Indonesian Journal of Global Health Research
Volume 8 Number 4, August 2026
e-ISSN 2715-1972; p-ISSN 2714-9749
https://jurnal2.globalhealthsciencegroup.com/index.php/IJGHR

ISOMETRIC EXERCISE TRAINING AND ITS EFFECTS ON CARDIOVASCULAR HEALTH INDICATORS IN PREHYPERTENSIVE INDIVIDUALS

Ari Wibawa[1]*, Bagus Komang Satriyasa[2], I Gusti Putu Suka Aryana[3], Luh Putu Ratna Sundari[4]
[1]Department of Physiotherapy, Faculty of Medicine, Universitas Udayana, Jl. P.B. Sudirman, Dangin Puri Klod, Denpasar Barat, Denpasar, Bali 80232, Indonesia
[2]Department of Pharmacology and Therapy, Faculty of Medicine, Universitas Udayana, Jl. P.B. Sudirman, Dangin Puri Klod, Denpasar Barat, Denpasar, Bali 80232, Indonesia
[3]Department of Internal Medicine, Faculty of Medicine, Universitas Udayana, Jl. P.B. Sudirman, Dangin Puri Klod, Denpasar Barat, Denpasar, Bali 80232, Indonesia
[4]Department of Physiology, Faculty of Medicine, Universitas Udayana, Jl. P.B. Sudirman, Dangin Puri Klod, Denpasar Barat, Denpasar, Bali 80232, Indonesia
*ariwibawafk@unud.ac.id

ABSTRACT

Hypertension is a chronic condition marked by consistently elevated blood pressure, defined as ⩾130/80 mmHg by the ACC/AHA. It is a major global health issue contributing to cardiovascular disease and mortality. As of 2021, the WHO reported 1.28 billion cases worldwide. Current strategies for managing hypertension emphasize lifestyle interventions alongside pharmacological treatments. Isometric exercise training has gained attention due to its potential in reducing blood pressure and improving vascular health. This study aims to evaluate the physiological mechanisms underlying isometric exercise training's impact on cardiovascular health indicators, focusing on NO production, BP, and RPP. This article was constructed using a literature review methodology. Scientific articles were sourced from PLoS ONE, Science Direct, PubMed, and Google Scholar. Articles published within the last 5 years were included to ensure the review's relevance and accuracy, resulting in 53 included studies. Isometric exercise training involves muscle contractions without changing muscle length or joint movement. It strengthens muscles, improves endurance and joint stability by activating type I and II fibers, enhancing neuromuscular coordination. This training triggers cardiovascular and musculoskeletal adaptations, such as nitric oxide release from hypoxia and shear stress, and activates the parasympathetic system to lower blood pressure. It reduces resting blood pressure, afterload, and rate pressure product, improving cardiac efficiency. Movements like wallsquats engage major lower limb muscles, with deeper knee flexion increasing force and stimulating endothelial NO production. Isometric exercise training is a low-risk, effective intervention that improves cardiovascular health and reduces blood pressure, highlighting its potential as a non-pharmacological approach for hypertension management.

Keywords: cardiovascular health indicators; isometric exercise training; nitric oxide; rate pressure product; resting blood pressure

How to cite (in APA style)
Wibawa, A., Satriyasa, B. K., Aryana, I. G. P. S., & Sundari, L. P. R. (2026). Isometric Exercise Training and Its Effects on Cardiovascular Health Indicators in Prehypertensive Individuals. *Indonesian Journal of Global Health Research*, *8*(4), 1079–1090. https://doi.org/10.37287/ijghr.v8i4.1810.

INTRODUCTION

Hypertension, a chronic condition marked by persistently elevated blood pressure. The ACC/AHA define it as SBP ≥130 mmHg and/or DBP ≥80 mmHg (Kabootari, 2023). In 2021, the WHO reported 1.28 billion global cases, with Southeast Asia ranking third highest at 25% of its population (WHO, 2021). Prevalence is 31.6% at ages 31–44, 45.3% at 45–54, and 55.2% at 55–64, showing it affects not only older adults but also people in their productive years (Kemkes, 2019). Hypertension is a major modifiable risk factor for cardiovascular diseases such as coronary artery disease, stroke, and heart failure, which are leading causes of death and disability (Tackling, 2025). Prehypertension is defined as blood pressure above normal (SBP 120–139 mmHg or DBP 80–89 mmHg) but not yet meeting hypertension criteria. Individuals with prehypertension have a 2–3× higher risk of developing hypertension and greater risk of cardiovascular disease and target organ damage, including atherosclerosis, microvascular injury, coronary artery calcification, vascular remodeling, and left ventricular hypertrophy (Rubio, 2013; Zhao, 2025). Progression to hypertension usually occurs within 28 months–5 years. Prehypertension affects 25–50% of adults worldwide, with annual incidence rates of 8–20% in short-term and 4–9% in long-term studies. About 28,5% develop hypertension within two years, – more frequent among men than in women – and among every 10 affected adults, intensive lifestyle modification is needed, with 4–6 likely requiring antihypertensive therapy to prevent progression (Li, 2023). Progression from prehypertension to hypertension is complex and partly driven by oxidative stress. Blood pressure is regulated by the central nervous system through sympathetic activity, vascular tone, peripheral resistance, and the renin–angiotensin–aldosterone system (RAAS) (Saxena, 2018). Chronic RAAS activation and sustained angiotensin II production promote hypertension by causing inflammation, fibrosis, and end-organ damage through oxidative stress (Arendshorst, 2024).

