Review

Cardiovascular adaptation and dysfunction in fighter pilots exposed to G-Forces and hypobaric environments: a comprehensive review

Mohammad Moradi [1,*], *Hanieh Habibi Jirdehi* [1]

[1] Department of Biomedical Engineering, Faculty of Khajeh Nasir al-Din Tusi Faculty, Tabriz University of Technology (Sahand), Sahand, Tabriz, Iran

*** Corresponding Author:**
Address: Tabriz University of Technology (Sahand), Sahand, Tabriz, Iran. **Postal code:** 5331817634; **Email:** Moradim506@yahoo.com

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Abstract

Objectives: Military fighter pilots routinely experience extreme physiological stressors, including high acceleration forces (+Gz), hypobaric hypoxia, rapid atmospheric pressure changes, and intense operational workloads. These environmental challenges impose substantial demands on the cardiovascular system and may affect cardiac function, autonomic regulation, vascular adaptation, and long-term cardiovascular health. Understanding the cardiovascular consequences of these stressors is essential for optimizing pilot performance, improving flight safety, and guiding aerospace medicine practice. This review study aimed to critically evaluate the effects of acceleration forces and atmospheric pressure changes on cardiovascular physiology in fighter pilots, with particular emphasis on hemodynamic regulation, autonomic nervous system responses, hypoxia-related cardiac effects, decompression sickness, and long-term cardiovascular adaptation.

Methods: This literature review utilized peer-reviewed publications indexed in major scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar. The samples included all studies published from 1986 to early 2025, including experimental and clinical studies, physiological research, aerospace medicine reports, and review articles examining cardiovascular responses to acceleration stress and hypobaric exposure.

Results: Exposure to positive acceleration (+Gz) produces significant cardiovascular alterations through blood redistribution toward the lower extremities, reduced venous return, decreased stroke volume, and diminished cerebral perfusion. Recent evidence suggests that improved G tolerance is mediated primarily by peripheral vascular adaptation rather than solely by enhanced baroreflex sensitivity. High-altitude exposure causes hypobaric hypoxia, increasing heart rate, elevating cardiac output, and enhancing sympathetic activation to preserve tissue oxygenation. Decompression sickness remains a potentially serious complication, with cardiovascular manifestations ranging from pulmonary vascular obstruction to coronary gas embolism. Heart rate variability studies consistently demonstrate increased sympathetic nervous system activity during military flight, with autonomic disturbances persisting for several hours after mission completion. Despite these acute physiological stresses, current evidence indicates that most healthy fighter pilots maintain preserved long-term cardiovascular function, although continued surveillance remains necessary.

Conclusions: The cardiovascular system exhibits remarkable adaptive capacity in response to the combined effects of acceleration forces and hypobaric environments encountered in military aviation. However, these stressors generate substantial acute hemodynamic and autonomic challenges that may affect operational performance and, in rare cases, contribute to serious cardiovascular events. Continued research into vascular adaptation, autonomic regulation, hypoxia tolerance, and long-term cardiovascular outcomes is required to improve pilot health, optimize training strategies, and enhance flight safety.

Keywords: Fighter pilots; G-force; +Gz acceleration; Cardiovascular physiology; Heart rate variability; Hypobaric hypoxia; Decompression sickness; Aerospace medicine; Military aviation; Autonomic nervous system

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Introduction

Modern military aviation exposes fighter pilots to some of the most demanding physiological conditions encountered in any occupational environment. Unlike civilian aviation, high-performance military flight requires repeated exposure to extreme acceleration forces, rapid altitude transitions, hypobaric environments, intense cognitive workload, and substantial psychological stress. These factors impose considerable demands on the cardiovascular system, which is essential for maintaining systemic perfusion, cerebral blood flow, and oxygen delivery during flight operations [1, 2]. Advances in aircraft technology have substantially increased the operational capabilities of modern fighter aircraft. Contemporary fourth- and fifth-generation combat aircraft can generate sustained acceleration loads exceeding +9 Gz during aerial combat maneuvers, tactical evasive actions, and high-performance turns. Utilizing anti-G suits, pressure-breathing systems, and advanced cockpit ergonomics have all enhanced pilot safety. However, maintaining cardiovascular compensation is still crucial to ensure physiological stability in these demanding conditions. Failure of these compensatory mechanisms may result in visual disturbances, cognitive impairment, G-induced loss of consciousness (G-LOC), mission failure, and potentially fatal aviation accidents [3]. Among the acceleration profiles encountered during military flight, positive acceleration along the head-to-foot axis (+Gz) is considered the most physiologically challenging. During exposure to +Gz forces, hydrostatic pressure gradients increase markedly, causing blood to redistribute toward the lower extremities and abdominal vasculature. This redistribution reduces venous return, ventricular preload, stroke volume, and ultimately cardiac output. As cerebral perfusion pressure declines, pilots may experience peripheral vision loss, gray-out, black-out, and eventually G-induced loss of consciousness if compensatory responses become insufficient [1,4]. To maintain cardiovascular homeostasis during acceleration exposure, the autonomic nervous system activates coordinated compensatory mechanisms. Arterial baroreceptors located within the carotid sinus and aortic arch detect reductions in arterial pressure and initiate reflex sympathetic activation with parasympathetic withdrawal. These responses increase heart rate, myocardial contractility, and peripheral vasoconstriction while redistributing blood flow toward vital organs, particularly the brain and heart [5]. Enhanced autonomic regulation has traditionally been considered the main mechanism for improved G tolerance; more recent evidence suggests that peripheral vascular adaptation, especially within the lower extremities, plays a major role in improving tolerance following repeated acceleration exposure [6].In addition to acceleration stress, fighter pilots frequently experience hypobaric environments associated with high-altitude flight. As altitude increases, ambient atmospheric pressure and inspired oxygen partial pressure decline, resulting in hypobaric hypoxia. Acute hypoxic exposure induces compensatory cardiovascular responses including increased heart rate, cardiac output, sympathetic activation, and coronary blood flow to preserve tissue oxygen delivery [7, 8]. Although these mechanisms generally maintain adequate oxygenation during short-term exposure, prolonged or severe hypoxia may impair cardiovascular function, reduce exercise capacity, and compromise operational performance. Hypobaric exposure may also increase the risk of decompression sickness (DCS), particularly during rapid decompression or high-altitude operations. DCS results from inert gas bubble formation within blood and tissues following reductions in ambient pressure. Although neurological and musculoskeletal manifestations are most common, cardiovascular complications may also occur. Intravascular gas emboli can increase pulmonary vascular resistance, impair right ventricular function, damage the vascular endothelium, and, in rare cases, obstruct coronary circulation, resulting in myocardial ischemia or cardiac arrhythmias [9, 10].Recent aerospace physiology research has increasingly focused on autonomic regulation during military flight. Heart rate variability (HRV), a non-invasive measure of beat-to-beat cardiac rhythm fluctuations, has become an important tool for evaluating autonomic nervous system activity. HRV provides valuable information regarding the balance between sympathetic and parasympathetic regulation and has been widely used to assess physiological stress, workload, fatigue, and recovery in military aircrew [11]. Reduced HRV generally reflects increased sympathetic activation and reduced autonomic flexibility, both of which may adversely influence pilot performance and cardiovascular resilience. Studies involving military pilots consistently demonstrate that both real and simulated flight operations induce significant autonomic alterations characterized by increased sympathetic activity and reduced parasympathetic modulation. Moreover, these autonomic disturbances may persist for several hours after landing, indicating that physiological recovery continues after flight

