International Journal of Health Science & Biomedicine (IJHSB)

e-ISSN: 3139-3691

RESEARCH ARTICLE OPEN ACCESS

Hemodynamic and Myocardial Workload Responses to Cold Pressor Stress in Apparently Healthy Young Adults

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Citation: Buhari A, Manta AF, Muhammad N (2026). Hemodynamic and Myocardial Workload Responses to Cold Pressor Stress in Apparently Healthy Young Adults. Int J Health Sci Biomed. 3(4): 1-5. DOI: 10.5281/zenodo.21708106

Received Date: 2026-07-26, **Accepted Date:** 2026-08-17, **Published Date:** 2026-08-28

Keywords: Cold Pressor Test; Sympathetic Nervous System; Blood Pressure; Heart Rate; Double Product; Cardiovascular Reactivity

Abstract

Background: The Cold Pressor Test (CPT) is a validated experimental model used to assess autonomic cardiovascular reactivity. It induces acute sympathetic stimulation resulting in measurable hemodynamic changes.
Objective: To evaluate cardiovascular responses to CPT in apparently healthy young adults, focusing on heart rate, blood pressure, and myocardial workload.
Methods: A total of 24 healthy participants (18–40 years) underwent standardized CPT (0–4°C hand immersion). Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were measured at baseline, during cold exposure, and recovery. Double product (DP = HR × SBP) was calculated. Data were analyzed using repeated measures ANOVA with significance set at $p < 0.05$.
Results: CPT induced a significant increase in SBP (120.4 ± 10.8 to 154.5 ± 14.4 mmHg), DBP (79.3 ± 4.8 to 99.2 ± 9.8 mmHg), HR (77.0 ± 7.5 to 92.1 ± 8.6 bpm), and DP (9083 ± 1271 to 14331 ± 2095 units) ($p < 0.001$). Partial recovery occurred post-stimulus, although values remained above baseline.
Conclusion: Cold pressor stress induces marked sympathetic cardiovascular activation and increased myocardial workload. CPT is a useful non-invasive tool for assessing autonomic cardiovascular function in humans.

Introduction

The cardiovascular system is central to physiological homeostasis, ensuring continuous perfusion of tissues and organs to meet metabolic demands. Its regulation is achieved through a highly integrated network involving the autonomic nervous system (ANS), endocrine signaling, and intrinsic vascular control mechanisms. Within this regulatory framework, the sympathetic and parasympathetic branches of the ANS exert continuous, dynamic modulation of heart rate, vascular tone, and myocardial contractility, thereby maintaining hemodynamic stability under both resting and stress conditions [1].

Under conditions of acute stress, this finely tuned autonomic balance is transiently disrupted to facilitate adaptive cardiovascular adjustments necessary for survival. Such stress responses are primarily mediated through activation of the sympathetic nervous system and stimulation of the hypothalamic–pituitary–adrenal (HPA) axis. The resultant surge in catecholamines, particularly epinephrine and norepinephrine, produces marked cardiovascular effects including increased heart rate, enhanced myocardial contractility, vasoconstriction of peripheral blood vessels, and elevated systemic arterial pressure. Collectively, these responses lead to an increase in cardiac output and redistribution of blood flow toward vital organs, thereby representing an essential physiological adaptation to stress [2].

One of the most established experimental paradigms for assessing sympathetic cardiovascular reactivity is the Cold Pressor Test (CPT). The CPT involves immersion of the hand or forearm into ice-cold water maintained at approximately 0–4°C for a defined period, typically one to three minutes. This cold stimulus activates cutaneous nociceptors and thermoreceptors, generating afferent neural signals that are transmitted via somatosensory pathways to the central nervous system. These signals are integrated within autonomic regulatory centers in the hypothalamus and brainstem, particularly the medullary cardiovascular control centers, leading to pronounced sympathetic efferent discharge [3].

The physiological outcome of this sympathetic activation is a coordinated cardiovascular response characterized by systemic vasoconstriction, increased total peripheral resistance, elevation of systolic and diastolic blood pressure, and augmentation of heart rate. These changes collectively increase myocardial workload and oxygen demand, making the CPT a valuable tool for probing autonomic cardiovascular regulation and stress reactivity [4].

