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The Hidden Link between Stress and Cardiovascular Health

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Abstract

Objective: To review the effects of psychological stress on cardiovascular disease (CVD), including basic mechanisms, clinical impact, assessment, and management strategies.

Design: Narrative review of current evidence on stress physiology and cardiovascular health.

Results: Chronic stress activates the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis, leading to inflammation, endothelial dysfunction, metabolic disturbances, hypertension, and atherosclerosis. Stress likewise contributes indirectly to cardiovascular risk through unhealthy behaviours such as smoking, poor diet, physical inactivity, obesity, poor sleep, and medication non-adherence. Acute stress may precipitate arrhythmias and Takotsubo cardiomyopathy. Screening tools such as the Perceived Stress Scale can help identify individuals at risk. Stress management approaches, including cognitive-behavioural therapy, mindfulness, breathing exercises, meditation, relaxation techniques, and habit adjustments, may improve psychological well-being and cardiovascular health.

Conclusion: Chronic stress is an important modifiable cardiovascular risk factor. Routine assessment and evidence-based stress management interventions may improve cardiovascular outcomes and general well-being.

Keywords: Stress, heart

Introduction

Stress is an inevitable aspect of human life and involves a range of emotional, psychological, and physical responses to situations perceived as challenging, threatening, or overwhelming. Although stress is often viewed primarily as a mental phenomenon, substantial evidence indicates that it significantly influences physical health, particularly the cardiovascular system [1]. Contemporary lifestyles characterized by occupational demands, financial difficulties, social isolation, caregiving burdens, educational pressures, and social uncertainty have contributed to increasing levels of chronic stress worldwide. Consequently, stress is now recognized not only as a source of emotional distress but also as an important and potentially modifiable risk factor for cardiovascular disease (CVD) [2].

The physiological stress response evolved as an adaptive survival mechanism that protects the organism during threatening circumstances. Exposure to a perceived threat activates complex neuroendocrine pathways that prepare the body for immediate action, commonly referred to as the "fight-or-flight" response [3]. This process mainly involves activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis, leading to the secretion of stress-related hormones, including adrenaline, noradrenaline, and cortisol [3]. These endocrine and autonomic changes produce transient increases in heart rate, blood pressure, glucose mobilization, and mental alertness, hence enhancing the body's capacity to react effectively to acute danger [3].

Under physiological conditions, the stress response is self-limiting, and homeostasis is restored once the stressor resolves [3]. Acute stress may therefore exert adaptive and beneficial effects by improving concentration, performance, and survival [3]. However, persistent or recurrent exposure to stressors results in prolonged activation of neuroendocrine pathways, preventing adequate physiological recovery. Chronic stress subsequently induces sustained alterations in inflammation, metabolism, autonomic function, and behaviour that contribute to adverse health outcomes [4].

Chronic stress has increasingly been implicated in the pathogenesis and progression of cardiovascular disease. Prolonged activation of stress-response systems promotes endothelial dysfunction, systemic inflammation, hypertension, autonomic imbalance, metabolic imbalance, and accelerated atherosclerosis [4]. In addition to these direct physiological effects, stress indirectly contributes to cardiovascular risk through maladaptive health behaviours, including smoking, physical inactivity, unhealthy dietary patterns, inadequate sleep, excessive alcohol consumption, medication non-adherence, and weight gain [5]. The interaction between these behavioural and biological mechanisms substantially increases the risk of hypertension, coronary artery disease, myocardial infarction, heart failure, arrhythmias, and cerebrovascular disease [5].

Importantly, the experience and perception of stress vary considerably among individuals. The magnitude of the stress response is influenced not only by the external stressor itself but also by cognitive appraisal and psychological interpretation of the event. Factors such as resilience, coping strategies, personality characteristics, social support, and prior life experiences significantly affect both stress perception and physiological reactivity [6]. Consequently, individuals exposed to similar stressors may display markedly different psychological and cardiovascular outcomes.

Recognition of the association between stress and cardiovascular disease has led to rising emphasis on stress assessment and management within primary care and lifestyle medicine. A range of evidence-based interventions [7], including cognitive-behavioural therapy (CBT), mindfulness-based practices, meditation, breathing exercises, relaxation techniques, yoga, and regular physical exercise, have demonstrated effectiveness in reducing stress-related symptoms and

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boosting cardiovascular outcomes. Given that stress represents a modifiable risk factor, its identification and management provide an important opportunity for cardiovascular disease prevention and the promotion of long-term health.