Hypertension involves endothelial dysfunction from an imbalance between nitric oxide (NO) and reactive oxygen species (ROS), leading to vasoconstriction, inflammation, thrombosis, and vascular wall proliferation. Blood flow–induced shear stress regulates endothelial function, NO production, and ROS formation (Gallo, 2022). Normally, laminar flow increases eNOS activity and NO, while in hypertension, oscillatory flow increases ROS and endothelial damage. Different shear stress patterns distinctly affect ROS–NO signaling and vascular remodeling (Amponsah, 2023). NO is produced by three nitric oxide synthase (NOS) isoforms: nNOS (brain), iNOS (inflammation), and eNOS (cardiovascular vasodilation) (Reis, 2017). Excess interaction between NO and superoxide reduces NO availability and increases oxidative stress, causing vascular dysfunction (Shaito, 2022). Chronic inflammation further increases ROS and endothelial injury and is linked to hypertension, while RAAS activation also promotes oxidative stress–driven vascular inflammation (Wang, 2026).

Angiotensin II, a central RAAS mediator, activates oxidases and proinflammatory pathways that damage the endothelium. NOX2 and NOX4 contribute to cardiovascular disease, including hypertension, by generating ROS and promoting endothelial dysfunction. Neutrophil activation further increases ROS and inflammatory cytokines, amplifying oxidative stress and inflammation—key drivers of hypertension (Xia, 2025). Because endothelial function, NO bioavailability, oxidative stress, and blood pressure are closely linked, interventions that modulate these pathways may offer important preventive benefits. Therefore, this study aims to evaluate the physiological mechanisms by which isometric exercise training affects cardiovascular health, focusing on nitric oxide (NO) production, blood pressure (BP), and rate pressure product (RPP).

METHOD

This article was developed using a narrative literature review methodology. Relevant scientific articles were identified through searches of PLoS ONE, ScienceDirect, PubMed, and Google Scholar. The keywords used included "Cardiovascular Health Indicators," "Isometric Exercise Training," "Nitric Oxide," "Rate Pressure Product," and "Resting Blood Pressure," combined using Boolean operators such as AND/OR to refine the search. Articles were restricted to those published within the last 5 years to ensure contemporary relevance. Only peer-reviewed publications available in full text and written in English were included. Studies were assessed based on relevance, methodological clarity, and consistency of findings. This study includes 53 studies, encompassing sources from various scientific journals. The selected literature was then synthesized using a narrative approach to summarize key mechanisms and outcomes related to isometric exercise, nitric oxide regulation, and cardiovascular health indicators.

RESULT

Prehypertension

The Seventh Report of the Joint National Committee (JNC 7) introduced the term prehypertension, defined as systolic blood pressure (SBP) of 120–139 mmHg and/or diastolic blood pressure (DBP) of 80–89 mmHg. This condition requires lifestyle modification to prevent progression to hypertension and reduce associated cardiovascular risks (Lin, 2024). Across the reviewed literature, several factors consistently contribute to the development of prehypertension and early-stage hypertension. Age is a major non-modifiable determinant, with prevalence rising from 0.3% at age 25 to 37% at age 65 (Whelton, 2017). Family history also plays a significant role; a 54-year longitudinal study demonstrated that having both parents with hypertension is a strong predictor of future blood pressure elevation. Lifetime risk estimates from ACC/AHA show the probability of developing hypertension by age 85 to be highest among African Americans (93%), followed by Hispanics (92%), Caucasians (86%), and Chinese individuals (84%) (Falkner, 2024).

Obesity is repeatedly identified as a central modifiable factor, with both BMI and waist-hip ratio positively correlated with blood pressure. Abdominal obesity and early-onset weight gain significantly elevate the risk of hypertension (Sun, 2022). Dietary and lifestyle contributors, such as high sodium intake, alcohol consumption, and low potassium intake, also increase risk, whereas higher levels of physical activity exhibit a protective effect (Whelton, 2017). Secondary hypertension may arise from medication use—including oral contraceptives, corticosteroids, decongestants, stimulants, atypical antipsychotics, and illicit drugs—or from underlying medical conditions such as renal disease, hyperaldosteronism, renovascular disorders, obstructive sleep apnea, pheochromocytoma, hyperthyroidism, and Cushing's syndrome (Hegde, 2023).

Prehypertension Pathophysiology

Prehypertension arises from a convergence of endothelial dysfunction, oxidative stress, inflammation, and autonomic dysregulation. Activation of angiotensin II type 1 receptors (AT1R) stimulates NADPH oxidase–driven production of reactive oxygen species (ROS), which impairs nitric oxide (NO) bioavailability and disrupts vascular permeability. These oxidative processes activate MAPK pathways and transcription factors such as NF-κB and AP-1, promoting proinflammatory cytokine release (TNF-α, IL-1β, IL-6) (Alim, 2023). Parallel PLA2–COX-2 activation increases PGE2 synthesis, further amplifying vascular inflammation (Xiao, 2026). TLR4 activation reinforces this inflammatory state by upregulating iNOS, COX-2, ICAM-1, and VCAM-1 (Turpin, 2026).

Peripheral inflammation extends centrally by altering the blood–brain barrier, activating microglia and disrupting neurovascular regulation (Li, 2025). The resulting neuroinflammation enhances sympathetic output. This, combined with overactive muscle reflexes (mechanoreflex and metaboreflex), central command, and impaired baroreflex sensitivity, contributes to sustained elevations in vascular tone and blood pressure (Kounoupis, 2020). These molecular and neural mechanisms create a self-reinforcing cycle of vascular inflammation, sympathetic overactivity, and endothelial dysfunction—key abnormalities underlying the progression of prehypertension to established hypertension.