Historically, the Cold Pressor Test was first introduced in 1932 by Hines and Brown as a simple clinical method to evaluate blood pressure responsiveness to stress and to identify individuals with exaggerated hypertensive tendencies. Since its introduction, it has

evolved into a widely accepted and reproducible experimental model in cardiovascular physiology, clinical autonomic testing, and psychophysiological research, offering important insights into sympathetic nervous system function and cardiovascular risk profiling [5].

Beyond traditional hemodynamic measures such as heart rate and blood pressure, the rate-pressure product (RPP) serves as an important derived index of cardiovascular performance. RPP, defined as the product of heart rate and systolic blood pressure (RPP = HR × SBP), provides a non-invasive estimate of myocardial oxygen consumption and overall cardiac workload. It is widely used in both clinical cardiology and exercise physiology as a surrogate marker of myocardial metabolic demand, with higher values indicating increased cardiac stress and oxygen requirement [6, 7].

From a mechanistic perspective, cold-induced cardiovascular responses follow a well-coordinated neurophysiological pathway. Cutaneous thermoreceptors and nociceptors are first activated by cold exposure, generating afferent sensory input that ascends to central autonomic centers. Integration within the hypothalamus and brainstem results in activation of sympathetic efferent pathways, culminating in norepinephrine release from postganglionic sympathetic fibers and epinephrine secretion from the adrenal medulla. This cascade produces vasoconstriction, increased cardiac contractility, and elevated arterial pressure, ultimately increasing myocardial oxygen demand and cardiovascular workload.

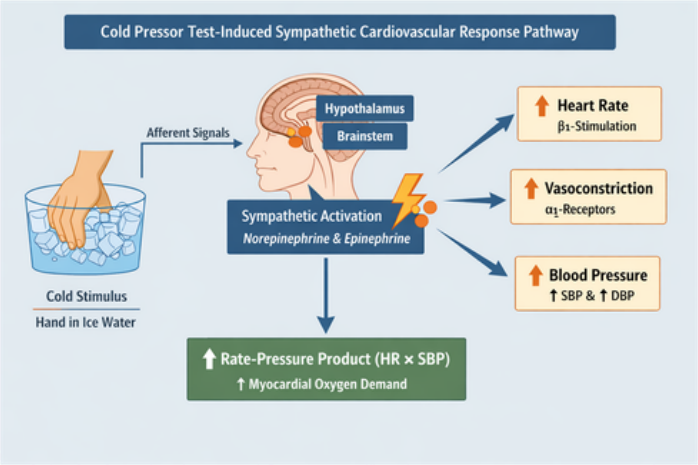

Figure 1: Schematic representation of the Cold Pressor Test–induced sympathetic cardiovascular response pathway

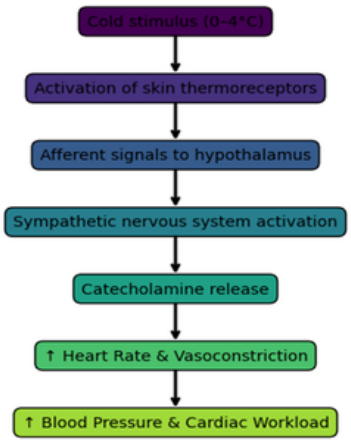

Figure 2: Physiological Mechanism of cardiovascular response to Cold Pressor Test

[Figure 1, 2], illustrates the sequence beginning with cold stimulus exposure to the hand or forearm, followed by activation of cutaneous thermoreceptors and nociceptors. Afferent neural signals are transmitted to the spinal cord and brainstem, with integration occurring in the hypothalamus and medullary cardiovascular centers. This results in enhanced sympathetic efferent outflow, leading to catecholamine release (norepinephrine and epinephrine), peripheral vasoconstriction, increased heart rate, elevated blood pressure, and increased rate-pressure product, ultimately reflecting heightened myocardial oxygen demand [Figure 2].

Accumulating evidence suggests that individuals who exhibit exaggerated cardiovascular responses to the Cold Pressor Test may have an increased predisposition to hypertension and future cardiovascular disease. Heightened sympathetic reactivity has been associated with autonomic imbalance, endothelial dysfunction, and altered vascular responsiveness, all of which contribute to long-term cardiovascular risk [8].