Mechanism of stress: Stress perception describes how individuals recognize, interpret, and respond to situations or events they perceive as stressful. The experience of stress is not determined solely by the presence of a difficult event, but rather by the individual's interpretation of the situation and their perceived ability to cope with it. As a result, two individuals may encounter the same circumstance, such as an academic examination, financial hardship, or workplace disagreement, yet respond to it in very different ways. Stress perception is therefore considered a subjective process affected by psychological, social, biological, and environmental factors.

The idea of stress perception is closely associated with the cognitive appraisal theory introduced by Richard Lazarus [8]. This theory proposes that stress arises when individuals believe that the demands of a situation exceed their available coping resources. The appraisal process occurs in two stages. First, during primary appraisal, the individual determines whether the event is threatening, harmful, or challenging. Second, during secondary appraisal, the person evaluates whether they possess sufficient resources or abilities to manage the circumstance effectively. Stress develops when the individual perceives their coping capacity as inadequate.

Personality traits and emotional strength significantly affect stress perception [9]. Individuals with cheerful and optimistic attitudes are more likely to view stressful experiences as manageable challenges rather than overwhelming threats. In contrast, individuals who experience anxiety, low self-confidence, or negative thinking patterns may perceive even minor problems as highly stressful. Past experiences additionally affect stress perception, as people who have successfully dealt with adversity previously may feel more capable of coping with future stressors.

Social and cultural influences add to how stress is perceived [10]. Supportive relationships with family, friends, and social networks can lessen the perceived severity of stressful situations by providing emotional comfort and practical assistance. Conversely, social isolation and unhealthy interpersonal relationships may increase feelings of stress. Cultural beliefs and social norms further shape stress perception by determining how individuals express and respond to emotional distress. In certain cultures, discussing stress openly is encouraged, whereas in others it may be discouraged or interpreted as a sign of weakness.

Biological mechanisms are closely involved in stress perception [11]. When a person perceives a threat, the brain initiates the sympathetic nervous system together with the hypothalamic–pituitary–adrenal (HPA) axis. This process stimulates the secretion of stress hormones, including adrenaline and cortisol, leading to bodily reactions such as increased heart rate, elevated blood pressure, rapid breathing, and enhanced alertness. These reactions are adaptive in acute situations because they prepare the organism for immediate action through the "fight-or-flight" response. However, prolonged activation resulting from chronic perceived stress can negatively affect both physical and psychological health.

Stress pathophysiology: Stress is a multifarious physical and mental process initiated within the brain when an individual perceives a situation as threatening, demanding, or challenging [1]. The stress response involves the coordinated interactions of multiple brain regions, neural circuits, endocrine pathways, and autonomic mechanisms that e the body to adapt to stressful stimuli.

Perception of Stress [12]

The stress response begins with the cognitive and affective interpretation of external or internal stimuli. Sensory information is processed by multiple important brain structures, including the amygdala, prefrontal cortex, and hippocampus. The amygdala plays a central function in evaluating the affective significance of stimuli, particularly fear and perceived danger. The prefrontal cortex contributes to rational assessment, decision-making, and regulation of emotional reactions, whereas the hippocampus compares current experiences with previous memories and situational information. When the amygdala interprets a situation as threatening, it initiates activation of the brain's stress-response pathways.

Activation of the Hypothalamus [12]

Signals from the amygdala are transmitted to the hypothalamus, which functions as the primary regulatory center of the stress response. The hypothalamus activates two major physiological systems involved in stress adaptation.

A. Sympathetic–Adrenal–Medullary (SAM) System [13]

The SAM system mediates the rapid "fight-or-flight" response. Activation of the sympathetic nervous system stimulates the adrenal medulla to secrete catecholamines, primarily adrenaline (epinephrine) and noradrenaline (norepinephrine). These hormones produce instant physiological changes, including elevated heart rate, elevated blood pressure, accelerated respiration, enhanced glucose mobilization, and enhanced alertness. Collectively, these responses prepare the organism for immediate action and survival.

Activation of the Hypothalamic–Pituitary–Adrenal (HPA) Axis [13]

In addition to the rapid SAM response, stress activates the slower but more sustained hypothalamic–pituitary–adrenal (HPA) axis. This process starts with the release of corticotropin-releasing hormone (CRH) from the hypothalamus. CRH stimulates the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH), which subsequently acts on the adrenal cortex to promote the release of cortisol, the principal glucocorticoid stress hormone.