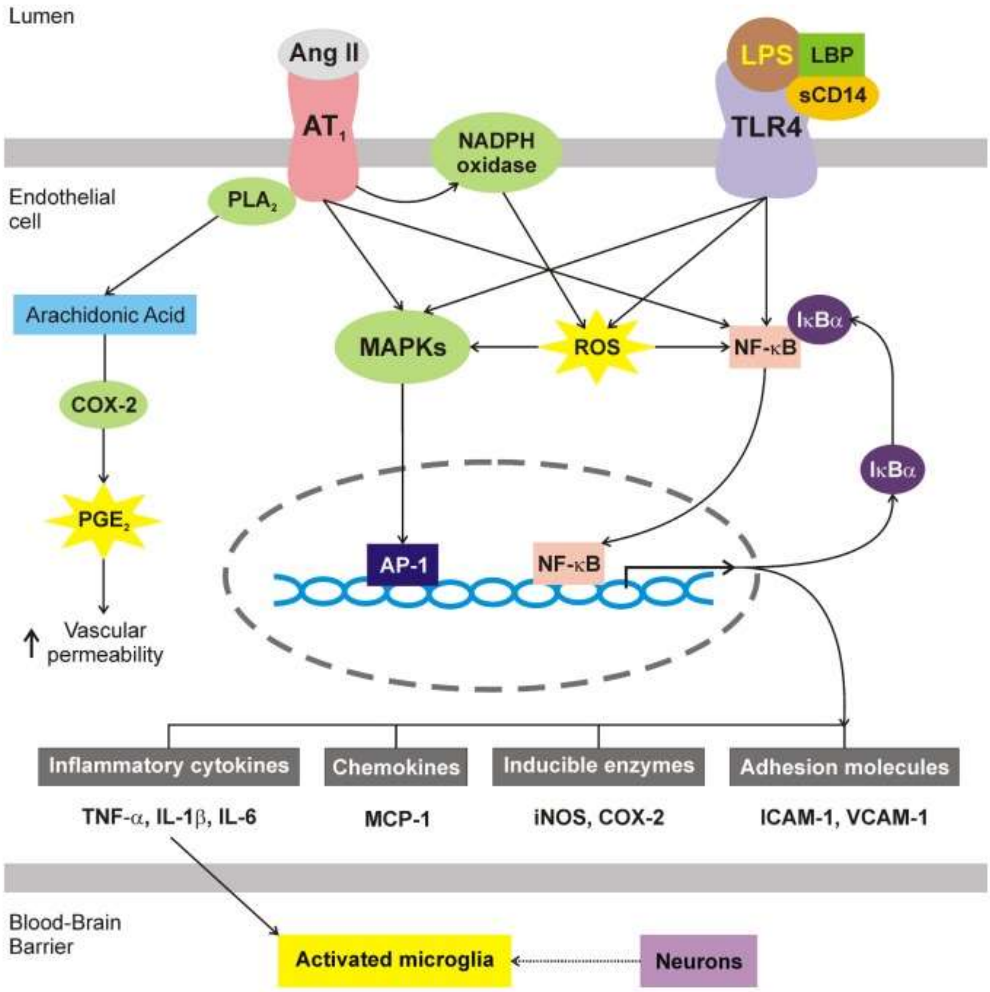

Figure 1: Molecular Pathophysiology of Hypertension (Benicky, 2009)

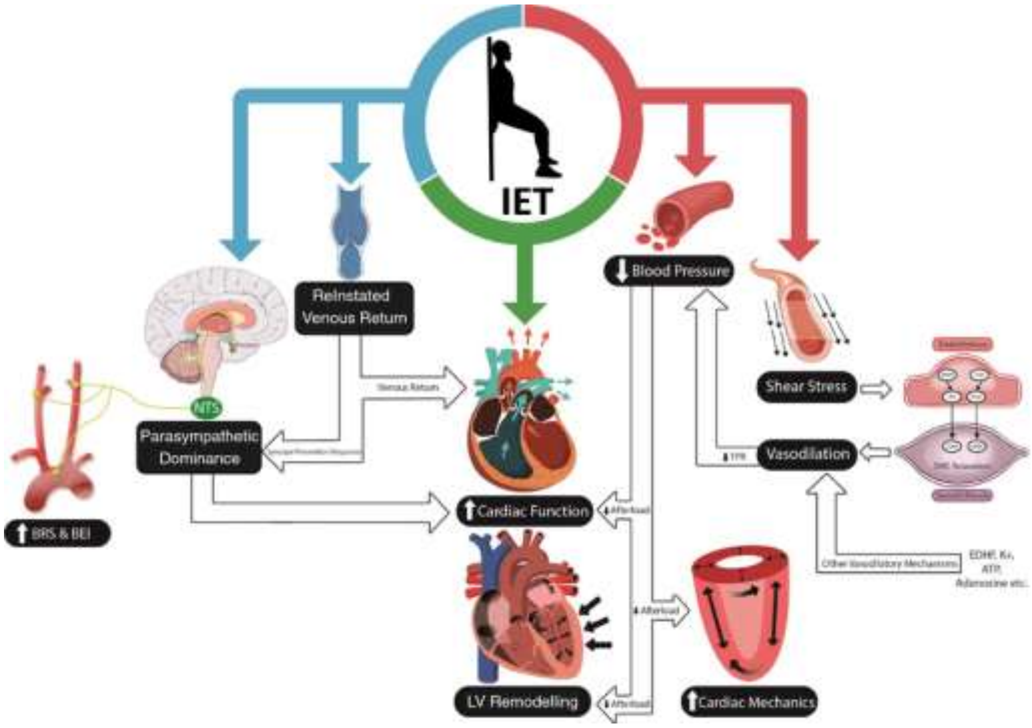

Figure 2. Acute Physiological Response Diagram Post-Isometric Exercise (Edwards, 2024).