Despite the extensive application of the Cold Pressor Test in cardiovascular and autonomic research globally, there remains a paucity of data describing cold-induced cardiovascular reactivity among young healthy adults in many developing regions, particularly within African populations. This gap is significant, as population-specific physiological reference values are essential for accurate interpretation of autonomic function and for early identification of individuals at risk of cardiovascular dysregulation.

Therefore, the present study is designed to investigate the effects of the Cold Pressor Test on heart rate, blood pressure, and rate-pressure product in apparently healthy young adults, with the aim of contributing to the understanding of sympathetic cardiovascular reactivity and establishing baseline physiological responses within this population.

Materials And Methods

Study Design

This study employed a cross-sectional experimental design to investigate cardiovascular responses to sympathetic stimulation induced by the Cold Pressor Test.

Study Population

The study involved apparently healthy adult volunteers recruited from the Department of Human Physiology at Federal University Birnin Kebbi, Nigeria. Participants were selected using convenience sampling.

Sample Size

A total of 24 participants were included in the study.

Inclusion Criteria

Participants were included if they:

- Were between 18 and 30 years old
- Were apparently healthy
- Had no history of cardiovascular disease
- Were not taking medications affecting cardiovascular function

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Exclusion Criteria

Participants were excluded if they had:

- Hypertension
- Diabetes mellitus
- Cardiovascular disorders
- Peripheral vascular disease

Experimental Protocol

Participants were instructed to avoid caffeine, alcohol, and strenuous physical activity for at least 12 hours before the experiment.
Upon arrival at the laboratory, participants rested quietly for approximately 10 minutes to ensure cardiovascular stabilization.

Baseline measurements were recorded:

- Heart rate (beats per minute)
- Systolic blood pressure (mmHg)
- Diastolic blood pressure (mmHg)

Heart rate was measured using a digital pulse monitor, while blood pressure was measured using a calibrated sphygmomanometer following standard procedures [9].

Cold Pressor Test Procedure

Participants immersed their non-dominant hand into a container filled with ice-cold water maintained at 0–4°C for one minute.
During cold exposure, cardiovascular parameters were measured again.

Calculation of Rate-Pressure Product

Rate-pressure product was calculated using heart rate and systolic blood pressure values obtained during the experiment.

Ethical Considerations

Ethical approval for the study was obtained from the Department of Human Physiology, Federal University Birnin Kebbi. All participants provided written informed consent prior to participation.

Statistical Analysis

Data were analyzed using descriptive statistics and inferential statistical tests.

Results were expressed as:

- Mean
- Standard deviation

Paired t-tests were used to compare baseline and cold-exposure values.

Statistical significance was set at $p < 0.05$.

Results

Hemodynamic Responses

Cold pressor exposure resulted in a significant increase in systolic blood pressure from 120.4 ± 10.8 mmHg at baseline to 154.5 ± 14.4 mmHg during exposure ($p < 0.001$). Diastolic blood pressure similarly increased from 79.3 ± 4.8 mmHg to 99.2 ± 9.8 mmHg ($p < 0.001$).

Heart Rate Response

Heart rate increased significantly from 77.0 ± 7.5 bpm at baseline to 92.1 ± 8.6 bpm during CPT ($p < 0.001$), reflecting sympathetic activation.

Double Product

Double product increased markedly from 9083 ± 1271 to 14331 ± 2095 units during CPT ($p < 0.001$), indicating increased myocardial oxygen demand.

Recovery Phase

All parameters decreased after removal of stimulus but remained above baseline, indicating sustained autonomic activation.

Heart Rate Response

[Figure 3], showed heart rate during the Cold Pressor Test compared with baseline values, reflecting sympathetic nervous system activation.

