type: Treadmill + cycling • Progression: Progressive workload via HR-based adjustments, fixed interval structure
He et al. (2018).[16]	• Frequency: 3x/week for 12 weeks • Intensity: 45-50% VO_2max, controlled using step frequency (Metronome) • Time: 60 minutes/session • Type: Outdoor brisk walking • Progression: Step frequency adjusted based on predicted VO2max. Intensity remained moderate.
Li et al. (2024).[32]	• Frequency: 4x/week for 12 months (At least 1 supervised group session per week; 3 sessions using uploaded videos at home) • Intensity: ○ Moderate intensity based on MHR (The target intensity was not specified as a percentage of MHR) ○ MHR formula: $208 - (0.7 \times \text{age in years})$ • Time: 60 minutes (10 minutes warm-up or cool-down, 40 minutes core aerobic exercise) • Type: Stair climbing, jogging, brisk walking, or cycling • Progression: Intensity was adjusted using heart rate monitoring (maintained at moderate intensity)

Lopes et al. (2021).[19]	• Frequency: 3x/week for 12 weeks • Intensity: started at 50% of VO$_2$ max, RPE 11-14 • Time: initial duration 20 minutes/session (10 minutes warm-up or cool-down, 40 minutes core aerobic exercise) • Type: Outdoor brisk walking • Progression: Weekly progression based on tolerance. Alternating between +5 minutes added to exercise duration and +5% of VO$_2$ max added to exercise intensity. Progression continued until reaching 40 minutes at 70% VO$_2$ max.
Maruf et al. (2013).[24]	• Frequency: 3x/week for 12 weeks • Intensity: 50–70% of HRR • Time: 45 minutes/session (5 minutes warm-up or cool-down, 35 minutes core aerobic exercise) • Type: Aerobic dance exercise using a 45-minute exercise video • Progression: Both intensity and duration of the aerobic dance segments were progressively increased. Progression continued until reaching 45 minutes at 50–70% HRR.
Pedralli et al. (2020).[21]	• Frequency: 2x/week for 12 weeks • Intensity: 50–75% of HRR, RPE 11-14 • Time: 40 minutes/session • Type: Aerobic exercise using a cycle ergometer • Progression: Progressive intensity increased within the 50–75% HRR range.
Pagonas et al. (2017).[18]	• Frequency: 3-5x/week for 12 weeks • Intensity: Started at 40% of HRR • Time: 60 minutes/session • Type: Aerobic exercise (Walking, jogging, cycling, or swimming) • Progression: No progression applied.
Schroeder et al. (2019).[20]	• Frequency: 3x/week for 8 weeks • Intensity: RPE 12-13 • Time: 30 minutes/session • Type: Aerobic exercise using a treadmill or ergocycle • Progression: ○ Intensity progressively increased from 40% to 70% HRR ○ Allowed self-selected increases as long as HRR remained ≤80%.

ET, evening training; HIIT, high-intensity interval training; HR, heart rate; HRR, heart rate reserve; MHR, maximum heart rate; MICT, moderate-intensity continuous training; MT, morning training; RPE, rating of perceived exertion; VO2max, maximal oxygen uptake; VO2peak, peak oxygen uptake.

Combined AE and RT (CT) was also clearly described in several trials, with most studies integrating AE and RT within a single session. CT sessions consisted of 30 minutes of AE (treadmill or cycle ergometer) followed by 30 minutes of RT, using the same RT exercises and intensities as the RT-only group but with reduced volume (2 sets and only 8 exercises).[20] One previous study used a protocol in which participants performed 40 minutes of treadmill-based AE at an RPE of 13–14 before 20 minutes of RT, following the priority principle to perform AE first.[22] Another study applied a balanced CT approach, prescribing 20 minutes each of AE and RT per session, with RT performed at 60–80% 1-RM.[21] Across these studies, the RT component of CT programs was consistently machine-based, moderate-to-high intensity, and matched to the independent RT protocols. In contrast, aerobic components were performed at moderate intensities defined via HRR or RPE. This demonstrates that the combined interventions were systematically structured, with clearly delineated durations, intensities, and exercise sequences that adhered to established exercise prescription principles.