DISCUSSION

Isometric Exercise Training

Isometric exercises are static muscle contractions performed without substantial changes in muscle length or joint movement, providing a controlled neuromuscular stimulus that enhances strength, endurance, and joint stability (Widodo, 2022). Unlike dynamic training, which involves repetitive concentric and eccentric actions, isometric exercise relies on maintaining tension against an immovable load, enabling the recruitment of a high proportion of both type I and type II muscle fibers depending on contraction intensity and duration. This sustained contraction increases intramuscular tension and activates mechanoreceptors—including muscle spindles and Golgi tendon organs—enhancing neuromuscular communication and promoting more efficient motor unit recruitment and synchronization over time (Stožer, 2020). These adaptations contribute not only to improved muscle strength and joint stabilization but also to broader physiological benefits across the cardiovascular and musculoskeletal systems (Aslam, 2025).

During static contractions, prolonged muscle tension compresses intramuscular blood vessels, producing a transient hypoxic environment that stimulates the release of nitric oxide (NO), a key vasodilatory molecule that improves endothelial function, reduces oxidative stress, and supports angiogenesis. These vascular adaptations enhance endothelial health and contribute to long-term improvements in blood pressure regulation. Neural adaptations also play a significant role in the effects of isometric training. Sustained contractions improve the central nervous system's ability to recruit and synchronize motor units, supporting tasks that require static control such as postural stability and load-bearing maintenance (Lin, 2024). Cardiovascular adaptations additionally emerge through improved autonomic regulation, with enhanced parasympathetic activity contributing to reductions in resting blood pressure and improved cardiovascular control (Mettler, 2016).

Isometric exercise influences cardiovascular homeostasis through several mechanisms. Shear stress generated by increased blood flow during relaxation phases stimulates endothelial release of vasodilatory mediators such as endothelium-derived hyperpolarizing factor (EDHF) and ATP, lowering vascular resistance and improving arterial compliance. The nucleus tractus solitarius (NTS) serves as a central processing site for baroreceptor afferents and modulates autonomic output to maintain blood pressure stability, and isometric training appears to enhance this baroreflex-mediated control (Lea, 2024; Luksha, 2009). Collectively, these hemodynamic and neural adaptations contribute to improved vascular tone, enhanced cardiac efficiency, and long-term reductions in resting blood pressure.

Standardized isometric training protocols commonly involve maintaining fixed joint angles under static load. One widely used protocol includes holding a knee angle of 135° for 2 minutes and reducing the angle by 10° every 2 minutes until reaching 95°, or until fatigue prevents maintenance within ±5° of the target angle (Cohen, 2023).

Participants capable of sustaining 95° for 2 minutes typically begin training at 125° and progress downward over time, while others begin at 135° and follow a similar progressive structure. Training is generally performed 3 days per week for 4 weeks, totaling 12 sessions, with each session comprising four 2-minute wall squat contractions separated by 2-minute rest periods (Cohen, 2023). Evidence consistently demonstrates that Isometric Resistance Training (IRT) reduces systolic blood pressure (SBP) by an average of 7.35 mmHg and diastolic blood pressure (DBP) by 3.29 mmHg, with joint angle changes—particularly at 10° intervals—modulating physiological responses such as heart rate and blood pressure (Constantinou, 2022). The magnitude of these reductions is comparable to those achieved with a single antihypertensive medication. This effect is attributed to improved endothelial NO availability, enhanced baroreflex sensitivity, and reductions in vascular resistance, positioning IRT as an effective, non-pharmacological intervention for lowering blood pressure and improving overall cardiovascular health (Baffour, 2023).

Biomechanics of Isometric Exercise Training

The biomechanics of isometric exercise training involve coordinated interactions across the hip, knee, and ankle joints to maintain static postures under load. The hip joint, a multiaxial synovial ball-and-socket joint, permits extensive mobility including flexion, extension, abduction, adduction, and rotational movements. During isometric tasks such as the wall-squat, the gluteus maximus and hamstring muscles serve as primary stabilizers, contributing to load distribution and maintenance of posture. The gluteus maximus operates near its optimal sarcomere length to sustain force, while the hamstrings exhibit maximal activation within the 10°–70° range of hip flexion, enabling efficient stabilization and resistance to posterior pelvic tilt (Straub, 2024). At the knee joint, the tibiofemoral and patellofemoral articulations function collectively as a modified hinge joint capable of flexion, extension, and limited axial rotation. During isometric contraction, the quadriceps femoris group generates substantial tension to resist knee flexion, acting concentrically to maintain joint position while the hamstrings provide complementary stabilization. This co-activation reduces anterior tibial translation and mitigates shear forces on the anterior cruciate ligament (ACL), enhancing joint integrity under static load (Biscarini, 2022).

The ankle complex, composed primarily of the talocrural and subtalar joints, contributes to postural control through dorsiflexion and plantar flexion. The triceps surae—particularly the gastrocnemius and soleus muscles—maintains dynamic ankle stability by counteracting valgus forces and supporting the kinetic chain during static lower-limb loading. Their sustained activation promotes effective force transfer from the ground through the lower limb, ensuring biomechanical efficiency across the entire kinetic chain. This integrated synergy of the hip, knee, and ankle joints allows for efficient stabilization, force distribution, and postural endurance during isometric exercise training (Gullu, 2024; Yavuz, 2024).