operation completion [12, 13]. Various interacting environmental and individual factors influence cardiovascular responses during military flight. These factors include acceleration stress, hypobaric hypoxia, dehydration, thermal stress, fatigue, psychological workload, physical conditioning, and individual cardiovascular characteristics. These factors collectively influence physiological performance and susceptibility to cardiovascular events. Understanding these interactions is essential to optimize pilot training, improve aeromedical screening, enhance operational performance, and reduce cardiovascular risk in military aviation. Therefore, this review critically evaluated the effects of G-forces and hypobaric exposure on cardiac function, hemodynamic regulation, vascular adaptation, and autonomic nervous system activity in fighter pilots. By synthesizing evidence published between 1986 and early 2025, this review provides an updated overview of the physiological mechanisms, identifies current knowledge gaps, and highlights priorities for future research in aerospace cardiovascular medicine.

Physiological Basis: Cardiovascular Response to Gravity Changes Physical Basis of G-Force

Understanding the cardiovascular effects of military flight requires a clear understanding of acceleration physiology and its influence on systemic hemodynamics. Acceleration force, commonly expressed as gravitational force (G-force), is the inertial force generated when the body experiences Earth's gravitational field. One G corresponds to the normal gravitational acceleration on Earth, which is 9.81 m/s². Meanwhile, fighter pilots may routinely experience acceleration several times greater during aerial combat maneuvers and tactical flight operations [1, 3]. In aerospace physiology, acceleration is described using a three-axis coordinate system: the x-axis extends from the chest to the back, the y-axis from one shoulder to the other, and the z-axis from the head to the feet. Among these, positive acceleration along the head-to-foot axis (+Gz) has the greatest physiological significance because of its profound effects on blood distribution and cerebral perfusion [1]. Exposure to +Gz increases hydrostatic pressure gradients within the vascular system, causing blood to pool in the lower extremities and abdominal circulation. Consequently, venous return decreases, ventricular preload is reduced, stroke volume falls through the Frank-Starling mechanism, and cardiac output declines. Arterial pressure at the level of the brain decreases because the hydrostatic distance between the heart and cerebral circulation increases progressively, compromising cerebral perfusion as acceleration increases [4, 14]. The resulting physiological responses occur along a predictable continuum. Initial reductions in retinal perfusion produce peripheral visual field loss ("gray-out"), followed by complete visual loss ("black-out"). When cerebral perfusion drops below the critical threshold needed to maintain consciousness, G-induced loss of consciousness (G-LOC) occurs, representing one of the most serious physiological hazards in military aviation because it can result in complete pilot incapacitation during flight [3]. The magnitude and duration of acceleration exposure are the main determining factors of cardiovascular stress. During routine military flight training, pilots are commonly exposed to approximately 3–4 Gz, levels that are generally tolerated through normal cardiovascular compensation. However, advanced tactical maneuvers frequently generate sustained acceleration approaching 7–9 Gz, substantially increasing cardiovascular strain and the risk of cerebral hypoperfusion. To improve G tolerance, fighter pilots employ anti-G straining maneuvers (AGSM), pressure-breathing systems, and anti-G garments, all of which reduce venous pooling and help maintain cerebral arterial pressure [1,15]. Emergency aircraft ejection represents an even more extreme acceleration event. Although exposure lasts only fractions of a second, ejection seats generate exceptionally high acceleration forces that produce abrupt biomechanical and cardiovascular stress. These forces induce rapid fluctuations in arterial pressure, marked sympathetic activation, and transient cardiac rhythm disturbances. Despite significant improvements in modern ejection-seat technology, emergency escape remains an important physiological challenge in aerospace medicine [3]. Individual tolerance to acceleration varies considerably, and hydration status,

physical fitness, body composition, fatigue, age, autonomic responsiveness, and underlying cardiovascular health can influence it. Repeated acceleration training improves G tolerance through multiple adaptive mechanisms, including enhanced cardiovascular regulation, improved AGSM effectiveness, and peripheral vascular adaptations that reduce excessive blood pooling during +Gz exposure [6,14].Overall, current evidence indicates that acceleration should be regarded not merely as a mechanical force but as a complex physiological stressor involving coordinated cardiovascular, autonomic, cerebrovascular, respiratory, and musculoskeletal responses. Understanding these physiological principles provides the basis for interpreting both adaptive and pathological cardiovascular responses observed in fighter pilots during high-performance flight.

Immediate Physiological Response to +Gz

Exposure to positive head-to-foot acceleration (+Gz) produces rapid cardiovascular adjustments aimed at maintaining systemic arterial pressure and cerebral perfusion. The primary challenge during +Gz exposure is the redistribution of blood volume from the thoracic compartment toward the lower extremities and splanchnic circulation due to increased hydrostatic pressure gradients. This venous pooling reduces central blood volume, decreases venous return, and subsequently lowers ventricular preload and stroke volume through the Frank–Starling mechanism [2, 4]. When cardiac output decreases, the brain's blood supply is progressively affected, as the brain is above the heart. Reduced cerebral and retinal blood flow produces a characteristic sequence of visual disturbances, including peripheral vision loss, gray-out, tunnel vision, and, in severe cases, G-induced loss of consciousness (G-LOC). G-LOC can cause complete pilot incapacitation during critical flight phases, making it a major physiological risk associated with high-performance military aviation [3]. The cardiovascular system rapidly activates compensatory mechanisms to counteract the hemodynamic changes, mediated primarily by the autonomic nervous system. When baroreceptors in the carotid sinus and aortic arch detect reduced arterial pressure, sympathetic activity increases while parasympathetic influence decreases. The resulting tachycardia, enhanced myocardial contractility, and peripheral vasoconstriction help maintain arterial pressure and preserve blood flow to vital organs, particularly the brain and heart [5]. Repeated exposure to acceleration stress may induce adaptive cardiovascular responses. Although improved +Gz tolerance has traditionally been attributed mainly to enhanced autonomic regulation, evidence from military aviators suggests that cardiovascular adaptation may also involve improved peripheral vascular responses and more efficient blood pressure regulation during acceleration exposure [4,6]. However, these compensatory mechanisms have physiological limitations. During sustained or high-intensity acceleration, reductions in venous return and cardiac output may exceed the capacity of cardiovascular regulation, resulting in cerebral hypoperfusion, impaired visual function, cognitive deterioration, and eventual G-LOC. Understanding these acute cardiovascular responses is therefore essential for optimizing anti-G protection systems, pilot training strategies, and operational safety in modern military aviation.

