American Journal of
Biomedical Science & Research

www.biomedgrid.com
ISSN: 2642-1747

Mini review

Copyright© Gulnara Jafar Valiyeva

Cardiac Output Regulation During Rest and Exercise

Gulnara Jafar Valiyeva*, Asiya Haji Aghayeva, Dilara Magsood Aliyeva, Azada Azad Rustamzada, Adil Allahyar Bayramov

Department of Normal Physiology, Azerbaijan Medical University, Azerbaijan

***Corresponding author:** Gulnara Jafar Valiyeva, Department of Normal Physiology, Azerbaijan Medical University, Azerbaijan.

To Cite This Article: *Gulnara Jafar Valiyeva*, Asiya Haji Aghayeva, Dilara Magsood Aliyeva, Azada Azad Rustamzada, Adil Allahyar Bayramov. Cardiac Output Regulation During Rest and Exercise. Am J Biomed Sci & Res. 2025 28(6) AJBSR.MS.ID.003737,* **DOI:** 10.34297/AJBSR.2025.28.003737

Received: October 10, 2025; **Published:** October 28, 2025

Abstract

Cardiac Output (CO), defined as the total volume of blood ejected by the ventricles per minute, serves as a critical determinant of tissue perfusion and overall cardiovascular performance. It reflects the efficiency with which the heart meets the metabolic demands of the body under varying physiological conditions. At rest, cardiac output is tightly regulated to sustain the oxygen and nutrient requirements of vital organs such as the brain, kidneys, and liver. This regulation is primarily mediated by autonomic nervous system balance, baroreceptor reflexes, and the intrinsic contractile properties of the myocardium. The parasympathetic nervous system predominates during rest, maintaining a relatively low heart rate and stable stroke volume through the Frank-Starling mechanism, which links venous return to cardiac performance.

During exercise, however, metabolic demand rises sharply, requiring a proportional increase in cardiac output to deliver sufficient oxygen to working skeletal muscles and to remove metabolic byproducts such as carbon dioxide and lactate. This increase is achieved through a complex integration of neural, hormonal, and local vascular mechanisms. The sympathetic nervous system stimulates both heart rate and myocardial contractility via β_1-adrenergic activation, while circulating catecholamines such as epinephrine and norepinephrine further augment cardiac output and redistribute blood flow from nonessential organs to active tissues. At the same time, peripheral vasodilation in skeletal muscle, mediated by local metabolites and endothelial factors like nitric oxide and adenosine, ensures efficient perfusion and reduces total peripheral resistance.

Adaptations of cardiac output during rest and exercise exemplify the dynamic interplay between central and peripheral regulatory systems. The rapid and coordinated adjustments in heart rate, stroke volume, venous return, and vascular tone highlight the heart's capacity to function as a highly adaptable pump capable of maintaining homeostasis even under extreme physiological stress. Understanding these mechanisms is essential not only for elucidating the foundations of normal cardiovascular physiology but also for interpreting pathophysiological conditions such as heart failure, autonomic dysfunction, and exercise intolerance.

Keywords: Cardiac Output, Exercise, Autonomic nervous system

Introduction

The regulation of cardiac output is one of the most fundamental processes ensuring the maintenance of adequate tissue perfusion and oxygen delivery throughout the body. Cardiac Output (CO), mathematically expressed as the product of Heart Rate (HR) and Stroke Volume (SV), reflects the efficiency with which the heart performs its pumping function under both resting and active physiological states. In healthy adults, resting cardiac output is typically about 4.5-5.5 liters per minute, though this value can vary depending on age, body size, and metabolic rate [1]. During conditions of physical exertion, the cardiac output may increase four- to sevenfold, reaching as high as 20-35 liters per minute in trained individuals. This wide physiological range demonstrates the remarkable adaptability of the cardiovascular system to fluctuations in metabolic demand.

Under resting conditions, the cardiovascular system maintains homeostasis primarily through autonomic balance and intrinsic myocardial properties. The parasympathetic (vagal) tone predominates at rest, resulting in a relatively low heart rate, usually between 60 and 80 beats per minute. This allows the heart to sustain an adequate stroke volume without unnecessary energy expenditure. The parasympathetic influence is balanced by a mild sympathetic tone, maintaining optimal vascular resistance and venous return. Additionally, the Frank-Starling mechanism ensures that stroke volume adjusts automatically to changes in venous return:

This work is licensed under Creative Commons Attribution 4.0 License

when the end-diastolic volume increases, the myocardial fibers stretch, leading to stronger contractions and higher stroke volume [2]. This intrinsic property enables the heart to synchronize output with venous inflow, maintaining circulatory stability even in the absence of neural input.