Phases of Movement in Isometric Exercise Training

In the ascending phase of the training protocol, the participant begins by standing with feet shoulder-width apart and leaning against a wall, maintaining a knee angle of 135°/25° flexion and an ankle positioned at 0° dorsiflexion (Kothurkar, 2022). The lumbopelvic region must remain neutral, with the lumbar curvature held flat against the wall to ensure proper spinal alignment (Atahan, 2025). At this angle, the exercise constitutes a closed kinetic chain movement requiring coordinated control of the knee, hip, and lumbar segments while maintaining a constant heel-to-wall distance. During this phase, the quadriceps muscle group—including the vastus lateralis, vastus medialis, vastus intermedius, and rectus femoris—contracts synergistically with the gluteus maximus. Electromyographic studies have shown that the quadriceps are primarily activated during the downward phase, whereas in the upward phase, the vastus medialis oblique (55–66% MVIC) and gluteus maximus (56–86% MVIC) exhibit higher activation levels (Glaviano, 2022).

The descending phase of the isometric protocol progresses through increasing knee flexion angles of 125°/35°, 115°/45°, 105°/55°, and 95°/65°, with ankle dorsiflexion maintained at 0°.40 As knee flexion deepens, the hip joint range of motion proportionally increases according to the hip–lumbo–pelvic rhythm, resulting in greater engagement of the hamstrings, gluteus maximus, adductor magnus, and quadriceps. An upright trunk posture enhances activation of the rectus femoris due to its biarticular nature, although the vastus muscles—particularly the vastus lateralis and medialis—produce approximately 50% greater force output (Lee, 2022). The rectus femoris simultaneously functions as a hip flexor and knee extensor, thereby undergoing minimal length change throughout the squat movement.

Joint loading parameters during the descending sequence demonstrate substantial variations in force across the knee structures. Maximum force on the quadriceps tendon peaks at approximately 8,000 N at 130° of knee flexion, subsequently decreasing to 5,500 N at 60° and further to 3,500 N at 30°. Correspondingly, quadriceps tendon loading reduces from 2,000 N at deeper flexion angles to lower values as extension increases. The patellar tendon withstands forces ranging from 6,000 N at high flexion to 2,000 N at shallower angles.

Isometric Exercise Training Enhances NO Levels

Isometric exercise training promotes increased nitric oxide (NO) availability through several interconnected vascular and neural mechanisms. During isometric contractions, blood flow within the working muscles becomes temporarily restricted, increasing intramuscular pressure. When the contraction ceases, blood rapidly re-enters the muscle—an effect known as reactive hyperemia—which generates substantial shear stress on the endothelial lining of blood vessels. Shear stress, a frictional force arising from the movement of blood across endothelial surfaces, is a primary stimulus for endothelial NO release, driving vasodilation and reducing total peripheral resistance (TPR) (Saxena, 2018). The magnitude of this shear stress depends on factors such as blood velocity and viscosity, and its increase serves as a direct signal for endothelial cells to activate nitric oxide synthase (eNOS), thereby enhancing NO production. Following the contraction phase, the recovery period is characterized by heightened parasympathetic nervous system activity, mediated primarily through the vagus nerve. This parasympathetic rebound promotes the release of acetylcholine, which stimulates both eNOS and neuronal nitric oxide synthase (nNOS), further increasing NO synthesis (Ma, 2023). This cholinergic activation not only aids in restoring cardiovascular homeostasis—such as reducing heart rate—but also enhances vasodilatory capacity via NO-dependent pathways (Baffour, 2023).

Reactive hyperemia occurring in the recovery phase adds an additional stimulus for NO production. The sudden surge in blood flow elevates shear stress within the vascular endothelium, reinforcing eNOS and nNOS activation and amplifying NO release (Sweet, 2024). Through the combined effects of increased shear stress and parasympathetic-mediated acetylcholine release, isometric exercise training significantly enhances NO bioavailability. This increase in NO contributes to improved vascular function, reduced TPR, and overall cardiovascular health adaptations associated with regular isometric training (Nurhasanah, 2023).

Isometric Exercise Training Reduces RPP Levels

Isometric exercise training has been shown to reduce the rate–pressure product (RPP), a key index of myocardial oxygen consumption and cardiac workload. A reduction in RPP reflects decreased myocardial oxygen demand, achieved primarily through reductions in heart rate (HR) and afterload. Afterload represents the pressure the left ventricle must overcome to open the aortic valve, which is largely determined by arterial resistance (Kounoupis, 2020). Conditions such as hypertension or arterial narrowing elevate afterload, forcing the heart to work harder and increasing oxygen consumption (Jozwiak, 2024).

Following isometric exercise training, meaningful cardiovascular adjustments occur, most notably a reduction in total peripheral resistance (TPR), which is the systemic resistance encountered by blood flow (Edwards, 2024). A decrease in TPR directly lowers afterload, reducing the mechanical burden placed on the left ventricle. For example, isometric protocols such as wall-squat holds induce sustained contraction of the lower-body musculature, temporarily affecting blood flow and intramuscular pressure; however, during recovery, a drop in TPR contributes to improved ventricular unloading.41 With reduced afterload, the left ventricle requires less force to eject blood, facilitating a more efficient opening of the aortic valve and decreasing myocardial workload. This reduction in mechanical stress lowers cardiac oxygen requirements, offering cardioprotective benefits particularly relevant to individuals with elevated cardiovascular risk (Ji, 2024). Lower afterload also contributes to enhanced long-term cardiac efficiency. By reducing the chronic strain placed on ventricular walls, isometric exercise training may help mitigate progression toward conditions such as heart failure, where the myocardium becomes unable to meet circulatory demands (Taylor, 2017). Over time, adaptations including reduced resting heart rate further lower

RPP, collectively diminishing myocardial oxygen consumption and improving overall cardiac function (Saxena, 2018). Regular isometric training therefore provides a practical, low-impact modality capable of reducing cardiac workload through sustained improvements in afterload, TPR, and HR.