Role of Cortisol [14]

Cortisol plays a critical function in maintaining physiological adaptation during stress. Its main functions include increasing blood glucose availability, amplifying energy mobilization, suppressing non-essential physiological activities such as digestion and reproduction, and regulating immune and inflammatory responses. Under normal conditions, cortisol exerts negative feedback on both the hypothalamus and pituitary gland, thereby limiting excessive activation of the HPA axis and restoring homeostasis.

Brain Changes During Chronic Stress [15]

Although acute stress responses are generally adaptive and protective, prolonged or chronic stress can adversely affect brain structure and function. Persistent activation of stress pathways may result in significant neurobiological alterations. The amygdala may become hyperactive, leading to exaggerated fear and anxiety responses. In contrast, chronic exposure to elevated cortisol levels may impair hippocampal function, reduce neurogenesis, and negatively affect memory processes. The prefrontal cortex is also vulnerable to chronic stress, with impairments observed in attention, executive functioning, affective regulation, and decision-making capacity.

Long-term dysregulation of stress systems has been associated with numerous adverse health outcomes, including depression, anxiety

disorders, sleep disturbances, cognitive impairment, and cardiovascular disease.

Neurotransmitters Involved in Stress Regulation [16]

Several neurotransmitters contribute to the modulation of stress responses. Dopamine is involved in reward processing and motivation; serotonin regulates mood and emotional stability; norepinephrine mediates arousal and watchfulness; and gamma-aminobutyric acid (GABA) exerts inhibitory effects that help reduce nerve cell excitability and anxiety. Dysregulation or disruption of these neurochemical systems may influence an individual's resilience to stress and heighten susceptibility to stress-related psychiatric and neurological disorders.

Stress theory: Stimulus-Based Theory (Stress as an External Event) [17]. The stimulus-based theory conceptualizes stress as an external event or environmental demand that places pressure on the individual. According to this perspective, stressful experiences arise from life events or situations that require significant adjustment or adaptation. Common examples include examinations, occupational pressures, financial difficulties, bereavement, and exposure to excessive noise. A major contribution to this theory was made by Thomas Holmes and Richard Rahe [18], who developed the *Social Readjustment Rating Scale (SRRS)* in 1967. The SRRS proposed that the accumulation of major life changes increases the chance of experiencing stress-related illness. The central assumption of this theory is that greater exposure to stressful events results in higher levels of stress. However, a major limitation of the stimulus-based approach is that it assumes individuals respond similarly to the same stressor, consequently neglecting individual differences in perception, coping, and toughness.

Response-Based Theory (Stress as a Reaction) [19,20]

The response-based theory, proposed by Hans Selye, defines stress as the body's physical response to demands or challenges. This perspective stresses the biological processes activated during exposure to stressors.

Selye introduced the concept of the *General Adaptation Syndrome (GAS)*, which describes the body's response to extended stress in three sequential stages:

- Alarm stage – the body initially recognizes the stressor and activates the fight-or-flight reaction.
- Resistance stage – the body tries to adjust and maintain functioning while coping with the stressor.
- Exhaustion stage – prolonged exposure to stress depletes physiological resources, heightening susceptibility to fatigue and illness.
- The key principle of this theory is that stress represents a universal biological response to surrounding stimuli.

Transactional (Cognitive) Theory [21]

The transactional or cognitive theory of stress, developed by Richard Lazarus and Susan Folkman, is among the most widely accepted models of stress. This theory emphasizes that stress is not determined solely by external situations, but by the individual's cognitive interpretation of them.

The model involves two stages of appraisal:

- Primary appraisal – the individual evaluates whether the situation is threatening, harmful, or challenging.
- Secondary appraisal – the individual assesses their ability and available resources to cope with the situation.

Stress occurs when perceived demands exceed perceived coping capacity. For example, one student may perceive an examination as overwhelming and stressful, whereas another may interpret the same examination as motivating or exciting.

This theory emphasizes the individual nature of stress and the role of cognitive processes in shaping stress responses.