Role of the Baroreflex System in G-Adaptation

Fighter pilots' capability to tolerate repeated exposure to high +Gz acceleration depends on integrated cardiovascular adaptations that preserve arterial pressure and cerebral perfusion during gravitational stress. The arterial baroreflex is the primary short-term mechanism maintaining cardiovascular stability during acceleration by regulating heart rate, myocardial contractility, and peripheral vascular resistance in response to changes in arterial pressure [5]. Historically, improved acceleration tolerance has been largely attributed to enhanced autonomic regulation and baroreflex adaptation. Newman et al. found that experienced fighter pilots exhibited greater cardiovascular responses during orthostatic stress compared to non-pilot controls. This suggests that experienced fighter pilots have better blood pressure regulation and cardiovascular compensation due to their repeated exposure to acceleration environments. [4].These findings contributed to the traditional view that enhanced baroreflex-mediated responses are an important component of G adaptation. However, recent evidence indicates that improved +Gz tolerance cannot be explained solely by changes in autonomic regulation. Eiken et al. investigated the effects of repeated +Gz exposure training and reported a significant improvement in rapid-onset-rate (ROR) G tolerance after five weeks of training. Importantly, this improvement was associated with increased arterial resistance and arterioles in the lower extremities, while systemic cardiovascular responses showed limited

changes [6]. These findings suggest that local vascular adaptations in dependent regions may represent a major mechanism underlying long-term G tolerance. Repeated exposure to elevated hydrostatic pressure during +Gz loading may induce functional and structural changes in lower-limb vasculature, reducing excessive vascular distension and limiting blood pooling during subsequent acceleration exposure. Improving peripheral vascular resistance may help maintain central blood volume and cerebral perfusion without enhancement of baroreflex sensitivity [6]. Nevertheless, the contribution of the baroreflex system remains essential during acute acceleration exposure. Rapid reductions in arterial pressure during +Gz loading activate sympathetic responses within seconds, producing tachycardia, increased myocardial contractility, and peripheral vasoconstriction.

Heart Rate Variability Studies

Heart rate variability (HRV) has become an important non-invasive method for assessing autonomic nervous system regulation in military aviation. By reflecting variations in consecutive cardiac intervals, HRV provides information regarding the balance between sympathetic and parasympathetic activity and has been widely used to evaluate physiological stress, workload, fatigue, and recovery in pilots [11]. Military flight imposes considerable autonomic demands due to the combined effects of acceleration stress, environmental challenges, cognitive workload, and operational pressure. Recent evidence indicates that flight exposure is associated with measurable alterations in autonomic regulation, characterized mainly by increased sympathetic activity and reduced parasympathetic modulation. These changes suggest that HRV may provide a sensitive marker of cardiovascular strain during demanding aviation operations [13 A recent scoping review by Soares et al. evaluated HRV responses in military pilots during real and simulated flight conditions and demonstrated consistent autonomic alterations following flight exposure. The authors reported that several HRV parameters, including RMSSD, SDNN, LF/HF ratio, and nonlinear indices such as SD1, were associated with changes in operational workload and flight demands. Importantly, some studies indicated that autonomic changes persisted beyond mission completion, suggesting that recovery of cardiovascular autonomic balance may require additional time after demanding flight activities [13]. Earlier research by Sauvet et al. also demonstrated prolonged autonomic effects following military flight operations. The findings revealed persistent sympathetic predominance during the post-flight period, suggesting that cardiovascular recovery may continue after active mission completion. This observation is particularly relevant for military aviation, where pilots may perform repeated missions within short intervals and may therefore experience cumulative physiological stress [12]. Beyond assessing cardiovascular stress, HRV has shown potential as an objective indicator of pilot workload and operational readiness. Changes in autonomic modulation may reveal physiological demands that are not always apparent from performance measures alone. Therefore, HRV monitoring may provide additional information for evaluating pilot fatigue, recovery status, and adaptation to repeated flight exposure [11, 13]. Overall, current evidence indicates that HRV is a valuable tool for investigating autonomic responses to military aviation stress. Increased sympathetic activation, reduced parasympathetic modulation, and delayed autonomic recovery appear to be common responses following demanding flight operations. Incorporating HRV assessment into aerospace medicine programs may improve understanding of pilot physiological strain and help implement strategies to optimize performance, recovery, and long-term cardiovascular health.

Effects of Atmospheric Pressure on Cardiac Function

Hypobaric Hypoxia and Cardiac Response (Revised)

Exposure to hypobaric environments poses a significant physiological challenge for fighter pilots because reduced atmospheric pressure at high altitude decreases the partial pressure of inspired oxygen and compromises oxygen availability to tissues. Above approximately 10,000 feet, these changes become physiologically significant and require rapid cardiovascular adaptation to maintain systemic oxygen delivery [7, 8].The primary cardiovascular response to acute hypobaric hypoxia involves sympathetic nervous system activation. Sympathetic nervous system activation increases heart rate, myocardial contractility, and cardiac output. These compensatory mechanisms help address the shortage in arterial oxygen content, ensuring that vital organs—especially the brain and heart—continue to receive an adequate oxygen supply [7, 8]. Coronary vasodilation may also occur during hypoxia to maintain myocardial oxygen supply; however, the effectiveness of these adaptations depends on the severity and duration of exposure [8]. Altitude exposure also induces

redistribution of blood flow, with preferential maintenance of perfusion to oxygen-sensitive organs such as the brain and heart. Although these responses are generally effective during short-term exposure, prolonged or severe hypoxia may exceed cardiovascular compensatory capacity and contribute to impaired myocardial function, reduced exercise tolerance, and electrical instability [7, 8]. Hypoxia-induced pulmonary vasoconstriction represents another important cardiovascular response. While this mechanism improves ventilation–perfusion matching, excessive pulmonary vascular constriction increases pulmonary vascular resistance and right ventricular workload, potentially contributing to cardiovascular strain during prolonged altitude exposure [7]. Individual variability significantly influences tolerance to hypobaric hypoxia. Rahadyan et al. demonstrated that cardiac morphology may contribute to differences in hypoxia tolerance among individuals exposed to simulated high altitude. In their study, trained participants showed shorter time of useful consciousness compared to untrained individuals, suggesting that greater cardiovascular fitness does not necessarily guarantee superior protection against acute hypoxic stress [8]. These findings highlight that hypoxia tolerance depends on multiple interacting factors, including cardiac structure, autonomic regulation, oxygen consumption, and individual physiological characteristics. From an operational perspective, hypobaric hypoxia remains a critical concern in military aviation because cognitive impairment, delayed decision-making, and reduced situational awareness may develop before pilots recognize oxygen deficiency. Therefore, understanding cardiovascular adaptation to hypoxia is essential to improving pilot screening, optimizing protective equipment, and lowering aviation-related hypoxia incidents [7, 8].