The afterload, determined largely by systemic arterial pressure, and the contractility, dependent on intracellular calcium dynamics and sympathetic stimulation, also play crucial roles in determining cardiac performance. At rest, these parameters are finely tuned by baroreceptor reflexes located in the carotid sinus and aortic arch. These reflexes detect minute fluctuations in arterial pressure and rapidly adjust heart rate, contractility, and vascular tone to stabilize cardiac output and systemic perfusion. Such mechanisms illustrate the precision with which cardiovascular control centers in the medulla oblongata maintain hemodynamic equilibrium.

When an individual transitions from rest to exercise, the demands placed upon the cardiovascular system change dramatically. Metabolic activity in skeletal muscles increases, leading to higher oxygen consumption and greater production of carbon dioxide and metabolic byproducts. To accommodate this, the body must elevate cardiac output through a coordinated response involving neural, hormonal, and local vascular mechanisms. The sympathetic nervous system becomes dominant, increasing heart rate, myocardial contractility, and venous return through activation of β_1-adrenergic receptors [3]. At the same time, local vasodilator substances—such as adenosine, nitric oxide, and potassium ions—act on the arterioles within working muscles to decrease vascular resistance, thereby promoting greater blood flow. These adjustments collectively ensure that oxygen delivery to active tissues rises in proportion to metabolic demand without compromising perfusion to vital organs such as the brain and heart.

Therefore, the regulation of cardiac output represents a highly integrated physiological process that harmonizes the activities of the heart, blood vessels, and autonomic nervous system. The capacity of this system to maintain adequate perfusion across varying levels of activity highlights the heart's role not merely as a mechanical pump but as a dynamic organ responding continuously to internal and external stimuli. Understanding these regulatory mechanisms forms the foundation for exploring both normal cardiovascular physiology and the pathophysiological states where these control systems become impaired, such as in heart failure, hypertension, or autonomic dysfunction.

Materials and Methods

This article is based on a comprehensive synthesis of empirical data and theoretical frameworks derived from peer-reviewed physiological and clinical research published in internationally recognized journals and authoritative textbooks. The reviewed literature encompasses both classical sources, which established the foundational understanding of cardiovascular regulation, and modern experimental studies that have expanded this knowledge through advanced methodologies such as echocardiography, Doppler flow analysis, and autonomic nervous system monitoring.

The analysis integrates findings from experimental, observational, and computational studies conducted on healthy human subjects of various ages and fitness levels to capture the full range of physiological variability. Emphasis was placed on studies that examined neural control of heart rate, hormonal influences on vascular tone, and mechanical determinants of stroke volume under resting and exercising conditions. These investigations typically employed precise measurement techniques such as cardiac catheterization, impedance cardiography, and Magnetic Resonance Imaging (MRI) to quantify cardiac output and its determinants with high temporal and spatial resolution.

The theoretical basis of this synthesis draws upon well-established physiological models, including the Frank-Starling law of the heart, which explains how myocardial stretch modulates contractile force and thus stroke volume; the baroreceptor reflex, which provides rapid adjustments in heart rate and vascular resistance in response to arterial pressure changes; and the intricate balance between sympathetic and parasympathetic divisions of the autonomic nervous system, which ensures appropriate modulation of cardiac output during both rest and activity [4,5].

To maintain objectivity and consistency, only studies employing validated methodologies and clearly defined physiological parameters were included. Data interpretation focused on the dynamic interactions between central command, peripheral feedback mechanisms, and endocrine modulation, which together coordinate the cardiovascular system's ability to match oxygen delivery with metabolic demand. No new experimental data were produced for this work; rather, the purpose was to integrate established findings into a unified conceptual model describing how cardiac output is regulated through overlapping neural, mechanical, and hormonal processes.