Evidence of The Effects of AE Alone and in Combination with RT

AE, whether performed alone or in combination with RT or other interventions such as a hypocaloric diet, has been consistently shown to reduce BP in individuals with hypertension (Table 1). The magnitude of the effect varies depending on population characteristics and clinical conditions. For instance, Ammar (2015, 2016) reported that in overweight postmenopausal women with hypertension, AE combined with antihypertensive medication significantly reduced both SBP and DBP, with the greatest reductions observed in afternoon sessions (SBP −27 mmHg, DBP −15.4 mmHg), highlighting the potential influence of exercise timing on hemodynamic responses.[23]

Similar findings were observed in patients with resistant hypertension, where a 12-week AE program significantly lowered both SBP and DBP, particularly during daytime measurements, while improving CRF without altering body composition or metabolic profile.[19] Conversely, in patients with uncontrolled essential hypertension, AE did not significantly change mean SBP or DBP compared to controls; however, a greater proportion of participants in the exercise group achieved BP control.[24] In specific populations, such as obese individuals with type 2 DM, continuous AE did not produce statistically significant reductions in BP. However, effect-size analysis suggested a stronger trend toward improvement in SBP.[25] Brisk walking interventions similarly demonstrated significant reductions in SBP across various intensities, whereas DBP remained largely unchanged, confirming that AE preferentially lowers SBP.[16] Collectively, these studies confirm that SBP reductions are more consistent and clinically relevant, as SBP contributes more substantially to cardiovascular risk in middle-aged and older adults.[25-26]

Exercise timing also plays a crucial role in BP outcomes. AE performed in the afternoon or evening tends to induce greater reductions than morning sessions, possibly due to modulation of circadian rhythms and autonomic regulation.[23,27-28] Findings from the included studies also provide insights into morning–evening BP variability, which is clinically relevant given its association with cardiovascular events in hypertensive patients. One study reported that afternoon exercise produced significantly greater improvements in SBP and DBP than morning exercise, aligning with earlier evidence suggesting that morning sessions may attenuate or negate post-exercise hypotension. These studies suggest that physiological responses to exercise differ across the circadian cycle, where morning training may not produce optimal reductions in peripheral vascular resistance, thereby contributing to variability in BP responses.[23]

Mechanistically, exercise interacts with neuroendocrine factors such as cortisol and melatonin, thereby enhancing the effects of Post-Exercise Hypotension (PEH) and modulating SVR, with afternoon exercise particularly effective in reducing SBP by decreasing vascular resistance.[29-30] More detailed evidence further demonstrates that only evening exercise elicited significant reductions in both clinic and ambulatory BP, accompanied by reductions in systemic vascular resistance, decreases in total variability of systolic BP, and increases in cardiac baroreflex sensitivity. In contrast, morning exercise produced minimal SBP reductions, comparable to those in the control group, likely due to interference from peak antihypertensive medication activity during the morning hours.[31] These findings collectively indicate that variations in BP across the morning and evening are not only present but also mechanistically relevant, reinforcing the notion that afternoon or evening AE may provide superior benefits in lowering BP and modulating autonomic control in hypertensive individuals.

Across the included studies, RT alone demonstrated heterogeneous effects on BP, with outcomes varying according to modality, intensity, and training duration. Dynamic RT in Schroeder et al. did not reduce either SBP or DBP despite increasing muscular strength, likely due to the short 8-week duration and relatively normal baseline BP of participants.[20] Isometric Handgrip (IHG) protocols, proposed as a time-efficient antihypertensive strategy, also failed to reduce office or ambulatory BP in the largest sham-controlled trial to date, highlighting that previous positive IHG findings in small samples may have been influenced by placebo effects or baseline normotension.[18] In contrast, another study reported modest but clinically relevant SBP reductions (≈4 mmHg) from dynamic RT performed at 60–80% 1RM, together with improvements in endothelial function, suggesting that sufficiently intense and properly dosed RT can improve vascular health in hypertensive adults.[21] Meanwhile, one study did not isolate RT-only effects. Still, it reinforced that the BP-lowering response of RT alone is typically smaller than that of AE, and that combining RT with AE confers broader benefits on BP variability and vascular regulation.[22] None of the four studies specifically evaluated the role of breathing regulation during RT, and no evidence emerged that breathing control contributed meaningfully to hemodynamic responses. Overall, the evidence suggests that RT alone can provide selected vasculoprotective benefits. Still, its antihypertensive effects, particularly for IHG, are inconsistent and generally inferior to those of AE or CT exercise modalities.