Diathesis–Stress Model [22]

The diathesis–stress model explains stress-related disorders through the interaction between vulnerability and environmental stressors. In this system, a *diathesis* refers to a predisposition or susceptibility that may be genetic, biological, or psychological. According to the model, stress alone may not lead to illness unless an underlying vulnerability is present. People with greater predispositions are more likely to develop psychological or physical disorders when exposed to considerable stress. This model is widely used in understanding conditions such as depression, anxiety disorders, and schizophrenia, as it combines both biological vulnerability and environmental effects.

Physiological (Fight-or-Flight) Theory [23]

The physiological theory of stress is closely associated with Walter Cannon, who introduced the concept of the fight-or-flight response. According to this theory, exposure to stress activates the sympathetic nervous system, readying the body for immediate action. This response involves the secretion of stress hormones, such as adrenaline, leading to physiological changes including increased heart rate, elevated blood pressure, enhanced alertness, and speedy energy mobilization. These responses are adaptive in the short term, as they boost survival during threatening situations. The theory emphasizes stress as an acute physiological survival mechanism that protects the organism from danger.

Allostatic Load Model [24]

The allostatic load model represents a modern biological perspective on stress. This theory proposes that stress involves not only immediate physiological reactions but also the cumulative effects of chronic stress exposure. Repeated or prolonged activation of stress-response systems results in *allostatic load*, the "wear and tear" on the body caused by chronic adaptation to stress. Persistent stress may negatively affect multiple organ systems, including the brain, cardiovascular system, endocrine system, and immune function. The central concept of this model is that long-term stress produces cumulative physiological strain, thereby increasing the chance of chronic disease and impaired health outcomes.

Critical appraisal of stress theories: Theoretical perspectives on stress have progressively evolved from narrow physiological explanations toward multidimensional biopsychosocial frameworks. The stimulus-based theory conceptualizes stress as originating from external environmental events or demands. Its principal contribution lies in recognizing the association between stressful life events and adverse health outcomes, thereby facilitating the development of stress assessment instruments. However, the theory has been criticized for assuming uniform responses to stressors while failing to adequately account for individual perceptions, coping capacity, and contextual variation.

The response-based theory developed by Hans Selye defines stress as a non-specific physiological reaction to environmental demands. Selye's General Adaptation Syndrome substantially advanced the understanding of the biological consequences of prolonged stress exposure. Nevertheless, the theory has been criticized for its reductionist orientation, as it inadequately incorporates psychological,

behavioural, and social determinants of stress responses. The transactional theory proposed by Richard Lazarus and Susan Folkman conceptualizes stress as a dynamic interaction between the individual and the environment. According to this model, stress depends upon cognitive appraisal and the perceived availability of coping resources. This framework is particularly influential because it acknowledges individual variability in stress perception and coping processes. However, cognitive appraisal remains difficult to measure objectively and consistently.

The diathesis–stress model emphasizes the interaction between predisposing vulnerabilities and environmental stressors in the development of illness. The model is valuable in explaining differential susceptibility to psychological and physical disorders, although the precise nature and measurement of vulnerability factors remain conceptually challenging. The physiological fight-or-flight theory introduced by Walter Cannon explains acute autonomic responses to perceived threats and remains foundational in stress physiology. However, it fails to adequately explain chronic stress and the complexity of human behavioural responses.Finally, the allostatic load model, as described by Bruce McEwen, describes the cumulative physiological burden produced by chronic stress exposure. Although highly integrative and supported by contemporary evidence, consistent measurement of allostatic load remains methodologically challenging.

Overall, contemporary stress research increasingly supports integrative models that combine biological, psychological, and social dimensions to provide a more comprehensive understanding of stress and health outcomes.

Types of Stress [25]

Stress may be classified by duration, intensity, and physiological impact (). Comprehending the various forms of stress is essential, as stress is not invariably detrimental. In certain circumstances, stress facilitates adaptation, resilience, and improved performance, whereas in others it plays a major role in the development and progression of disease.

Acute Stress [26]

Acute stress refers to a short-term bodily and mental response to an immediate challenge or perceived threat. Common examples include academic examinations, public speaking, job interviews, athletic competitions, or narrowly avoiding an accident. During episodes of acute stress, the sympathetic nervous system is rapidly activated, resulting in increased heart rate, blood pressure, respiratory rate, and energy mobilization.

This response is generally adaptive and transient. Following the resolution of the stressful event, physiological functions typically return to baseline levels. Brief episodes of acute stress may boost concentration, alertness, and performance. Nevertheless, severe acute emotional stress can occasionally precipitate adverse cardiovascular events in vulnerable individuals, including myocardial infarction, cardiac arrhythmias, or stress-induced cardiomyopathy.