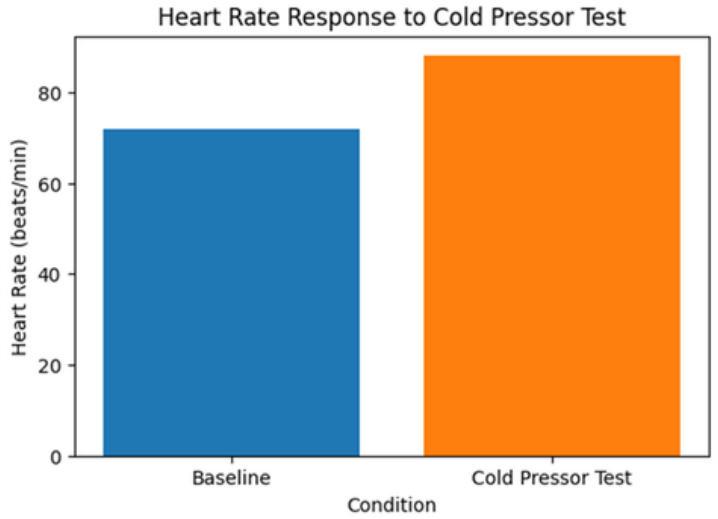

Figure 3: Heart Rate

Systolic Blood Pressure Response

Systolic blood pressure increased during cold exposure, indicating increased cardiac output and peripheral vascular resistance [Figure 4].

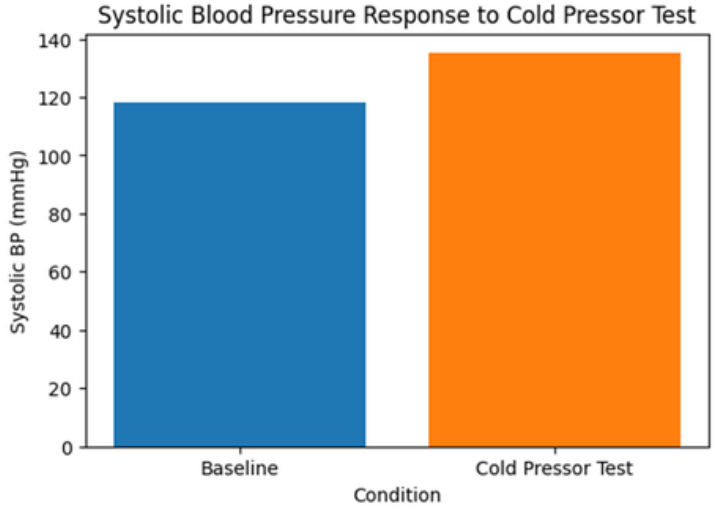

Figure 4: Systolic Blood Pressure

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Diastolic Blood Pressure Response

Diastolic blood pressure also increased during cold exposure, reflecting increased peripheral vasoconstriction [Figure 5].

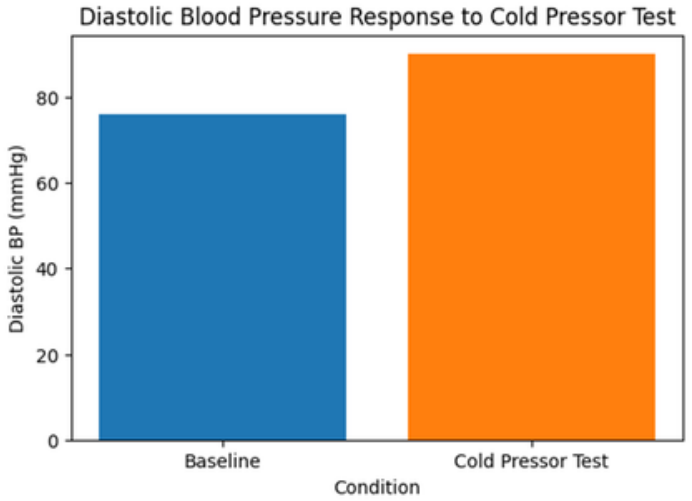

Figure 5: Diastolic Blood Pressure

Rate-Pressure Product Response

Rate-pressure product increased during cold exposure, indicating increased myocardial oxygen demand [Figure 6].

Overall, these findings demonstrate that the Cold Pressor Test induces significant cardiovascular responses through activation of sympathetic pathways.