By consolidating diverse strands of evidence, this review provides a coherent overview of how intrinsic cardiac properties, autonomic input, and vascular feedback mechanisms interact to sustain hemodynamic stability across varying physiological states. Such a synthesis not only clarifies the mechanisms underlying cardiac output regulation but also serves as a theoretical foundation for future research in exercise physiology and cardiovascular homeostasis.

Results

The integrated analysis of existing literature reveals that cardiac output during rest and exercise is regulated through a multifactorial system involving neural, hormonal, and intrinsic myocardial mechanisms that operate in close coordination to maintain circulatory homeostasis. Under resting conditions, cardiac output remains relatively stable owing to a balance between vagal tone, baroreceptor-mediated feedback, and the intrinsic contractile properties of the myocardium [6]. The parasympathetic nervous system, primarily via the vagus nerve, exerts a dominant inhibitory influence on the Sinoatrial (SA) node, thereby maintaining a lower resting heart

rate. Simultaneously, the baroreceptor reflex located in the carotid sinus and aortic arch continuously monitors arterial pressure and modulates autonomic outflow to stabilize cardiac output in response to minor fluctuations in blood volume or posture.

A fundamental mechanism ensuring hemodynamic stability at rest is the Frank-Starling law of the heart, which states that increased end-diastolic volume stretches myocardial fibers, enhancing the force of contraction and consequently stroke volume. This intrinsic autoregulatory capacity allows the heart to adjust its output precisely according to venous return, ensuring equilibrium between the right and left ventricles without the need for external modulation. Small variations in preload or afterload are therefore effectively compensated, maintaining a consistent cardiac output despite transient circulatory changes [6].

During physical exercise, however, the regulation of cardiac output undergoes profound transformation driven by sympathetic activation and vagal withdrawal. This coordinated shift in autonomic balance results in rapid acceleration of the sinoatrial node firing rate, leading to an increased heart rate. Sympathetic efferents stimulate β_1-adrenergic receptors within the myocardium, enhancing inotropy (contractile strength) and chronotropy (rate), while also facilitating faster atrioventricular conduction [7]. These effects collectively elevate both heart rate and stroke volume, producing a marked rise in cardiac output to meet the metabolic demands of working muscles.

In parallel, hormonal modulation plays a vital role in sustaining this response. Circulating catecholamines such as epinephrine and norepinephrine, released from the adrenal medulla, potentiate sympathetic effects by binding to cardiac β_1-receptors and vascular α-receptors. This dual action not only augments myocardial performance but also redistributes blood flow by inducing vasoconstriction in splanchnic, renal, and cutaneous circulations, thereby prioritizing perfusion to skeletal muscles and the myocardium [8].

Additional regulation occurs through the Renin-Angiotensin-Aldosterone System (RAAS), which maintains arterial pressure by promoting sodium and water retention, stabilizing blood volume during prolonged exertion. Meanwhile, local metabolic vasodilators such as adenosine, carbon dioxide, potassium ions, and nitric oxide accumulate in active skeletal muscles, counteracting systemic vasoconstriction and ensuring adequate oxygen delivery to tissues [9]. This interplay between systemic sympathetic vasoconstriction and local metabolic vasodilation allows optimal redistribution of cardiac output according to the intensity and location of muscular activity.

Stroke volume also rises during exercise as a consequence of enhanced venous return, mediated by the muscle pump, respiratory pump, and increased venous tone. In well-trained individuals, chronic cardiovascular adaptations—such as ventricular hypertrophy, expanded plasma volume, and increased myocardial compliance—further augment end-diastolic filling and ejection efficiency. Consequently, elite athletes often achieve a higher cardiac output at a lower heart rate compared to untrained individuals, reflecting superior stroke volume performance and autonomic efficiency [9].

Following cessation of exercise, parasympathetic reactivation and sympathetic withdrawal dominate, initiating a gradual decline of cardiac output toward baseline values. The rate of this post-exercise decline, known as Heart Rate Recovery (HRR), serves as a sensitive indicator of autonomic balance and cardiovascular fitness [10]. A rapid HRR reflects robust vagal reactivation and effective cardiovascular regulation, whereas delayed recovery may indicate reduced autonomic flexibility or underlying cardiovascular dysfunction. Thus, the dynamic interplay between sympathetic and parasympathetic mechanisms during and after exercise exemplifies the remarkable adaptability of the human cardiovascular system to continuously varying physiological demands.