Chronic Stress [26]

Chronic stress develops when stressors persist over prolonged periods and the body is unable to restore physiological equilibrium. Factors such as occupational strain, financial hardship, interpersonal conflict, caregiving responsibilities, chronic illness, social adversity, and continuing emotional trauma may contribute to chronic stress. Unlike acute stress, chronic stress is characterised by sustained activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system. Prolonged exposure to cortisol and catecholamines produces widespread physiological alterations affecting cardiovascular, metabolic, immune, and neurological systems. Chronic stress is particularly harmful because it promotes inflammation, endothelial dysfunction, hypertension, insulin resistance, obesity, and maladaptive behavioural patterns that increase cardiovascular risk.

Stressors may be objectively present or subjectively perceived. Even in the absence of actual danger, situations perceived as threatening can trigger the stress response. Consequently, cognitive appraisal and affective evaluation play central roles in determining the physical and psychological effects of stress.

Positive and Negative Stress

Stress may also be categorized according to whether it promotes adaptation or produces harmful consequences.

Eustress (Positive Stress) [27]

Eustress refers to positive stress that enhances motivation, resilience, and adaptive functioning. Moderate challenges may stimulate learning, individual development, and the development of effective coping skills. Examples include preparing for an important event, commencing a new occupation, or participating in competitive sports.
Positive stress may improve self-confidence, performance, and emotional strength. Psychological characteristics such as optimism, self-esteem, impulse control, and effective decision-making contribute to more effective reactions to stressors. Furthermore, challenging experiences may ultimately promote personal development and emotional maturity.

Distress (Negative Stress) [28]

Distress refers to harmful stress that exceeds an individual's coping capacity and disrupts both bodily and psychological functioning. Severe or prolonged stress is associated with anxiety, depression, fatigue, sleep disturbances, impaired concentration, and physical illness. Distress may adversely affect social relationships, occupational performance, and overall quality of life. Physiologically, persistent distress results in prolonged activation of stress pathways, thereby heightening susceptibility to chronic disease.

Tolerable Stress [29]

Tolerable stress occurs when individuals experience adversity while maintaining adequate coping mechanisms and social support systems. Although such experiences may be emotionally challenging, supportive relationships and successful coping strategies promote recovery and accommodation. In some cases, these experiences may strengthen resilience as well as improve the ability to manage future stressors.

Toxic Stress [29]

Toxic stress arises when stress is severe, repetitive, or prolonged and occurs in the absence of adequate emotional support or coping resources. This maladaptive stress response leads to persistent physiological dysregulation, behavioural disturbances, and an increased long-term risk of disease. Toxic stress has been associated with chronic inflammation, impaired affective regulation, unhealthy behavioural patterns, and greater susceptibility to cardiovascular disease, mental disorders, and metabolic abnormalities.

Effects of Chronic Stress on Health

Chronic stress exerts widespread effects on nearly every organ system. Sustained activation of stress pathways produces both compensatory and maladaptive bodily changes that play a major role to disease development [30].

Immune Dysfunction

Stress and immune function are closely interconnected [31]. Acute stress may transiently boost immune activity, preparing the body in responding to injury or infection. In contrast, chronic stress suppresses and dysregulates both innate and adaptive immune responses. Persistent activation of the stress response promotes low-grade systemic inflammation while impairing the function of protective immune cells [31]. Elevated levels of inflammatory cytokines contribute to tissue injury, endothelial dysfunction, insulin resistance, and the progression of atherosclerosis.

Persistent Sickness Behaviour [32]

Chronic stress–related inflammation may induce persistent "sickness behaviour," characterized by fatigue, social withdrawal, depressed mood, reduced appetite, sleep disturbances, and heightened pain sensitivity. Although this response is adaptive during acute illness, its prolonged persistence contributes to depression and diminished quality of life.

Hormonal Alterations [33]

Chronic stress results in sustained elevations of cortisol, adrenaline, and noradrenaline levels. Over time, glucocorticoid receptor resistance may develop, impairing normal negative feedback regulation of the HPA axis and perpetuating inflammatory activity. Stress-induced hormonal alterations also affect neural signalling systems. Increased metabolism of tryptophan may reduce serotonin availability, thereby contributing to depression, anxiety, and emotional dysregulation.