The combination of AE and RT demonstrates variable effects, depending on the population and the measured outcomes. In older adults, combined AE-RT reduced 24-hour and nighttime BP variability more than AE alone. However, mean BP reductions were similar, except for greater nighttime DBP reduction in the AE group.[22] In prehypertensive and hypertensive adults, Combined Training (CT) improved endothelial function, with absolute SBP reductions observed in AE and RT alone, whereas the combination more influenced DBP.[21] Schroeder et al. (2019) reported that only AE-RT effectively lowered both peripheral and central DBP, whereas AE or RT alone had a limited impact, and that both AE-RT and AE or RT alone also enhanced functional capacity and cardiovascular risk profiles.[20] Pagonas et al. (2017) further confirmed that AE exerts broader antihypertensive effects than RT alone, particularly IHG, which did not significantly modulate SVR or arterial elasticity. Collectively, these findings indicate that the antihypertensive benefits primarily derive from the aerobic component, while RT serves a complementary role by improving vascular function, functional capacity, and body composition.[18,20-22]

Alternative modalities such as Tai Chi may offer comparable or even superior BP reductions in prehypertensive populations. In a 12-month intervention, Tai Chi reduced office SBP by −2.40 mmHg more than AE, with greater improvements in 24-hour SBP, nighttime SBP, heart rate, and SBP load, highlighting its potential as an effective non-pharmacological strategy.[32]

AE remains the cornerstone of non-pharmacological hypertension management, consistently lowering SBP and offering additional benefits for CRF and vascular health. Exercise timing, population characteristics, and combination with RT or dietary interventions can modulate the magnitude of BP reductions, supporting individualized prescription in clinical practice.[16,18-26,28,31-32]

Table 2. Evidence of implementation of aerobic exercise in hypertension management.

Author (Year)	Objective	Subjects	Results
Ammar (2015).[23]	To investigate the effect of AE timing (morning vs evening) on BP and lipid profile in postmenopausal overweight women with hypertension	• N = 45 postmenopausal overweight women with hypertension • Age: 49–60 years • Group allocation: ○ Group A (medication control): ACE inhibitor only, once daily for 3 months ○ Group B (morning AE + medication): morning exercise (09:00–11:00) + ACE inhibitor ○ Group C (evening AE + medication): evening exercise (16:00–18:00) + ACE inhibitor.	• SBP decreased significantly in all groups, largest reduction in evening AE (−27 mmHg, 9.6% improvement) • DBP decreased significantly in all groups, largest reduction in evening AE (−15.4 mmHg, 16.3% improvement) • HDL increased significantly, highest in evening AE (+14.2 mg/dL) • LDL decreased significantly, greatest in evening AE (−36 mg/dL) • Triglycerides decreased significantly, greatest in morning AE (−47 mg/dL, 19.5% improvement) • Total cholesterol decreased in all groups, largest reduction in evening AE (−34.8 mg/dL).
Belozo et al. (2018).[25]	To examine whether continuous AE (3x/week, total 90 min/week, 70–80% HRmax) improves BP and HR in obese hypertensive individuals	• N = 8 adults (3 men, 5 women • Age: 45.3 ± 3.9 years • Characteristics: obese (BMI 33.44 ± 8.6 kg/m²), hypertension, type 2 DM • Weight: 89.09 ± 22.0 kg; height: 1.63 ± 0.1 m	• No significant changes in BP pre- and post-15-min exercise within each session • Area under the curve analysis for SBP/DBP not significant, but effect size: systolic large (ES = 0.85), diastolic small (ES = 0.33)