Isometric Exercise Training in Reducing Resting Blood Pressure (RBP) Levels
Isometric exercise training contributes significantly to reductions in resting blood pressure (RBP) through a series of autonomic and vascular adaptations. Following isometric exercise, activation of the parasympathetic nervous system becomes prominent, reflected by an increase in high-frequency normalized units (HFnu), a widely used index of cardiac parasympathetic activity (Taylor, 2017). Enhanced parasympathetic tone plays a key role in post-exercise recovery by reducing cardiovascular stress and promoting blood pressure normalization. A major consequence of parasympathetic activation is the marked enhancement—up to threefold—of baroreceptor sensitivity. Baroreceptors, located in the carotid sinus and aortic arch, serve as essential short-term regulators of arterial pressure, continuously monitoring stretch within arterial walls (Pikkarainen, 2023). When these receptors detect changes in blood pressure, they relay signals to the medulla oblongata to initiate corrective responses (Armstrong, 2023). Increased baroreceptor sensitivity following isometric training improves the body's capacity to recognize and correct blood pressure fluctuations rapidly. This heightened responsiveness triggers vasodilatory mechanisms, reducing peripheral resistance and facilitating quicker stabilization of blood pressure during recovery (Suarez, 2021). By enabling more efficient short-term regulation, improved baroreceptor sensitivity holds particular therapeutic value for individuals with hypertension.

Sustained reductions in resting blood pressure decrease myocardial workload, allowing the heart to eject blood more efficiently and decreasing the risk of long-term structural and functional damage to the myocardium and vasculature (Mahe, 2017). Through repeated training, enhanced baroreflex function not only supports acute hemodynamic control but also provides protective effects against the development or progression of cardiovascular disease (Suarez, 2021). Parasympathetic activation and improved baroreceptor sensitivity also contribute to reductions in pro-inflammatory markers such as interleukin-6 (IL-6) and asymmetric dimethylarginine (ADMA). Lower ADMA levels increase nitric oxide (NO) bioavailability, promoting vasodilation and decreasing total peripheral resistance (TPR) (Taylor, 2017). The resulting reduction in arterial resistance contributes to lower resting blood pressure and enhances cardiac efficiency. Additionally, decreased afterload facilitates more effective left ventricular ejection, increasing stroke volume (SV) and stabilizing heart rate (HR), which together improve overall cardiac output (Banks, 2024). These combined autonomic, vascular, and inflammatory adaptations illustrate how isometric exercise training exerts meaningful and sustained benefits on cardiovascular health.

Limitations
This review is constrained by its dependence on secondary data sourced from previously published literature, which introduces variability related to study design, sample characteristics, and methodological approaches. Many of the available studies emphasize short-term physiological outcomes, resulting in limited evidence regarding the long-term sustainability of blood pressure reduction following isometric exercise training. Furthermore, heterogeneity in exercise protocols—including differences in contraction intensity, duration, joint angles, and muscle groups targeted—limits the comparability and standardization of findings across studies. The exclusion of non-English publications may also restrict the scope of the review by omitting potentially relevant evidence from non-English-speaking regions.

CONCLUSION
Isometric exercise training demonstrates significant efficacy in reducing resting blood pressure and improving overall cardiovascular efficiency. During the exercise phase, sympathetic activation

increases blood pressure through elevated vascular resistance and enhanced left ventricular contractility, accompanied by baroreflex resetting to support the acute metabolic demands of muscle contraction. In the recovery phase, increased nitric oxide (NO) release promotes vascular relaxation and decreases total peripheral resistance, contributing to blood pressure reduction. Concurrently, enhanced parasympathetic activity improves baroreceptor sensitivity, stabilizing heart rate and supporting more efficient cardiovascular regulation. Compared with traditional aerobic exercise, isometric exercise training is associated with a lower rate-pressure product (RPP), indicating reduced myocardial oxygen demand. This makes isometric protocols a safe, effective, and accessible training option for individuals at elevated cardiovascular risk

REFERENCES

Alim, N., Sri Sulistyawati Anton, Rusman Hasanuddin, Muhammad Ihsan, & Ni Ketut Sinarsih. (2023). Uji teratogenik ekstrak etanol biji beligo (Benincasa hispida (Thunb.) Cogn.) pada mencit betina (Mus musculus). Jurnal Yoga dan Kesehatan, 6(2), 243–253.

Amponsah-Offeh, M., Diaba-Nuhoho, P., Speier, S., & Morawietz, H. (2023). Oxidative stress, antioxidants and hypertension. Antioxidants, 12(2).

Arendshorst, W. J., Vendrov, A. E., Kumar, N., Ganesh, S. K., & Madamanchi, N. R. (2024). Oxidative stress in kidney injury and hypertension. Antioxidants, 13(12), 1–47.

Armstrong, M., Moore, R.A. and Kerndt, C.C. (2023). Physiology, Baroreceptors. [online] National LIbrary of Medicine. Available at: https://www.ncbi.nlm.nih.gov/books/NBK538172/.