Effects on Glucose and Lipid Metabolism [34]

Excess cortisol stimulates gluconeogenesis and promotes insulin resistance. Consequently, chronic stress is associated with hyperglycaemia, type 2 diabetes mellitus, hyperlipidaemia, visceral obesity, and reduced lean muscle mass. These metabolic disturbances substantially increase cardiovascular risk and aid in the development of metabolic syndrome.

Atherosclerosis [35]

Chronic stress contributes to all stages of atherosclerosis, ranging from endothelial injury to plaque instability and rupture. The basic mechanisms include chronic inflammation, endothelial dysfunction, autonomic imbalance, abnormal platelet activation, hyperlipidaemia, and hypertension. Inflammatory cytokines and oxidative stress damage the vascular endothelium, impair nitric oxide production, and promote atherosclerotic plaque formation. In addition, stress-related hemodynamic changes further accelerate vascular injury and disease progression.

Stress and Cardiovascular Disease

The relationship between stress and cardiovascular disease is complex and multifactorial, involving both direct physiological mechanisms and indirect behavioural pathways that contribute to cardiovascular morbidity and mortality [36].

Behavioural Mechanisms

Individuals exposed to chronic stress are more likely to adopt unhealthy lifestyle behaviours that increase cardiovascular risk [37]. These behaviours include smoking, physical inactivity, unhealthy eating habits, excessive alcohol consumption, poor sleep quality, medication non-adherence, and obesity. Stress-related emotional eating and diminished motivation to engage in physical activity add to weight gain and metabolic disturbances. In addition, sleep disturbances associated with chronic stress further intensify sympathetic nervous system activity, systemic inflammation, and hypertension.

Endothelial Dysfunction [39]

Psychological stress adversely affects endothelial function via several mechanisms, including sympathetic nervous system activation, the release of inflammatory cytokines, oxidative stress, and reduced nitric oxide bioavailability [39]. Endothelial dysfunction impairs vascular relaxation and promotes the development of hypertension and atherosclerosis [40,41].

Hypertension

Persistent activation of the sympathetic nervous system increases vascular tone, heart rate, and arterial smooth muscle proliferation, therefore contributing to sustained hypertension and vascular hypertrophy [40]. Chronic elevations in cortisol and catecholamines, associated with stress, also alter sodium regulation and vascular reactivity, additionally raising blood pressure.

Inflammation and Atherosclerosis

Chronic stress is associated with persistent low-grade sterile inflammation, which promotes the formation, progression, and destabilization of atherosclerotic plaques [41]. Inflammatory mediators weaken plaque integrity, raising the probability of plaque rupture and thrombus formation. Furthermore, stress-induced platelet activation and hypercoagulability enhance the risk of acute cardiovascular events.

Arrhythmias

Emotional stress may precipitate cardiac arrhythmias through autonomic imbalance and disturbances in cardiac repolarisation [42,43]. Increased sympathetic activity accompanied by reduced parasympathetic tone can trigger atrial fibrillation, ventricular arrhythmias, and sudden cardiac death in susceptible individuals [43].

Acute Emotional Stress

Severe acute emotional stress can induce excessive catecholamine release, leading to calcium overload, coronary vasoconstriction, myocardial injury, and increased ventricular afterload [44]. These physiological alterations may precipitate myocardial infarction, arrhythmias, or acute heart failure syndromes [44].

Takotsubo Cardiomyopathy

Takotsubo cardiomyopathy [45], also referred to as stress cardiomyopathy or "broken heart syndrome," is a transient cardiac disorder precipitated by acute emotional or physical stress. The condition is characterized by reversible left ventricular dysfunction and apical ballooning that clinically mimics acute myocardial infarction [45]. Excessive catecholamine release is believed to play a central role through mechanisms involving myocardial stunning, coronary microvascular dysfunction, and direct myocardial toxicity [45]. Women, particularly postmenopausal women, appear to be more susceptible than men to stress-induced myocardial ischemia and Takotsubo cardiomyopathy. Differences in neural stress responses and hormonal regulation may contribute to this enhanced vulnerability.

Assessment of Stress in Primary Care

Stress-related symptoms constitute a significant proportion of presentations in primary healthcare settings. Chronic stress exerts widespread effects on multiple physiological systems and contributes to the development of both physical and psychological disorders. Despite its substantial clinical impact, structured assessment and counselling for stress remain insufficiently integrated into routine clinical practice [46]. Causes contributing to the under recognition of stress-related disorders may include limited training in behavioural medicine, restricted consultation time, and inadequate emphasis on psychosocial assessment within medical systems [46].