Conclusion

Cardiac output regulation represents one of the most vital adaptive processes in human physiology, serving as a cornerstone for maintaining adequate tissue perfusion and ensuring that metabolic demands are met across varying conditions. Under resting circumstances, homeostasis is preserved through a delicate interplay of parasympathetic control, baroreceptor-mediated reflexes, and intrinsic myocardial properties. The parasympathetic nervous system maintains a relatively low heart rate, while the Frank-Starling mechanism adjusts stroke volume in response to venous return, ensuring that cardiac output remains steady despite minor fluctuations in circulatory volume or posture. This finely tuned balance allows vital organs such as the brain, kidneys, and liver to receive a continuous and sufficient supply of oxygen and nutrients without unnecessary energetic expenditure.

During physical activity, the cardiovascular system demonstrates remarkable adaptability by undergoing rapid reorganization in response to increased metabolic demand. Sympathetic activation elevates heart rate and contractility, while hormonal influences, including circulating catecholamines and renin-angiotensin-aldosterone system activation, enhance cardiac performance and support blood pressure maintenance. Simultaneously, local metabolic control within active skeletal muscles promotes vasodilation, reducing peripheral resistance and facilitating optimal oxygen delivery. These adjustments are highly coordinated, allowing cardiac output to rise proportionally to the intensity of exercise while maintaining perfusion to essential organs.

The dynamic regulation of cardiac output exemplifies the plasticity of the cardiovascular system, highlighting its capacity to respond to a broad range of physiological stresses, from quiet rest to intense exertion. Structural adaptations of the heart, such as increased ventricular compliance and myocardial contractile efficiency in trained individuals, further amplify this flexibility, demonstrating how chronic exposure to physical demands refines cardiovascular performance.

Understanding these regulatory mechanisms provides not only

a framework for appreciating normal human physiology but also serves as a critical foundation for assessing pathological conditions that impair cardiac function. Disorders such as heart failure, autonomic dysfunction, or impaired vascular responsiveness can disrupt the delicate balance between neural, hormonal, and intrinsic myocardial controls, leading to inadequate tissue perfusion and exercise intolerance. Moreover, insights into cardiac output regulation underpin clinical strategies for optimizing cardiovascular health, guiding interventions ranging from exercise training programs to pharmacological modulation of autonomic activity.

In summary, the orchestration of neural, hormonal, and local mechanical mechanisms in regulating cardiac output reflects the heart's exceptional capacity for adaptive control, ensuring that circulatory demands are met efficiently under a wide spectrum of physiological conditions. This integrated understanding underscores the importance of cardiovascular regulation as both a marker of health and a target for therapeutic intervention.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Guyton AC, Hall JE (2021) Textbook of Medical Physiology, 14th ed, Elsevier pp. 230-235.
2. Berne RM, Levy MN, Koeppen BM, Stanton BA (2022) Physiology, 6th ed, Elsevier pp. 418-420.
3. Katz AM (2002) The Frank-Starling Law of the Heart: Mechanisms and Clinical Implications, J. Am. Coll. Cardiol 40(12): 1850-1858.
4. Dampney RAL (2017) Central Mechanisms Regulating Coordinated Cardiovascular and Respiratory Function During Exercise, Am J Physiol Regul Integr Comp Physiol 312(4): R520-R526.
5. Joyner MJ Casey DP (2015) Regulation of Increased Blood Flow (Hyperemia) to Muscles During Exercise: A Hierarchy of Competing Physiological Needs. Physiol Rev 95(2): 549-601.
6. Goldstein DS (2010) Catecholamines and the Integrated Stress Response, Cell Mol Neurobiol 30(8): 1433-1444.
7. Fleg JL, Lakatta EG (1988) Role of Muscle Blood Flow in the Age-Associated Reduction in Maximal Aerobic Capacity, J Appl Physiol 65(2): 874-881.
8. Hellsten Y, Nyberg M (2015) Cardiovascular Adaptations to Exercise Training, Compr Physiol 6(1): 1-32.
9. Imai K (1994) Vagal Activity and Cardiac Function in the Recovery Phase After Exercise, Am J Physiol 267(1): H23-H29.
10. Mortensen SP, Saltin B (2014) Regulation of the Skeletal Muscle Blood Flow in Humans, Exp Physiol 99(12): 1552-1558.