Aslam, S., Habyarimana, J.D.D. and Bin, S.Y. (2025). Neuromuscular adaptations to resistance training in elite versus recreational athletes. Frontiers in Physiology, 16. doi:https://doi.org/10.3389/fphys.2025.1598149.

Atahan Durbas, Caglar Yilgor and Ahmet Alanay (2025). Functional Anatomy of the Spine. pp.787–801. doi:https://doi.org/10.1007/978-3-031-58351-3_350.

Baffour-Awuah, B., Pearson, M. J., Dieberg, G., & Smart, N. A. (2023). Isometric resistance training to manage hypertension: Systematic review and meta-analysis. Current Hypertension Reports, 25(4), 35–49.

Baffour-Awuah, B., Pearson, M. J., Dieberg, G., Wiles, J. D., & Smart, N. A. (2023). An evidence-based guide to the efficacy and safety of isometric resistance training in hypertension and clinical implications. Clinical Hypertension, 29(1), 1–12.

Banks, N. F., Rogers, E. M., Stanhewicz, A. E., Whitaker, K. M., & Jenkins, N. D. M. (2024). Resistance exercise lowers blood pressure and improves vascular endothelial function in individuals with elevated blood pressure or stage-1 hypertension. American Journal of Physiology-Heart and Circulatory Physiology, 326(1), H256–H269.

Biscarini, A., Dieni, C. V., Losavio, R., Panichi, R., & Contemori, S. (2022). Effect of forefoot–rearfoot and pelvic–scapular weight shifts on lower-limb and lumbar muscle activity during static wall-squat exercises. Applied Sciences, 12(8).

Cohen, D. D., Aroca-Martinez, G., Carreño-Robayo, J., Castañeda-Hernández, A., Herazo-Beltran, Y., Camacho, P. A., et al. (2023). Reductions in systolic blood pressure achieved by hypertensives with three isometric training sessions per week are maintained with a single session per week. Journal of Clinical Hypertension, 25(4), 380–387.

Constantinou, A., Mamais, I., Papathanasiou, G., Lamnisos, D., & Stasinopoulos, D. (2022). Comparing hip and knee focused exercises versus hip and knee focused exercises with blood flow restriction in patellofemoral pain. European Journal of Physical and Rehabilitation Medicine, 58(2), 225–235.

Edwards, J. J., Coleman, D. A., Ritti-Dias, R. M., Farah, B. Q., Stensel, D. J., Lucas, S. J. E., et al. (2024). Isometric exercise training and arterial hypertension: An updated review. Sports Medicine, 54, 1459–1497.

Falkner, B., & Daniels, S. R. (2024). Diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Hypertension, 44(4), 387–388.

Gallo, G., Volpe, M., & Savoia, C. (2022). Endothelial dysfunction in hypertension. Frontiers in Medicine, 8, 1–8.

Glaviano, N.R. and Saliba, S. (2022). Differences in Gluteal and Quadriceps Muscle Activation During Weight-Bearing Exercises Between Female Subjects With and Without Patellofemoral Pain. Journal of Strength and Conditioning Research, p.1. doi:https://doi.org/10.1519/jsc.0000000000003392.

Gullu, B., Subasi, O. and Aslan, L. (2024). Biomechanics of the Foot and Ankle. Clinical and Radiological Examination of the Foot and Ankle, pp.11–32. doi:https://doi.org/10.1007/978-981-97-4202-8_2.

Hegde, S., Ahmed, I. and Aeddula, N. (2023). Secondary Hypertension. StatPearls. [online] Available at: https://www.statpearls.com/point-of-care/28819.

Ji, A., Davies, J., Phan, P. T., Nguyen, C. C., Sharma, B., Zhu, K., et al. (2024). Development of a self-deploying extra-aortic compression device. Advanced Science.

Kabootari, M., Saeed, S., Mitra Hasheminia, Azizi, F. and Farzad Hadaegh (2023). Change in blood pressure status defined by 2017 ACC/AHA hypertension guideline and risk of cardiovascular disease: results of over a decade of follow-up of the Iranian population. Frontiers in Cardiovascular Medicine, 10. doi:https://doi.org/10.3389/fcvm.2023.1044638.

Kementerian Kesehatan RI. (2019). Hipertensi penyakit paling banyak diidap masyarakat. https://sehatnegeriku.kemkes.go.id

Kothurkar, R., Lekurwale, R., Gad, M. and Rathod, C.M. (2022). Estimation and Comparison of Knee Joint Contact Forces During Heel Contact and Heel Rise Deep Squatting. Indian Journal of Orthopaedics, 57(2), pp.310–318. doi:https://doi.org/10.1007/s43465-022-00798-y.

Kounoupis, A., Papadopoulos, S., Galanis, N., Dipla, K., & Zafeiridis, A. (2020). Blood pressure responses in isometric vs dynamic resistance exercise. Sports, 8(4).

Lea, J. W. D., O'Driscoll, J. M., & Wiles, J. D. (2024). Home-based isometric wall squat intervention. European Journal of Applied Physiology, 124(1), 281–293.

Lee, J. H., Kim, S., Heo, J., Park, D. H., & Chang, E. (2022). Muscle activity during squat exercises. BMC Sports Science, Medicine and Rehabilitation, 14(1).

Li, Z., Cao, L., Zhou, Z., Han, M. and Fu, C. (2023). Factors influencing the progression from prehypertension to hypertension among Chinese middle-aged and older adults: a 2-year longitudinal study. BMC Public Health, 23(1). doi:https://doi.org/10.1186/s12889-022-14410-3.