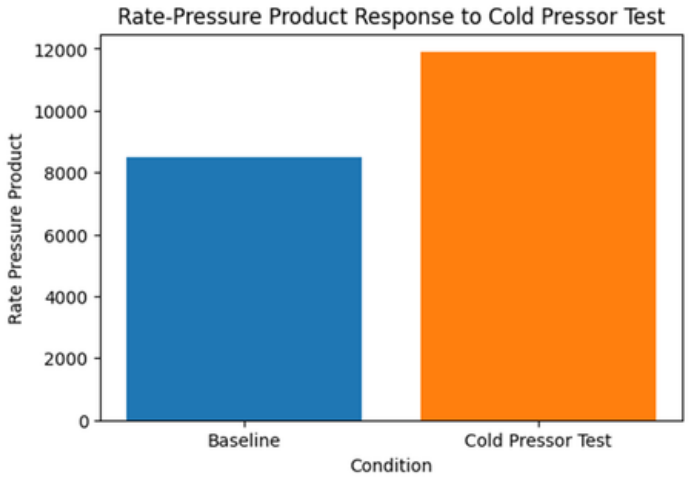

Figure 6: Rate-Pressure Product

Discussion

The present study investigated the acute cardiovascular responses to the Cold Pressor Test (CPT) in apparently healthy young adults, with specific focus on heart rate, systolic and diastolic blood pressure, and rate-pressure product, an established surrogate index of myocardial oxygen consumption. The findings demonstrated a significant increase in all measured cardiovascular parameters during cold exposure, followed by a partial recovery toward baseline after stimulus withdrawal. These results reflect a coordinated and physiologically consistent sympathetic–adrenergic response to acute cold stress.

The Cold Pressor Test is a well-established experimental paradigm for evaluating sympathetic nervous system reactivity and cardiovascular control. Cold stimulation of peripheral tissues activates cutaneous nociceptors and thermoreceptors, which transmit afferent signals to central autonomic regulatory centers in the hypothalamus and brainstem. This integration results in increased sympathetic outflow and reduced parasympathetic activity, producing systemic cardiovascular adjustments aimed at maintaining perfusion pressure during stress [3]. The overall response represents an integrated neurocardiovascular adaptation to environmental challenge.

A major finding of this study was the significant increase in heart rate during cold exposure. This tachycardic response is primarily mediated by sympathetic stimulation of the sinoatrial node through catecholamine release, particularly norepinephrine, acting on β_1-adrenergic receptors. This increases the rate of spontaneous depolarization of pacemaker cells, thereby accelerating heart rate. Concurrent vagal withdrawal further amplifies this response. These findings are consistent with previous studies demonstrating rapid chronotropic responses during CPT and increased sympathetic nerve activity during cold stress [1,3,8].

The study also demonstrated significant elevations in both systolic and diastolic blood pressures during CPT. The increase in systolic blood pressure reflects enhanced myocardial contractility and stroke volume mediated by β_1-adrenergic stimulation, as well as increased afterload secondary to peripheral vasoconstriction. In contrast, the rise in diastolic blood pressure is primarily driven by α_1-adrenergic-mediated vasoconstriction of arterioles, leading to increased total peripheral resistance. While this vasoconstrictive response serves an important thermoregulatory function by minimizing heat loss, it concurrently elevates systemic vascular resistance and arterial pressure [4].

Another important observation was the significant increase in rate-pressure product (RPP), which reflects myocardial oxygen demand and overall cardiac workload. Since RPP is derived from the product of heart rate and systolic blood pressure, its elevation indicates a substantial increase in myocardial metabolic requirement during cold stress. This suggests that even short-duration cold exposure imposes measurable cardiac energetic demand in healthy individuals. Although this response is physiologically normal, exaggerated increases in RPP may indicate heightened cardiovascular stress and potential susceptibility to future cardiovascular dysfunction.

The integrated cardiovascular response observed in this study reflects a tightly coordinated autonomic pattern involving sympathetic activation and parasympathetic withdrawal. Cold exposure initiates afferent sensory signaling from peripheral receptors to the hypothalamus and brainstem, resulting in efferent sympathetic discharge. This produces vasoconstriction, increased cardiac contractility, and tachycardia, collectively contributing to elevated blood pressure and myocardial workload. The partial recovery observed after cessation of the stimulus suggests preserved autonomic flexibility and intact baroreflex-mediated regulation in the study population.