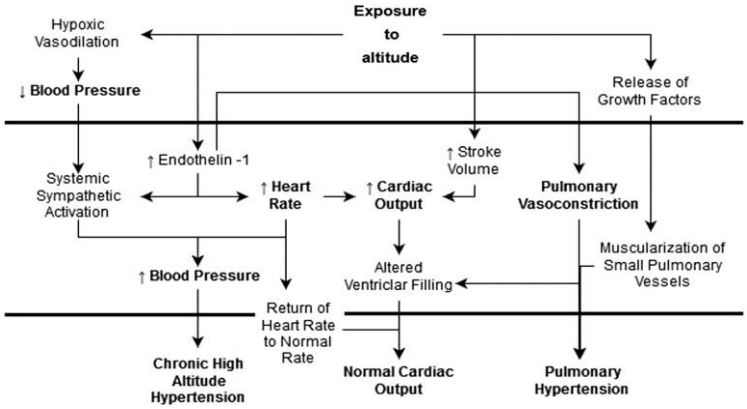

Figure 1. Cardiovascular adaptations during hypobaric hypoxia including increased heart rate, sympathetic activation, and altered pulmonary vascular resistance.

Decompression Sickness and Cardiac Complications

Decompression sickness (DCS) represents one of the most serious physiological hazards associated with high-altitude aviation and aerospace operations. Although modern aircraft pressurization systems have substantially reduced DCS incidence, the condition remains a significant concern during high-altitude military flights, rapid cabin depressurization events, and operations requiring exposure to extreme altitudes. The underlying mechanism involves inert gas bubble formation, predominantly nitrogen, within blood and tissues as ambient pressure decreases. According to Henry's law, gases dissolved in body fluids under pressure become supersaturated during decompression, promoting free gas bubble formation that can interfere with normal physiological processes [9]. The pathophysiology of DCS extends beyond simple mechanical obstruction by gas bubbles. Contemporary research indicates that intravascular bubbles trigger a complex cascade of biological responses. These biological responses include endothelial dysfunction, platelet activation, coagulation abnormalities, inflammatory mediator release, and microvascular injury. These processes may amplify tissue damage even when bubble burden is relatively low. Consequently, DCS is recognized as both a mechanical and inflammatory vascular disorder rather than merely a consequence of gas embolization [9]. One of the most serious cardiovascular manifestations of DCS is arterial gas embolism involving the coronary circulation.

Although relatively uncommon, coronary gas embolism can occur when bubbles enter the arterial system directly or pass from the venous circulation through intracardiac or intrapulmonary right-to-left shunts. Once within the coronary arteries, gas emboli may obstruct blood flow and produce acute myocardial ischemia. Depending on the location and extent of vascular occlusion, affected individuals may develop chest pain, electrocardiographic abnormalities, myocardial infarction, or severe impairment of ventricular function. In extreme cases, coronary embolization can precipitate cardiogenic shock and sudden cardiovascular collapse [10, 9]. Cardiac rhythm disturbances constitute another important potential consequence of decompression-related gas embolization. Bubble-induced myocardial ischemia, alterations in autonomic regulation, and direct interference with myocardial microcirculation may increase electrical instability within cardiac tissue. Reported manifestations include premature atrial and ventricular contractions, conduction abnormalities, supraventricular tachyarrhythmias, and, in severe cases, life-threatening ventricular arrhythmias. Although clinically significant arrhythmias are uncommon, their occurrence in high-performance aviation environments may have serious operational implications because even brief episodes of cardiovascular instability can impair pilot performance and flight safety [9]. The clinical presentation of cardiopulmonary DCS is often variable and may overlap with other altitude-related disorders. Common symptoms include dyspnea, tachypnea, substernal or retrosternal chest discomfort, cough, fatigue, and exercise intolerance. More severe cases may present with cyanosis, hypotension, altered mental status, or signs of circulatory shock. Because neurological and musculoskeletal manifestations frequently coexist with cardiopulmonary symptoms, diagnosis may be challenging, particularly during aviation operations where multiple environmental stressors are present simultaneously [10]. An important factor influencing susceptibility to cardiovascular complications is the magnitude and rate of decompression. Rapid decompression events produce more inert gas supersaturation and therefore increase the likelihood of bubble formation. Individual factors such as age, hydration status, physical activity, body composition, and intracardiac shunts may further modify risk. In military aviation, pre-breathing protocols involving 100% oxygen before high-altitude exposure are frequently used to reduce tissue nitrogen stores and minimize the probability of decompression-related complications [16]. Prompt recognition and treatment are essential because delayed management may result in irreversible tissue injury. Hyperbaric oxygen therapy (HBOT) remains the definitive treatment for significant DCS and arterial gas embolism. Boyle's law states that recompression reduces bubble volume, while administration of high-concentration oxygen accelerates nitrogen elimination and improves tissue oxygenation. In addition, oxygen therapy may attenuate inflammatory responses and support recovery of ischemic tissues. Early HBOT initiation has been consistently associated with improved clinical outcomes and reduced long-term complications [9, 10]. Overall, although severe cardiovascular manifestations of decompression sickness are relatively rare compared with neurological or musculoskeletal forms of DCS, their potential consequences are profound. Coronary gas embolism, pulmonary vascular obstruction, right ventricular overload, and cardiac arrhythmias represent potentially life-threatening complications that require immediate recognition and intervention. Therefore, understanding the cardiovascular effects of decompression is essential in aerospace medicine and high-altitude flight safety.

Push–Pull Effect

The push–pull effect is a critical physiological phenomenon encountered during high-performance flight maneuvers in which exposure to negative acceleration (−Gz) or near-weightless conditions is followed by rapid transition to positive acceleration (+Gz). This sequence markedly reduces acceleration tolerance and increases the risk of cerebral hypoperfusion and G-induced loss of consciousness (G-LOC) compared with isolated +Gz exposure [17]. During normal +Gz exposure, cardiovascular compensation depends on rapid activation of sympathetic mechanisms, peripheral vasoconstriction, and maintenance of arterial pressure through baroreflex-mediated responses. However, during −Gz exposure, transient increases in cephalic blood volume and vascular alterations impair the cardiovascular system's ability to compensate effectively during subsequent positive acceleration. Consequently, the transition from −Gz to +Gz results in a more pronounced reduction in cerebral perfusion pressure and increases the likelihood of visual impairment and G-LOC [17]. Several experimental studies have found that the push–pull effect is primarily associated with

impaired vascular regulation and reduced effectiveness of mechanisms that maintain cerebral blood flow during rapid acceleration transitions. These findings highlight the importance of peripheral vascular responses in determining acceleration tolerance during complex flight maneuvers [17]. Recent human investigations have explored potential countermeasures against push–pull-induced cerebral hypoperfusion. Xing et al. demonstrated that temporary femoral artery occlusion during simulated push–pull maneuvers significantly reduced the decline in cerebral blood flow velocity and cerebral perfusion pressure. These findings suggest that limiting excessive blood pooling in the lower extremities may represent a potential strategy for preserving cerebral circulation during rapid changes in acceleration direction [18]. From an operational perspective, the push–pull effect remains a critical concern in fighter aviation because modern air-combat maneuvers frequently involve rapid changes in acceleration magnitude and direction. Therefore, acceleration physiology training emphasizes minimizing unnecessary negative-G exposure immediately before high positive-G maneuvers whenever operational conditions permit. Continued investigation of vascular and mechanical countermeasures may improve protection against acceleration-induced cerebral hypoperfusion and enhance pilot safety [19].