In response to growing evidence regarding the health consequences

of chronic stress [47], lifestyle medicine organizations progressively advocate for the routine assessment of stress as a clinical "vital sign." The use of brief validated instruments, such as the Perceived Stress Scale (PSS) [48], may facilitate the identification of individuals at elevated risk of stress-related morbidity. Comprehensive clinical evaluation should include assessment of:

- Sources of stress
- Coping mechanisms
- Availability of social support
- Behavioural consequences of stress
- Sleep quality
- Emotional wellbeing
- Cardiovascular risk factors.

The Perceived Stress Scale (PSS) [48]

The Perceived Stress Scale (PSS) is a validated psychological instrument extensively used to assess the degree to which individuals perceive life situations as stressful. Originally developed in 1983 by Sheldon Cohen and colleagues [49], the scale measures the extent to which individuals appraise circumstances in their lives as unpredictable, uncontrollable, and overwhelming. In contrast to physiological indicators of stress, the PSS focuses on cognitive and emotional appraisal, recognising that stress responses are influenced by individual coping capacity, resilience, and psychological interpretation of events. Due to its strong psychometric properties and ease of administration, the PSS has become widely implemented in clinical practice, behavioural medicine, epidemiological research, and public health studies.

The instrument is available in 14-item, 10-item, and 4-item versions, with the 10-item (appendix 1) format being the most utilised in healthcare and research settings. Respondents evaluate the frequency of stress-related thoughts and feelings experienced during the preceding month using a Likert-type response scale ranging from "never" to "very often." Higher cumulative scores indicate greater levels of perceived stress. Empirical evidence demonstrates that the PSS exhibits high internal consistency, satisfactory reliability, and strong construct validity across diverse demographic and cultural populations.

Appendix 1: *Perceived Stress Scale (PSS-10).*

For each question choose from the following alternatives:
0 - never 1 - almost never 2 - sometimes 3 - fairly often 4 - very often

_____ 1. In the last month, how often have you been upset because of something that happened unexpectedly?

_____ 2. In the last month, how often have you felt that you were unable to control the important things in your life?

_____ 3. In the last month, how often have you felt nervous and stressed?

_____ 4. In the last month, how often have you felt confident about your ability to handle your personal problems?

_____ 5. In the last month, how often have you felt that things were going your way?

_____ 6. In the last month, how often have you found that you could not cope with all the things that you had to do?

_____ 7. In the last month, how often have you been able to control irritations in your life?

_____ 8. In the last month, how often have you felt that you were on top of things?

_____ 9. In the last month, how often have you been angered because of things that happened that were outside of your control?

_____ 10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

Interpretation:

Scores ranging from 0 -13 considered low stress
Scores ranging from 14 -26 considered moderate stress
Scores ranging from 27- 40 considered high stress

An extensive evidence base has identified significant associations between elevated PSS scores and adverse psychological and physiological outcomes [50]. High levels of perceived stress have been correlated with anxiety, depressive symptoms, sleep disturbances, impaired immune function, hypertension, and increased cardiovascular risk. Furthermore, elevated perceived stress is associated with maladaptive health behaviours, including tobacco use, sedentary lifestyle, unhealthy dietary practices, alcohol misuse, and poor adherence to prescribed treatment regimens [51,52]. Consequently, the PSS is frequently employed as a screening and evaluative tool in healthcare settings to identify individuals at risk of stress-related disorders and to assess the effectiveness of interventions such as mindfulness-based stress reduction, cognitive behavioural therapy, physical activity programmes, and relaxation-based approaches.

Identifying Modifiable Contributors to Stress

Effective stress management requires identifying and modifying factors that contribute to stress exposure and maladaptive stress responses [53,54].

External Stressors

Patients should be encouraged to recognize external stressors and evaluate whether aspects of these stressors might be modified, minimized, or cognitively reframed. Emphasizing controllable elements of stressful situations may enhance coping capacity and lessen feelings of helplessness.

Bodily Reactions

Stress-related manifestations, including palpitations, muscle tension, headaches, and hyperventilation, commonly result from activation of the autonomic nervous system. Relaxation strategies and regulated breathing exercises may promote parasympathetic nervous system activity, therefore reducing physiological arousal and improving symptom control.