These findings are consistent with classical and contemporary literature [5]. First demonstrated that cold immersion produces significant pressor responses via sympathetic activation [3].Further confirmed increased sympathetic nerve traffic during CPT, while [8], showed that exaggerated pressor responses may be associated with increased risk of developing hypertension [4].Also emphasized the role of cold-induced vasoconstriction in mediating increases in arterial pressure during cold stress.

Beyond hemodynamic regulation, CPT responses are closely linked to

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thermoregulatory physiology. Cold exposure activates cutaneous vasoconstriction to conserve core body temperature, a process mediated by sympathetic adrenergic pathways. While essential for survival in cold environments, this response contributes significantly to increased peripheral resistance and diastolic blood pressure elevation. Additionally, activation of the hypothalamic–pituitary–adrenal axis during stress further amplifies cardiovascular responses through hormonal release, including catecholamines and cortisol, which increase vascular tone and cardiac output.

Individual variability in CPT responses may be influenced by factors such as age, sex, physical fitness, and genetic predisposition. Individuals with higher cardiovascular fitness typically exhibit attenuated responses due to enhanced parasympathetic tone and improved autonomic efficiency, whereas reduced fitness or aging is often associated with exaggerated responses and delayed recovery kinetics. These differences highlight the importance of autonomic balance in cardiovascular adaptability to stress.

The physiological significance of the present findings lies in the demonstration of intact and efficient autonomic cardiovascular regulation in apparently healthy young adults. The Cold Pressor Test elicited predictable and reproducible increases in heart rate, blood pressure, and myocardial workload, underscoring its utility as a simple, non-invasive tool for assessing sympathetic cardiovascular reactivity. These responses highlight the sensitivity of the cardiovascular system to acute environmental stress and the effectiveness of compensatory autonomic mechanisms in maintaining homeostasis.

From a clinical perspective, the Cold Pressor Test has important implications for cardiovascular risk stratification. Exaggerated sympathetic and pressor responses have been associated with increased risk of developing hypertension and cardiovascular disease. Such responses may reflect underlying autonomic imbalance characterized by increased sympathetic drive or reduced parasympathetic modulation, both of which contribute to long-term cardiovascular risk development.

In conclusion, the present study demonstrates that Cold Pressor Test exposure significantly increases heart rate, blood pressure, and rate-pressure product in apparently healthy young adults. These findings reflect robust sympathetic nervous system activation and increased myocardial workload, providing valuable insight into cardiovascular stress reactivity and autonomic regulation. Overall, the Cold Pressor Test remains a reliable and informative tool for evaluating autonomic cardiovascular function and may be useful in identifying individuals with heightened cardiovascular reactivity who may be at increased risk of future cardiovascular dysfunction.

Limitation

The present study has some limitations that should be acknowledged. First, the relatively small sample size may limit the generalizability of the findings to a broader population. Second, the restricted age range of participants may not fully capture age-related variations in autonomic cardiovascular responses to cold stress. Third, the short duration of cardiovascular monitoring may not have fully captured the complete recovery profile following cold exposure, particularly the late-phase autonomic adjustments.

Future studies should address these limitations by recruiting larger and more diverse populations across different age groups and physiological backgrounds. In addition, incorporating more comprehensive physiological markers such as heart rate variability, plasma catecholamine levels, and endothelial function indices would provide deeper insight into autonomic and neuroendocrine mechanisms underlying cold-induced cardiovascular responses.

Conclusion

The Cold Pressor Test elicited significant increases in heart rate, blood pressure, and rate-pressure product in apparently healthy adults, reflecting robust activation of the sympathetic nervous system in response to acute cold stress. These findings underscore the central role of sympathetic cardiovascular regulation in maintaining hemodynamic stability during environmental challenges.

Overall, the Cold Pressor Test remains a simple, reliable, and non-invasive experimental tool for assessing autonomic cardiovascular function and stress reactivity. It provides valuable insight into sympathetic cardiovascular control and may be useful in identifying individuals with exaggerated cardiovascular responses who could be at increased risk of future cardiovascular dysfunction.

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