Effects of Ejection Shock on the Heart

Acceleration Force Magnitude during Ejection

Emergency ejection from a fighter aircraft represents one of the most extreme acceleration events encountered in military aviation. Unlike routine flight maneuvers, ejection exposes the pilot to a sudden high-intensity acceleration impulse designed to rapidly separate the occupant from the aircraft. Although modern ejection seat systems have substantially improved survival rates, the process still imposes considerable mechanical stress on multiple physiological systems, including the cardiovascular system [20]. The acceleration profile during ejection differs substantially from conventional flight-related acceleration exposure. While trained fighter pilots can tolerate sustained +Gz acceleration through physiological adaptation, anti-G protection systems, and straining maneuvers, ejection produces a short-duration acceleration pulse with rapid changes in body momentum and mechanical loading. The extremely rapid onset of these forces limits the effectiveness of cardiovascular compensatory mechanisms and may result in transient alterations in venous return, cardiac loading conditions, and arterial pressure regulation [20]. Mechanical stress during ejection is transmitted through the axial skeleton and thoracic structures. These forces may influence cardiovascular function through changes in intrathoracic pressure, displacement of blood volume, and transient alterations in autonomic activity. Although cardiovascular injury is less common than musculoskeletal trauma following ejection, the possibility of transient cardiovascular disturbances should be considered during medical evaluation of ejection survivors [20]. Besides direct mechanical effects, cardiovascular responses after ejection may be influenced by concurrent physiological stressors, including hypoxia, emotional stress, and acceleration exposure. These factors can increase sympathetic activation and myocardial workload during an already demanding physiological event. Modern ejection systems are therefore designed not only to maximize escape probability but also to reduce peak acceleration forces and distribute mechanical loads more effectively across the body. More research on the interaction between acceleration forces, cardiovascular regulation, and mechanical stress is essential to improve pilot protection and reduce the physiological consequences of emergency escape systems [20].

Age-Related Cardiovascular Responses to Ejection Stress

Age is an important determinant of cardiovascular performance and physiological adaptation to extreme acceleration environments. Most military aviators are relatively young and medically fit; however, increasing age is associated with structural and functional changes in the cardiovascular system that may influence tolerance to ejection-related acceleration forces. These changes include reduced arterial compliance, alterations in autonomic nervous system regulation, diminished β-adrenergic responsiveness, and decreased cardiovascular reserve [21, 22]. The cardiovascular response to sudden acceleration relies on the quick activation of autonomic compensatory mechanisms that help maintain arterial pressure and ensure adequate tissue perfusion. During ejection, acceleration forces activate within milliseconds, causing rapid hemodynamic disturbances that necessitate immediate cardiovascular adjustments. Aging has been shown to negatively affect several aspects of this response, particularly baroreflex sensitivity and vascular responsiveness. Monahan noted that as people age, the progressive decline in arterial

baroreflex function diminishes the body's ability to adapt to sudden alterations in blood pressure[9].Such alterations may theoretically decrease cardiovascular resilience during extreme acceleration events. Structural vascular changes also contribute to age-dependent differences in physiological responses. Aging is associated with progressive arterial stiffening, increased pulse-wave velocity, and reduced elasticity of large arteries. These changes affect pressure wave transmission throughout the circulation and may impair the rapid cardiovascular adjustments required during acceleration exposure [21].Furthermore, age-related endothelial dysfunction can reduce vasodilatory capacity and alter regional blood flow regulation during periods of physiological stress [20]. Research on aerospace physiology has consistently demonstrated that acceleration tolerance depends on the integrated performance of the cardiovascular, autonomic, and cerebrovascular systems. Although direct investigations of age-related responses during ejection-seat acceleration remain limited, studies examining orthostatic stress, centrifuge exposure, and G tolerance suggest that older individuals may exhibit diminished cardiovascular adaptability compared with younger subjects. Reduced autonomic responsiveness and lower baroreflex gain may contribute to less effective maintenance of arterial pressure during sudden hemodynamic challenges [4, 14]. Age-related alterations in heart rate regulation may also influence adaptation to acceleration stress. Healthy aging is associated with reductions in maximal heart rate, decreased chronotropic responsiveness, and diminished sympathetic reserve. As a result, older pilots may slowly adapt to acceleration stress and are unable to increase cardiac output when faced with acute physiological challenges. Maintaining cerebral perfusion during acceleration relies on instant cardiovascular responses. Therefore, these limitations could potentially heighten the risk of performance declines related to acceleration [19]. These observations have important operational implications. These observations have important operational implications. As military aviation personnel get older and remain active, understanding age-related physiological changes becomes increasingly relevant for pilot selection, training, and medical surveillance. Current evidence suggests that individualized assessment of cardiovascular fitness, autonomic function, and acceleration tolerance may become increasingly important with advanced age. More research is needed to investigate age-related responses to ejection-seat acceleration and to develop evidence-based recommendations for training protocols and operational risk management.

Mechanisms of Ejection-Related Cardiac Injury

Cardiac injury associated with aircraft ejection is a multifactorial process resulting from the interaction of extreme acceleration forces, abrupt hemodynamic alterations, intense autonomic activation, and mechanical stress transmitted through the thorax. Although modern ejection systems have substantially improved pilot survivability, the physiological demands imposed during escape remain among the most severe encountered in aerospace medicine [23, 24]. Immediately after ejection-seat activation, the pilot experiences a rapid acceleration impulse that may exceed 12–20 G over a very short duration. This sudden increase in inertial loading produces abrupt hemodynamic changes, including blood volume redistribution, transient reductions in venous return, and alterations in cardiac preload. Because acceleration develops within fractions of a second, cardiovascular compensatory mechanisms have only limited time to respond, increasing the physiological burden imposed on the heart [23, 24]. Arterial baroreceptors activate the sympathetic nervous system to adapt to fluctuations in blood pressure. This increases heart rate, myocardial contractility, and peripheral vasoconstriction, thereby helping to restore arterial pressure and maintain cerebral perfusion. These mechanisms are essential for survival during acceleration stress; however, they increase myocardial oxygen demand and cardiac workload [5, 14]. Besides hemodynamic alterations, the rapid propulsion of the ejection seat generates substantial compressive forces transmitted through the thorax and vertebral column. These mechanical loads primarily account for the high incidence of spinal compression fractures and thoracic musculoskeletal injuries observed after ejection. Although clinically significant cardiac injury is uncommon, transient mechanical stress may influence cardiac function through temporary alterations in intrathoracic pressure and myocardial loading conditions [23, 24]. The cardiovascular response to ejection may be further amplified by psychological stress, hypoxia, dehydration, and previous exposure to high +Gz acceleration before escape. These factors increase sympathetic activation and may further elevate myocardial oxygen consumption during an already demanding physiological event [5, 24]. Clinical follow-up studies of ejection survivors indicate that orthopedic

injuries remain the predominant medical consequence of ejection-seat escape. Nevertheless, cardiovascular evaluation remains an important component of post-ejection medical assessment because transient arrhythmias, myocardial strain, or other cardiovascular abnormalities may occasionally occur following severe acceleration exposure [23, 24]. Overall, the cardiovascular consequences of ejection result from the combined effects of abrupt hemodynamic changes, autonomic activation, and intense mechanical loading. Although serious cardiac injury is relatively uncommon compared with musculoskeletal trauma, understanding these physiological responses remains essential for improving pilot safety, optimizing post-ejection medical evaluation, and guiding the continued development of advanced ejection-seat systems [11, 23, 24].