Brito et al. (2019).[31]	To compare 10-week morning AE (7–9 am) vs evening AE (6–8 pm) on BP (clinic & ambulatory) and cardiovascular hemodynamic/autonomic mechanisms	• Men, 30–65 years, mild–moderate hypertension, controlled with antihypertensive medication for≥4 months • RCT, 3 groups: morning AE (n=15), evening AE (n=15), CON (stretching, n=20, divided morning/evening)	Clinic BP • Morning SBP decreased in morning AE and evening AE; only evening AE was significantly lower than control and > morning AE • Evening SBP decreased only after evening AE, significant vs morning AE and CON • DBP decreased after evening AE, not significant vs CON ABP • 24h SBP: unchanged • 24h DBP & asleep DBP decreased only after evening AE, significant vs morning AE and CON Hypotensive effect • Evening AE: Clinic SBP −5/−8 mmHg (morning/evening), DBP −3/−4 mmHg; 24h DBP & asleep DBP −3 mmHg • Morning AE: minimal SBP reduction, very small effect
Caminiti et al. (2021).[22]	To compare the 12-week AE vs AE-RT combination on 24-h BP variability and 24-h BP	• N = 55 completed protocol (AE=27, CT=28) • All on antihypertensive medication, mean age ± 68 years • Groups: AE only and AE-RT combination (CT)	• CT is more effective than AE in reducing systolic BP variability, especially 24-h and nighttime SBP variability • No significant difference for daytime systolic BP variability • Both modalities reduced SBP & DBP, except nighttime DBP decreased more with AE
Feairheller et al. (2014).[53]	To test whether 6-month AE improves vascular health in previously sedentary African-American adults, focusing on NMD, plasma NO, carotid IMT, office BP, ABP	• N = 26 • Characteristics: African-American, 40–75 y, 21 women (10 pre-, 11 postmenopause), 5 men • Mean age 53.4 ± 6.2 y, VO_2max 27 mL/kg/min, BMI 31.4 ± 5.9 kg/m²	• 6-month AE improved vascular health: ○ ↓ carotid IMT ○ ↑ FMD ○ ↓ endothelial microparticles ○ ↑ plasma NO • No significant changes in BP (office, ABP) or NMD
Gorostegi-Anduaga et al. (2018).[17]	To investigate the effects of different AE intensities/volumes (High-volume-MICT, high-volume-HIIT, low-volume-HIIT) combined with a hypocaloric diet on BP, body composition, CRF, and antihyper tensive medication use in sedentary overweight/obese adults with hypertension	• N = 175 (120 men, 55 women) • 4 intervention groups: Diet only (DO), high-volume-MICT, high-volume-HIIT, low-volume-HIIT	• All groups, including DO, had significant reductions in SBP, DBP, and MAP; no between-group differences • SBP −7–9 mmHg, DBP −3–5 mmHg; effects driven mainly by DASH diet, independent of exercise intensity/volume
He et al. (2018).[16]	To evaluate whether 12-week brisk walking reduces BP in essential hypertensive (EP) patients and attenuates the BP increase during heavy physical activity	• N = 69 postmenopausal women (55–60 y): 46 EP, 23 NBP control • Groups: EP-AE (brisk walking), EP-CON (no exercise), and NBP control	• Before intervention: EP > NBP in SBP and DBP • After intervention: ○ EP-AE: resting SBP ↓8.3 mmHg, low-intensity SBP ↓15.6 mmHg, high-intensity SBP ↓22.6 mmHg ○ DBP unchanged ○ NBP stable ○ CON showed a trend of increased BP