Li, W., Fang, W., Huang, Z., Wang, X., Cai, Z., Chen, G., et al. (2022). Age at onset of overweight and hypertension risk. Heart, 108(9), 683–688.

Lin, S., Sun, P., Huang, L., Hernandez, M., Yu, H., & Jan, Y. K. (2024). Isometric exercise and blood pressure regulation. Frontiers in Bioengineering and Biotechnology, 12.

Luksha, L., Agewall, S., & Kublickiene, K. (2009). Endothelium-derived hyperpolarizing factor. Atherosclerosis, 202(2), 330–344.

Ma, J., Li, Y., Yang, X., Liu, K., Zhang, X., Zuo, X., et al. (2023). Signaling pathways in hypertension. Signal Transduction and Targeted Therapy, 8(1).

Mahe, G., Comets, E., Nouni, A., Paillard, F., Dourmap, C., & Le Faucheur, A. (2017). Resting time before BP measurement. Scientific Reports, 7(1).

Mettler, J. A., & Griffin, L. (2016). Muscular endurance and motor unit firing. Experimental Brain Research, 234(1), 267–276.

Nurhasanah, N., Pardede, I. T., Nauli, F., Hayati, I., Nasution, F. R., & Hermawan, A. R. (2023). Nitric oxide and blood pressure. Jurnal Gizi Klinik Indonesia, 19(3), 96.

Pikkarainen, K., Valtonen, R. I. P., Hintsala, H. E., et al. (2023). Baroreflex sensitivity after exercise. Frontiers in Physiology, 14.

Reis, P. A., Albuquerque, C. F. G., et al. (2017). Nitric oxide synthase in CNS.

Rubio-Guerra, A. F., Vargas-Robles, H., et al. (2013). Prehypertension biomarkers.

Saxena, T., Ali, A. O., & Saxena, M. (2018). Essential hypertension update. Expert Review of Cardiovascular Therapy, 16(12), 879–887.

Shaito, Abdullah A, Aramouni, K., Assaf, R.K., Parenti, A., Orekhov, A.N., Yazbi, A.E., Pintus, G. and Eid, A.H. (2022). Oxidative Stress-Induced Endothelial Dysfunction in Cardiovascular Diseases. Aub.edu.lb. [online] doi:https://doi.org/10.31083/j.fbl2703105.

Sweet, D.K., Patterson, P.D., Reis, S. and Hostler, D. (2024). Changes in the reactive hyperemia index after continuous and interval exercise. Vascular Medicine, 30(1), pp.3–9. doi:https://doi.org/10.1177/1358863x241288139.

Stožer, A., Vodopivc, P., & Bombek, L. K. (2020). Exercise-induced muscle damage. Physiological Research, 69(4), 565–598.

Straub, R. K., & Powers, C. M. (2024). Biomechanics of squat. International Journal of Sports Physical Therapy, 19(4), 490–501.

Suarez-Roca, H., Mamoun, N., Sigurdson, M. I., & Maixner, W. (2021). Baroreceptor modulation. Comprehensive Physiology, 11(2), 1373–1423.

Sun, J. Y., Ma, Y. X., Liu, H. L., et al. (2022). Waist circumference and hypertension. Journal of Clinical Hypertension, 24(3), 320–328.

Tackling, G. and Borhade, M.B. (2025). Hypertensive Heart Disease. [online] National Library of Medicine. Available at: https://www.ncbi.nlm.nih.gov/books/NBK539800/.

Turpin, T., Taïlé, J., Thouvenot, K. and Gonthier, M.-P. (2026). Polyphenols Limit Cerebral Endothelial Cell Dysfunction Under Inflammatory Conditions Related to Oral and Gut Microbiota. Nutrients, [online] 18(4), p.568. doi:https://doi.org/10.3390/nu18040568.

Wang, Y., Chen, J., Wang, W., Yi, X., Xing, S., Lei, X., Chen, H. and Xie, X. (2026). The core mechanism of hypertension-linked renal fibrosis: ‘RAAS-ROS-inflammation-fibrosis’ axis. iScience, 29(4), p.115353. doi:https://doi.org/10.1016/j.isci.2026.115353.

Whelton, P. K., Carey, R. M., Aronow, W. S., et al. (2018). Hypertension guideline. Hypertension, 71, 1269–1324.

Widodo, A.F., Tien, C.-W., Chen, C.-W. and Lai, S.-C. (2022). Isotonic and Isometric Exercise Interventions Improve the Hamstring Muscles’ Strength and Flexibility: A Narrative Review. Healthcare, 10(5), p.811.

World Health Organization. (2021). More than 700 million people with untreated hypertension. https://www.who.int

Xia, T., Yu, J., Du, M., et al. (2025). Endothelial injury mechanisms. MedComm, 6(2).

Xiao, X., Zhen, W., Bai, D., Zheng, B., Wang, Y., Ma, X., Xie, X., Zhang, Y., Guo, F., Ito, K., Zhang, B., Zhang, C. and Ma, Y. (2026). The Role of Cyclooxygenase-2 in Oxidative Stress and Inflammation: Implications for Poultry Health and Production. Poultry Science, p.106909. doi:https://doi.org/10.1016/j.psj.2026.106909.

Yavuz, U., Arın Celayir and Bedri Karaismailoglu (2024). Clinical Anatomy of the Foot and Ankle. pp.33–51. doi:https://doi.org/10.1007/978-981-97-4202-8_3.

Meng, X., Zhang, Q. Y., et al. (2021). Prehypertension review. Annals of Translational Medicine, 9(2), 170.