Clinical Evidence: Field Studies and Case Reports
Comens et al.: Physiological Responses to Combat Missions

Comens and colleagues (1987) were the first field researchers investigating the cardiovascular effects of operational high-performance flight. Unlike centrifuge-based studies conducted under controlled laboratory conditions, this investigation evaluated physiological responses during actual fighter aircraft operations, providing valuable insight into the cardiovascular demands experienced during realistic combat training missions [25]. The study involved F-4C fighter pilots and aircrew participating in surface attack training (SAT) missions. Each mission consisted of six successive weapons-delivery maneuvers performed at approximately 80-second intervals, with progressively steeper dive profiles and pull-out phases generating acceleration loads of approximately +5.5 to +6 Gz. Continuous electrocardiographic monitoring was performed throughout the missions, and blood samples were collected before and after flight to evaluate biochemical markers of myocardial injury, including creatine phosphokinase (CPK), CPK isoenzymes, lactate dehydrogenase (LDH), LDH isoenzymes, myoglobin, and high-density lipoprotein cholesterol (HDL-C) [25]. The investigators found no significant post-flight increases in biochemical markers of myocardial injury. CPK and LDH isoenzyme concentrations remained essentially unchanged, suggesting that repeated exposure to high-G maneuvers during these training missions did not produce detectable myocardial cell damage in healthy aircrew [25]. Despite the absence of biochemical evidence of cardiac injury, several participants revealed transient electrocardiographic abnormalities. These included temporary ST-segment elevation or depression, T-wave inversion, sinus arrhythmia, and increased heart rate during or shortly after flight. Importantly, these findings were transient, resolved spontaneously, and were not accompanied by clinical symptoms or laboratory evidence of myocardial necrosis [25]. These observations indicate that the cardiovascular response to repeated high-performance flight is predominantly functional rather than structural. Increased sympathetic activation during flight contributes to elevations in heart rate, myocardial contractility, and cardiac output, thereby supporting maintenance of cerebral and systemic perfusion during acceleration exposure. At the same time, transient changes in ventricular repolarization may occur secondary to autonomic activation, altered intrathoracic pressure, or temporary changes in myocardial oxygen demand rather than permanent myocardial injury [5, 25]. From an aeromedical perspective, the study highlights the distinction between physiological adaptation and pathological cardiac injury. High-performance flight imposes considerable cardiovascular stress; healthy fighter pilots generally tolerate these demands without evidence of clinically significant myocardial damage. Nevertheless, the transient electrocardiographic changes observed during operational missions support the continued importance of cardiovascular surveillance, particularly in pilots with underlying cardiovascular risk factors or prolonged exposure to high-performance flying [25]. Overall, Comens and colleagues provided important field-based evidence that operational exposure to repeated +5.5 to +6 Gz acceleration induces measurable autonomic and electrophysiological responses without producing significant myocardial injury in healthy military aviators [25].

In-Flight Myocardial Infarction and Acute Coronary Events

Although acute cardiovascular events are uncommon among active military aviators, they remain an important aeromedical concern because even brief cardiovascular incapacitation during flight may have catastrophic operational consequences. Case reports have demonstrated that acute coronary syndromes may occur despite regular medical surveillance and high levels of physical fitness [26]. Manchanda and Narang described the case of a 43-year-old fighter

pilot who developed an acute myocardial infarction while on active flying duty. Despite satisfying aeromedical fitness requirements before the event, the pilot experienced symptoms consistent with acute coronary ischemia during military flight operations. This case illustrates that clinically silent coronary artery disease may remain undetected until physiological stress associated with flight exceeds the individual's cardiovascular reserve [26]. Exposure to high +Gz acceleration is associated with substantial increases in heart rate, arterial pressure, myocardial contractility, and sympathetic nervous system activity. These responses increase myocardial oxygen consumption and cardiac workload. In individuals with underlying coronary artery disease, even if previously asymptomatic, such physiological stress may reduce myocardial oxygen supply-demand balance and potentially contribute to ischemic events [27]. These observations reinforce current aeromedical recommendations emphasizing periodic cardiovascular evaluation throughout a pilot's flying career. Current guidelines suggest assessing cardiovascular risk factors, blood pressure, lipid levels, exercise capacity, and electrocardiographic results. This approach facilitates early identification of coronary artery disease and reduces the risk of in-flight cardiovascular incapacitation [24, 28]. Overall, available clinical evidence indicates that acute coronary events remain rare among fighter pilots but have disproportionate operational significance because of their potential consequences for both pilot safety and mission success. Consequently, comprehensive cardiovascular screening and continuous medical surveillance remain essential components of military aviation medicine [24, 26, 28].

Long-Term Cardiovascular Health of Fighter Pilots

One of the most important questions in aerospace cardiovascular medicine is whether repeated exposure to acceleration stress, hypobaric environments, and operational workload results in long-term adverse cardiovascular consequences. While fighter pilots are routinely subjected to physiological conditions that challenge cardiovascular homeostasis, available evidence suggests that significant long-term cardiovascular disease is relatively uncommon among medically qualified aviators [24, 28]. Repeated exposure to acceleration forces produces transient increases in arterial pressure, sympathetic nervous system activity, and myocardial workload. Over many years of operational service, these physiological stresses may theoretically contribute to cardiovascular remodeling. However, distinguishing physiological adaptation from pathological cardiovascular remodeling remains challenging. Studies of fighter pilots indicate that cardiovascular adaptations are generally compatible with normal cardiac function and reflect adaptation to repeated occupational stress rather than overt cardiovascular disease [29]. Longitudinal investigations have generally reported favorable cardiovascular outcomes among fighter pilots. Froom and colleagues observed no increased incidence of hypertension during 12–15 years of follow-up among military fighter pilots, suggesting that long-term operational flying does not necessarily increase the risk of chronic hypertension or clinically significant cardiovascular disease [30]. Nevertheless, aging remains an important determinant of cardiovascular performance. Progressive reductions in arterial compliance, endothelial function, and baroreflex sensitivity may reduce cardiovascular reserve and influence physiological responses to acceleration stress [19–22]. Consequently, periodic cardiovascular surveillance remains essential throughout a pilot's flying career. Current aeromedical recommendations advocate risk-stratified cardiovascular assessment based on age, occupational exposure, and individual cardiovascular risk factors. Recommended evaluations include resting and exercise electrocardiography, echocardiography, laboratory assessment of cardiovascular risk factors, and advanced cardiovascular imaging when clinically indicated [24,28]. Overall, current evidence indicates that most medically qualified fighter pilots maintain normal cardiovascular function throughout their operational careers. However, to better understand the long-term effects of repeated exposure to acceleration, autonomic stress, and hypobaric environments on cardiovascular aging and disease risk, more ongoing longitudinal studies are needed.