Li et al. (2024).[32]	To assess the effectiveness of Tai Chi vs AE in lowering BP in prehypertensive patients	• N = 342 (173 Tai Chi, 169 AE) • Age 18–65 y, prehypertension (SBP 120–139 or DBP 80–89 mmHg)	• 12-month Tai Chi decreased office SBP more than AE (−2.40 mmHg) • ABP: Tai Chi better for 24h SBP (−2.16 mmHg) and nighttime SBP (−4.08 mmHg) • Tai Chi also reduced nighttime pulse rate and SBP load compared to AE
Lopes et al. (2021).[19]	To determine whether 12-week AE is more effective than UC in lowering 24-h ambulatory BP in resistant hypertension patients	• N = 53 completed follow-up, 24 women (45%), mean age 60.1 ± 8.7 y • Groups: Exercise (AE + UC, n=26), CON (UC only, n=27)	• AE 12-week significantly reduced 24h ABP and office SBP compared with UC • Effect most pronounced on daytime BP • Increased CRF without changes in body composition/metabolic parameters • Program feasible, safe, and highly adherent
Maruf et al. (2013).[24]	To evaluate the additional effect of AE on BP reduction and antihypertensive medication use in uncontrolled essential hypertension patients already on ≥2 medications	• N = 63 (AE: 32, CON: 31), mean age AE 50.3 ± 8.4 y, CON 52.3 ± 8.1 y • Overweight (BMI 28.4 vs 26.0 kg/m²)	• No significant differences in SBP and DBP between AE + medication vs medication only • BP control rate higher in AE group (56.7% vs 35.5%) • Both groups showed SBP and DBP reduction, more pronounced in the AE group
Pedralli et al. (2020).[21]	To evaluate the effects of AE, RT, and CT on endothelial function (FMD), 24h ABP, cardiac structure/function, VO_2max, and maximal muscle strength	• Adults with prehypertension or hypertension (SBP ≥130 or DBP ≥80 mmHg), N = 37 (21F,16M) • Groups: AT (n=14), RT (n=14), CT (n=14)	• FMD increased significantly in all groups: AE +3.2%, RT +4.0%, CT +6.8% (Cohen's d 0.6–0.9), no between-group differences • 24h ABP: SBP ↓ in AE (−5.1 mmHg) and RT (−4.0 mmHg), not in CT; DBP ↓ in CT (−3.2 mmHg) • Daytime BP reduction more pronounced; night unchanged • VO_2max ↑ in AE and RT; EF ↑ in AE; muscle strength ↑ in RT; waist circumference ↓ in AE and CT; triglycerides ↓ in all groups
Pagonas et al. (2017).[18]	To compare the effects of AE and IHG training on office BP, 24h ABP, systemic vascular resistance, and arterial elasticity	• N = 75 hypertensive patients, groups: AE (n=25), IHG (n=25), sham training (n=25) • 9 dropouts, per-protocol analysis N = 66	• Only AE significantly reduced office BP and 24h ABP • IHG and sham training had no antihypertensive effect
Schroeder et al. (2019).[20]	To compare the effects of AE, RT, and CT on peripheral & central BP and CVD risk factors	• N = 69 (45–74 y, 61% female), mild–moderate hypertension or high BP (SBP 120–149 / DBP 80–99 mmHg, no antihypertensives), overweight/obese (BMI 25–40), sedentary • Baseline: age 58 ± 7 y, BMI 32.4 ± 5.2, SBP 131 ± 13, DBP 81 ± 9	• BP: CT: DBP ↓4 mmHg (peripheral & central, significant) • AT and RT: no significant SBP/DBP change • AT + CT: HR ↓2 bpm vs control ($p<0.02$) • Body composition & CVD risk: AT best for BMI, weight, fat mass ↓; RT: waist −1.7 cm, TG −26 mg/dL; CT: lean body mass +0.8 kg, ↑ CRF & strength; composite CVD risk score ↓ more than control

ABP, ambulatory blood pressure; ACE, angiotensin-converting enzyme; AE, aerobic exercise; BMI, body mass index; BP, blood pressure; CON, control; CRF, cardiorespiratory fitness; CT, combined training; CVD, cardiovascular disease; DASH, dietary approaches to stop hypertension; DBP, diastolic blood pressure; EF, ejection fraction; EP, essential hypertension; EP-AE, essential hypertension-aerobic exercise; EP-CON, essential hypertension-control; ES, effect size; FMD, flow-mediated dilatation; HDL, high-density lipoprotein; HIIT, high-intensity interval training; HR, heart rate; HRmax, maximum heart rate; IHG, isometric hand grip; IMT, intima-media thickness; LDL, low-density lipoprotein; MICT, moderate-intensity continuous training; NBP, normal blood pressure; NMD, nitroglycerin-mediated dilation; NO, nitric oxide; RCT, randomized controlled trial; SBP, systolic blood pressure; SHT, UC, usual care; VO2max, maximum oxygen uptake.