Discussion

The present review synthesized evidence from experimental studies, field investigations, physiological experiments, clinical observations, and aerospace medicine literature to examine the cardiovascular effects of acceleration forces and atmospheric pressure changes in fighter pilots. Collectively, the available evidence demonstrates that cardiovascular responses to military flight are multifactorial and involve complex interactions

among autonomic regulation, vascular adaptation, cardiac function, and environmental stressors.

Theme 1. Peripheral Vascular Adaptation Appears to Be a Major Mechanism Underlying Improved G Tolerance

Recent evidence suggests that improvements in G tolerance depend not only on autonomic reflexes but also on adaptations within the peripheral vasculature. Prior studies emphasized enhanced baroreflex responsiveness as the main mechanism to improve tolerance after repeated +Gz exposure [4]. However, more recent centrifuge studies revealed that repeated hypergravity exposure increases vascular resistance in the lower extremities and improves rapid-onset G tolerance without measurable enhancement of carotid baroreflex sensitivity [6]. These findings suggest that peripheral vascular remodeling may play a more important role than previously recognized. From an operational perspective, these observations suggest that progressive acceleration exposure during pilot training may promote vascular adaptations contributing to improved acceleration tolerance. Further research is needed to characterize these vascular adaptations and determine how we can optimize them in military flight training.

Theme 2. Persistent Autonomic Activation and Recovery Requirements

Studies evaluating heart rate variability consistently demonstrate marked sympathetic activation during both real and simulated military flight operations. Importantly, alterations in autonomic balance frequently persist for several hours following flight completion, indicating that cardiovascular recovery extends beyond the active flight period [12, 13Persistent sympathetic predominance suggests that cumulative physiological stress may arise when demanding flights are conducted without adequate recovery time. Consequently, post-flight monitoring of autonomic recovery using HRV analysis may provide an objective tool to assess pilot readiness and optimize operational scheduling [11–13].

Theme 3. Decompression Sickness and Acceleration Stress Can Produce Serious Cardiovascular Consequences

Although relatively uncommon, decompression sickness and extreme acceleration exposure remain capable of producing serious cardiovascular complications. Intravascular gas bubbles formed during decompression may increase pulmonary vascular resistance, impair right ventricular function, and, in severe cases, produce coronary arterial gas embolism resulting in myocardial ischemia or cardiac arrhythmias [9, 10]. Likewise, emergency ejection exposes pilots to extremely high acceleration forces over a very short duration. Although clinically significant cardiac injury is uncommon, abrupt hemodynamic changes, sympathetic activation, and mechanical loading of the thorax may produce transient cardiovascular instability. These observations support continued cardiovascular evaluation following ejection and ongoing improvements in protective equipment [23, 24].

Theme 4. Age-Related Cardiovascular Responses to Flight Stress

Current evidence indicates that aging influences cardiovascular responses to acceleration mainly by reductions in arterial compliance, endothelial function, baroreflex sensitivity, and cardiovascular reserve [19–22]. Although direct evidence regarding age-related responses to ejection remains limited, findings from cardiovascular physiology suggest that these changes may reduce the efficiency of cardiovascular compensation during acceleration exposure [14, 19]. These observations support the importance of age-specific cardiovascular assessment within aeromedical surveillance programs and emphasize the need for additional longitudinal studies examining the interaction between aging, repeated acceleration exposure, and long-term cardiovascular adaptation in military aviators.

Practical Recommendations

The available evidence suggests several practical recommendations for aerospace medicine practitioners, military organizations, and flight training programs. First, comprehensive cardiovascular screening should include aeromedical evaluation. These evaluations should involve regular assessments of blood pressure, lipid profiles, exercise capacity, cardiac rhythm disturbances, and overall cardiovascular risk throughout a pilot's career. Age-related cardiovascular risk factors and the early detection of subclinical coronary artery disease need special attention [3, 22]. Second, flight schedules and operational planning should consider the persistence of autonomic activation after high-workload missions. Adequate recovery periods between flights may help facilitate autonomic normalization and reduce cumulative physiological stress [24, 28]. Third, there should be a continued focus on training in acceleration physiology. This includes educating

individuals on anti-G straining maneuvers, recognizing acceleration-related symptoms, and understanding conditions that may hinder G tolerance, such as dehydration, fatigue, sleep deprivation, and illness [11, 21]. Fourth, strict management of modifiable cardiovascular risk factors—including hypertension, dyslipidemia, obesity, diabetes mellitus, and tobacco use—should remain a priority. Even relatively low levels of cardiovascular risk may have important operational consequences in aviation environments where sudden incapacitation can be catastrophic [3, 22]. Finally, future research should focus on longitudinal cardiovascular follow-ups for military aviators, detailed assessment of vascular adaptation mechanisms, long-term autonomic consequences of repeated flight exposure, and developing novel physiological monitoring technologies capable of identifying early cardiovascular deterioration before it affects flight performance. Continued integration of cardiovascular science and aerospace medicine will further optimize pilot health, operational safety, and long-term outcomes for military aircrew.

Future Research Directions

Despite substantial advances in aerospace physiology, several important gaps remain in our understanding of cardiovascular adaptations to military flight environments. Most available studies have focused on acute physiological responses to acceleration stress, whereas relatively little is known about the cumulative cardiovascular effects of repeated exposure throughout an entire flying career. Longitudinal cohort studies following fighter pilots from early training through retirement are needed to determine whether repeated exposure to high-G maneuvers, hypobaric environments, and operational stress contributes to accelerate cardiovascular aging, vascular remodeling, or subclinical cardiac dysfunction [2, 21]. Another important area for future research is the integration of wearable physiological monitoring technologies into military aviation. Advances in miniaturized electrocardiographic sensors, continuous blood pressure monitoring, and real-time heart rate variability analysis may allow continuous assessment of cardiovascular strain during operational flight. Future studies should evaluate the feasibility, reliability, and operational value of incorporating these technologies into military aviation practice. Furthermore, future investigations should examine sex-related differences in cardiovascular responses to acceleration and hypobaric exposure. As the number of female military aviators continues to increase, a better understanding of potential differences in G tolerance, autonomic regulation, vascular adaptation, and cardiovascular recovery will become increasingly important [21]. Finally, additional research is needed to evaluate novel countermeasures against acceleration-induced cardiovascular impairment. To further enhance cardiovascular protection during high-performance flight, we should explore advanced anti-G straining techniques, advanced pressure-breathing systems, innovative compression technologies, and other physiological countermeasures, both in laboratory settings and under real operational conditions. [11, 21].