Evidence of The Effects of AE in Combination with a Hypocaloric Diet

The study by Gorostegi-Anduaga et al. (2018) provides valuable insights into the interaction between AE and dietary interventions in the management of hypertension. In this trial, all intervention groups, including those receiving only a hypocaloric diet and those combining the diet with various forms of AE (high-volume Moderate Intensity Continuous Training [MICT], high-volume HIIT, and low-volume HIIT), demonstrated significant reductions in SBP and DBP as well as MAP. The magnitude of SBP reduction (approximately −7 to −9 mmHg) and DBP reduction (approximately −3 to −5 mmHg) was similar across groups, indicating that dietary factors played a dominant role in lowering BP, regardless of exercise intensity or volume.[17]

These findings align with previous evidence showing that dietary interventions, particularly hypocaloric approaches based on the Dietary Approaches to Stop Hypertension (DASH) pattern, exert strong antihypertensive effects and can serve as a primary non-pharmacological strategy. In this context, AE offers additional benefits, notably improvements in CRF, body composition, and potential reductions in antihypertensive medication requirements. However, when combined with diet, the relative contribution of exercise to BP reduction may appear less pronounced, given the predominant effect of nutritional intervention.[17]

This evidence highlights the value of a multimodal approach to hypertension management, where a healthy diet serves as the core component, and AE provides complementary benefits beyond BP control, including improvements in functional capacity, metabolism, and overall cardiovascular health.[17,33]

Mechanistic Links Between AE and BP Reduction

AE has been shown to reduce BP through a complex network of central and peripheral adaptations. A key pathway involves improvements in endothelial function: repeated increases in limb blood flow during rhythmic, large-muscle activity elevate shear stress along the arterial intima, thereby upregulating endothelial Nitric Oxide Synthase (eNOS) and enhancing Nitric Oxide (NO) bioavailability. This adaptation augments vasodilatory capacity and progressively lowers systemic vascular resistance both at rest and during submaximal effort.[34-36] Regular AE also improves arterial compliance and reduces vascular stiffness, effects that are strongly linked to enhanced baroreflex sensitivity and greater hemodynamic stability.[37] Moreover, exercise training downregulates sympathetic vasomotor tone while shifting autonomic balance toward parasympathetic dominance, thereby dampening excessive sympathetic drive, a mechanism supported by both experimental and clinical studies.[38] Collectively, these vascular and autonomic adaptations explain the sustained reductions in BP observed following long-term AE.

Beyond vascular adaptations, metabolic changes play a crucial role in mediating the BP-lowering effects of AE. Regular AE promotes skeletal muscle capillarization and mitochondrial biogenesis, enhancing oxidative capacity, increasing VO2max, and improving insulin sensitivity.[39-41] Improved glycemic control attenuates hyperinsulinemia-driven sympathetic activation and renal sodium retention, mechanisms closely linked to the pathophysiology of hypertension.[42] In parallel, AE induces favorable shifts in adipokine secretion, reduces systemic inflammation, and mitigates oxidative stress, all of which contribute to vascular protection.[43] Exercise also exerts beneficial effects on lipid metabolism, typically characterized by higher HDL cholesterol, lower triglycerides, modest reductions in LDL cholesterol, and preferential loss of visceral adiposity, thereby lowering hemodynamic load.[44] Although weight reduction independently reduces BP, consistent evidence shows that the antihypertensive benefits of AE often occur even in the absence of significant weight loss, highlighting the importance of weight-independent hemodynamic and autonomic adaptations.[45-46]

The BP response is also exercise-dose dependent. Higher volumes of moderate-to-vigorous AE generally produce greater reductions, up to a ceiling beyond which additional time yields diminishing returns. Interval formats can create strong shear stimuli in shorter sessions, whereas continuous moderate-intensity work may be preferable for adherence in some patients. Program personalization, such as balancing intensity, volume, enjoyment, and orthopedic tolerance, is central to sustained benefit.[47]

Factors Influencing the BP Response to AE

The hypotensive effects of AE are multifactorial and are shaped by individual characteristics, clinical conditions, vascular properties, and exercise parameters. Key individual determinants include age, sex, BMI, and baseline physical activity, while clinical factors encompass hypertension severity, pharmacological status, and the presence of resistant hypertension. Vascular factors, such as arterial stiffness, also modulate the response, whereas exercise type, duration, and intensity dictate the magnitude of BP reduction.[9,48-51]

Acute and chronic AE consistently demonstrate significant reductions in BP. Carpio-Rivera et al. (2016) reported that a single AE session lowers SBP by approximately −4.8 mmHg and DBP by −3.2 mmHg, independent of intensity, duration, or participant characteristics. This PEH has clinically meaningful implications, as a 2 mmHg decrease in SBP can reduce stroke mortality by 6% and coronary heart disease mortality by 4%.[9]

Individual responses are modulated by physiological and demographic factors. Men typically exhibit greater reductions in SBP than women, potentially due to differences in autonomic tone and baroreflex sensitivity, with menstrual cycle phases also influencing outcomes. Age and BMI further modify the hypotensive response; aging increases arterial stiffness and microvascular remodeling, attenuating BP reductions, whereas lower BMI is associated with greater SBP decreases, consistent with obesity-related sympathetic overactivity, endothelial dysfunction, and chronic inflammation.[9,48]

Arterial stiffness is a critical determinant of exercise-induced modulation of BP. Elevated pulse wave velocity and augmentation index, common in aging and hypertensive populations, precede isolated systolic hypertension. AE that enhances arterial elasticity can therefore slow vascular stiffening and the progression of age-related hypertension.[48]

Exercise program characteristics further influence PEH. Carpio-Rivera et al. (2016) found that jogging is more effective than walking in lowering both SBP and DBP, while longer duration and progressive-intensity protocols yield larger reductions. This underscores that total exercise load, rather than intensity alone, primarily determines hemodynamic response.[9]

Genetic factors appear to have minimal impact. A meta-analysis by Bruneau Jr et al. (2016) indicated that sample characteristics, such as age, sex, BMI, and baseline BP, accounted for more than half of the variability in BP response. In contrast, gene polymorphisms contributed less than 1%. Only one genetic factor variant showed a modest association with DBP, highlighting the dominant role of non-genetic factors.[49]

Clinical characteristics also shape exercise outcomes. Nascimento et al. (2017) noted that patients with resistant hypertension may display variable responses, including potential adverse effects.[50] Similarly, Saco-Ledo et al. (2021) demonstrated that a single AE session can lower 24-hour ambulatory BP in both medicated and unmedicated hypertensive patients by reducing sympathetic activity, increasing local vasodilator release, and eliciting vascular adaptations. Larger reductions are generally observed in non-medicated individuals, likely due to interactions between pharmacotherapy and exercise effects.[50-51] Overall, these findings reinforce the need for individualized exercise prescriptions in hypertension management, accounting for personal, clinical, vascular, and program-specific factors to optimize the BP-lowering effects of AE.

Safety Issues in the Implementation of AE in Hypertension Management

AE is generally safe for individuals with hypertension when introduced progressively and tailored to their comorbid status. Clinical guidelines recommend halting exercise if abnormal physiological responses occur, such as inappropriate heart rate response to intensity, a drop in SBP ≥20 mmHg with increasing workload, a rise in DBP ≥15 mmHg, new or worsening angina, severe dyspnea, pallor or cyanosis, dizziness, confusion, or leg ischemia consistent with claudication. In such cases, training should be paused until medical evaluation and program adjustment are completed.[10,52]

Musculoskeletal injuries are the most common exercise-related complications. These are usually linked to sudden increases in intensity or volume, insufficient recovery, biomechanical issues, or prior injuries. Gradual progression, cross-training, appropriate footwear, and inclusion of resistance exercises to strengthen key kinetic chains effectively reduce injury risk and support sustained AE participation. Serious cardiovascular events during moderate-intensity AE are rare under supervision, and pre-participation screening further minimizes risk.[10]