Conclusion

Military flight exposes fighter pilots to a unique combination of cardiovascular stressors, including high acceleration forces, hypobaric environments, hypoxia, decompression stress, and intense autonomic activation. Current evidence indicates that cardiovascular adaptation to repeated acceleration exposure involves both autonomic regulation and peripheral vascular adaptation, with recent studies highlighting the important contribution of local vascular mechanisms to improved G tolerance [8]. High-G maneuvers produce substantial hemodynamic alterations that challenge cerebral perfusion and cardiovascular stability, whereas hypobaric exposure imposes additional physiological stress through hypoxia and decompression-related mechanisms [17, 31]. Studies evaluating heart rate variability consistently demonstrate pronounced sympathetic activation during military flight, with autonomic recovery often extending beyond mission completion [24, 28]. Despite these physiological challenges, available evidence suggests that most healthy fighter pilots maintain normal cardiovascular function throughout their operational careers because of effective physiological adaptation, rigorous aeromedical selection, and continuous medical surveillance [3, 22]. Nevertheless, continued cardiovascular surveillance, age-appropriate screening, and well-designed longitudinal investigations are essential to optimize pilot health, maintain operational performance, and further improve flight safety [3, 21, 22].

Ethical Statements

This review article is based on previously published literature and does not involve any new experiments, human participants, animal studies, or collection of personal data. Therefore, ethical approval was not

required for this study.

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Authors' Contributions
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Conflicts of Interest
The authors declare that they have no conflicts of interest related to this manuscript.

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References

1. Green ND. Protection against long-duration acceleration. Ernsting's Aviation and Space Medicine. 4th ed. *Edward Arnold*. 2006:159-67.
2. Antuñano MJ, Wade K. Index of International Publications in Aerospace Medicine. *Federal Aviation Administration*. 2014.
3. Burton RR. G-induced loss of consciousness: definition, history, current status. *Aviat Space Environ Med*. 1998; 59(1); 2-5.
4. Newman DG, White SW, Callister R. Evidence of baroreflex adaptation to repetitive Gz in fighter pilots. *Aviat Space Environ Med*. 1998; 69(5):446-51.
5. Low PA, editor. Primer on the autonomic nervous system. *Academic Press*. 2011.
6. Eiken O, Keramidas ME, Sköldefors H, et al. Human cardiovascular adaptation to hypergravity. *Am J Physiol Regul Integr Comp Physiol*. 2022; 322(6):R597-R608.
7. Bärtsch P, Gibbs JS. Effect of altitude on the heart and the lungs. *Circulation*. 2007; 116(19):2191-202.
8. West JB. IX World Congress on High Altitude Medicine and Physiology, Taipei, Taiwan, November 3–6, 2012. *High Alt Med Biol*. 2012; 13(3):140.
9. Vann RD, Butler FK, Mitchell SJ, et al. Decompression illness. *Lancet*. 2011; 377(9760):153-64.
10. Kohshi K, Denoble PJ, Tamaki H, et al. Decompression Illness in Repetitive Breath-Hold Diving: Why Ischemic Lesions Involve the Brain? *Front Physiol*. 2021; 12:711850.
11. Shaffer F, Ginsberg JP. An overview of heart rate variability metrics and norms. *Front Public Health*. 2017; 5:258.
12. Sauvet F, Jouanin JC, Langrume C, et al. Heart rate variability in novice pilots during and after a multi-leg cross-country flight. *Aviat Space Environ Med*. 2009; 80(10):862-9.
13. Soares AB, Almeida MF, Franchini E, et al. Heart rate variability in military pilots during flight: a scoping review. *Mil Med*. 2025; 190(3-4):e515-e522.
14. Convertino VA. Status of cardiovascular issues related to space flight: Implications for future research directions. *Respir Physiol Neurobiol*. 2009; 169:S34-7.
15. Pollock RD, Hodkinson PD, Smith TG. Oh G: The x, y and z of human physiological responses to acceleration. *Exp Physiol*. 2021; 106(12):2367-2384.
16. Webb JT, Fischer MD, Heaps CL, et al. Exercise-enhanced preoxygenation increases protection from decompression sickness. *Aviat Space Environ Med*. 1996; 67(7):618-24.
17. Xing C, Wang X, Gao Y, et al. Lower body negative pressure protects brain perfusion in aviation gravitational stress induced by push–pull manoeuvre. *J Physiol*. 2020; 598(15):3173-3186.
18. Xing C, Gao Y, Wang X, et al. Cuff-Method Thigh Arterial Occlusion Counteracts Cerebral Hypoperfusion Against the Push–Pull Effect in Humans. *Front Physiol*. 2021; 12:672351.
19. Pollock RD, Hodkinson PD, Smith TG. Oh G: The x, y and z of human physiological responses to acceleration. *Exp Physiol*. 2021; 106(12):2367-2384.
20. Tripathy NK, Divya N, Raghunandan V. Windblast testing of an aircrew helmet: An approach to neck load analysis. *Ind J Aerospace Med*. 2019; 63(2):77-82.
21. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part I: aging arteries: a "set up" for vascular disease. *Circulation*. 2003; 107(1):139-46.
22. Lakatta EG, Levy D. Arterial and cardiac aging: major shareholders in cardiovascular disease enterprises: Part II: the aging heart in health: links to heart disease. *Circulation*. 2003; 107(2):346-54.
23. Sommer F, Gadjradj PS, Pippig T. Spinal injuries after ejection seat evacuation in fighter aircraft of the German Armed Forces between 1975 and 2021. *J Neurosurg Spine*. 2022; 38(2):271-278.
24. Rayman RB. Clinical aviation medicine. *Castle Connolly Graduate Medical*. 2000.
25. Comens P, Reed D, Mette M. Physiologic responses

of pilots flying high-performance aircraft. *Aviat Space Environ Med*. 1987; 58(3):205-10.
26. Nicol ED, Rienks R, Gray G, et al. An introduction to aviation cardiology. *Heart*. 2019; 105(Suppl 1):s3-s8.
27. Steptoe A, Kivimäki M. Stress and cardiovascular disease. *Nat Rev Cardiol*. 2012; 9(6):360-70.
28. Abela M, Borjesson M, Broekhuizen L, et al. Cardiac evaluation in the armed forces. A statement of the European Association of Preventive Cardiology of the ESC. *Eur J Prev Cardiol*. 2026:zwag323.
29. Grossman A, Wand O, Harpaz D, et al. Acceleration forces and cardiac and aortic indexes in jet fighter pilots. *Aviat Space Enviro Med*. 2011; 82(9):901-3.
30. Froom P, Gross M, Barzilay J, et al. Systolic blood pressure in fighter pilots after 12–15 years of service. *Aviat Space Environ Med*. 1986;57(4):367-9.