Evidence from clinical trials confirms the safety of structured AE programs. In a 16-week randomized controlled trial, no major complications or cardiac events occurred, indicating that both MICT and HIIT can be safely implemented with appropriate supervision in patients receiving pharmacological therapy.[17] Similarly, a 12-month study comparing AE and Tai Chi reported excellent tolerability with no serious adverse events, demonstrating that structured exercise is feasible even in individuals with cardiovascular risk factors.[32]

Other studies reinforce these findings. In a trial by Lopes et al. (2021), adherence to the exercise program was high (98.8% of sessions attended), with only minor events such as transient dizziness or mild musculoskeletal discomfort reported. No participants withdrew due to adverse events, highlighting that careful monitoring and individualized adjustments can ensure safety.[19] Maruf et al. (2013) reported similar outcomes in middle-aged, overweight hypertensive adults, with no adverse events during the intervention and adherence rates of approximately 77%.[24] Pedralli et al. (2020) confirmed that both AE and CT protocols were feasible and safe for individuals with elevated BP and low baseline physical activity levels over 8 weeks.[21]

Even in studies involving different exercise modalities, safety remained consistent. Pagonas et al. (2017) showed that AE and IHG training were well tolerated, with no serious complications, although some participants discontinued due to changes in medication or adherence challenges.[18] Schroeder et al. (2019) observed excellent compliance and tolerability across all training types, with participants consistently achieving prescribed intensity and volume, and no adverse events reported over 8 weeks.[20]

Overall, these findings demonstrate that AE, whether delivered as MICT, HIIT, or in combination with RT or alternative exercises like Tai Chi, is safe and well-tolerated for individuals with hypertension when programs are supervised, progressively implemented, and adapted to participant capacity. High adherence rates and minimal minor events further support the feasibility and clinical relevance of AE as a cornerstone of non-pharmacological hypertension management.[32]

Conclusion

AE is an effective and safe non-pharmacological strategy for the prevention and management of hypertension. Consistently, it produces greater reductions in SBP than in DBP through mechanisms that enhance endothelial function, improve autonomic regulation, and reduce SVR, with benefits that are independent of weight loss. The effectiveness of the intervention is influenced by factors such as age, sex, BMI, medication status, and timing of exercise sessions. Combining AE with RT or a hypocaloric diet further enhances cardiometabolic benefits, although BP reduction is primarily driven by the aerobic component. Moreover, AE exhibits a favorable safety profile, with minimal risk of adverse effects and high adherence rates. These findings underscore that AE is a key component in hypertension management and can be readily integrated into routine clinical practice.

List of Abbreviations

ABP	Ambulatory Blood Pressure
AE	Aerobic Exercise
BMI	Body Mass Index
BP	Blood Pressure
CAD	Coronary Artery Disease
CO	Cardiac Output
CRF	Cardiorespiratory Fitness
CT	Combined Training
CVD	Cardiovascular Disease
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic Blood Pressure
eNOS	Endothelial Nitric Oxide Synthase
FMD	Flow-Mediated Dilatation
HDL	High-Density Lipoprotein
HIIT	High-Intensity Interval Training
HR	Heart Rate
HRmax	Maximum Heart Rate
HRR	Heart Rate Reserve
IHG	Isometric Handgrip
IMT	Intima-Media Thickness
LDL	Low-Density Lipoprotein
MAP	Mean Arterial Pressure
MICT	Moderate-Intensity Continuous Training
MVC	Maximal Voluntary Contraction
NO	Nitric Oxide
PEH	Post-Exercise Hypotension
RCT	Randomized Controlled Trial
RPE	Rating of Perceived Exertion
RT	Resistance Training
SBP	Systolic Blood Pressure
SV	Stroke Volume

SVR	Systemic Vascular Resistance
VO_2max	Maximal Oxygen Uptake
VO_2peak	Peak Oxygen Uptake

Ethical Clearance

Not applicable.

Publication Approval

All authors are consent to the publication of this manuscript.

Authors Contributions

(a) Conception and design, or analysis and interpretation of data; WN and AN (b) Drafting the article or revising it critically for important intellectual content; WN, AN, and TP (c) Final approval of the version to be published; WN, AN, and TP.

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