Mental Reactions

Cognitive appraisal plays a central role in determining stress reactivity. Maladaptive cognitive patterns, including catastrophic thinking, rumination, and negative self-evaluation, may prolong and intensify stress responses. Interventions such as cognitive-behavioural therapy (CBT), mindfulness-based practices, and meditation can help individuals recognize and modify dysfunctional thought patterns while strengthening fortitude and self-efficacy.

- Strategies designed to improve psychic resilience include:
- reinforcing previous successful coping experiences
- strengthening social support systems
- fostering optimism and a sense of meaning
- encouraging adaptive decision-making skills.

Stress Management

Stress is amenable to intervention, and applying structured management strategies can relieve symptoms, enhance quality of life, and potentially reduce cardiovascular risk.

Breathing Exercises: Diaphragmatic breathing techniques [55] activate the parasympathetic nervous system while inhibiting sympathetic overactivity. This bodily shift contributes to reductions in heart rate, blood pressure, and cortisol levels. Short periods of controlled breathing have been shown to decrease perceived stress and physiological arousal [56].

Mindfulness: Mindfulness entails cultivating non-judgmental awareness of present-moment experiences. Diminishing rumination about past events and anticipatory anxiety about the future enhance affec-

tive regulation and mental health. Regular mindfulness practice has been associated with reductions in stress, anxiety, and depressive symptomatology [57].

Meditation: Meditative practices modulate stress-related neurobiological pathways [58], including attenuation of sympathetic nervous system activity and hypothalamic–pituitary–adrenal (HPA) axis responsiveness, while improving parasympathetic tone. Evidence indicates that meditation supports improved stress regulation, mental well-being, and cardiovascular outcomes, including lowered blood pressure.

Yoga, Tai Chi, and Qi Gong: These integrative mind–body interventions combine physical movement, breath regulation, and focused attention [59,60]. They have been associated with improvements in flexibility, balance, sleep quality, mental health, and stress resilience. When used alongside other therapeutic approaches, they may substantially reduce symptoms of anxiety and depression.

Relaxation Techniques: Techniques such as progressive muscle relaxation aim to reduce somatic tension and physiological arousal [61]. Reported benefits include reductions in fatigue, headache frequency, insomnia, and anxiety symptoms [61].

Guided Imagery: Guided imagery, also known as visualization or cognitive rehearsal, involves intentionally using sensory imagery to evoke calming mental experiences [62]. Activating multiple sensory modalities can facilitate relaxation responses and reduce stress-related arousal.

Self-Help Cognitive Behavioural Therapy (CBT): Self-directed CBT strategies [63] help people identify and restructure maladaptive cognitions that contribute to stress. These interventions support improved coping mechanisms, emotional strength, and reduced psychological distress.

Protecting Cardiovascular Health During Stress

Stress management should be considered an integral element of overall physical health. Individuals experiencing chronic stress are encouraged to discuss their symptoms with medical practitioners, particularly when coexisting cardiovascular risk factors such as obesity, hypertension, diabetes, or smoking are present [64]. Regular physical exercise is among the most efficient interventions, as it concurrently reduces psychological stress and improves cardiovascular function. Exercise decreases sympathetic activation, enhances endothelial function, improves affect regulation, and promotes sleep quality [65].

Additional protective strategies include maintaining balanced nutrition, maintaining sufficient sleep, strengthening social support networks, avoiding tobacco use, moderating alcohol intake, following prescribed pharmacotherapy, and seeking psychological support when appropriate [65]. Importantly, stress may also serve as a catalyst for positive behavioural change, prompting individuals to adopt healthier lifestyles, including greater physical activity, improved dietary habits, and enhanced self-care.

Conclusion

Stress has significant, multiple effects on heart and vascular health, mediated by interconnected neuroendocrine, inflammatory, metabolic, autonomic, and behavioural mechanisms. While acute stress responses are adaptive and indispensable for survival, chronic stress leads to sustained physiological dysregulation, which contributes to endothelial dysfunction, hypertension, systemic inflammation, atherosclerotic progression, cardiac arrhythmias, metabolic imbalance, and maladaptive health behaviours.

The association between stress and cardiovascular disease is bidirectional and mutually reinforcing. Prolonged psychological stress increases susceptibility to cardiovascular pathology, whereas the presence of cardiovascular disease can, in turn, exacerbate psychological distress and heighten stress responsiveness.

The recognition of stress as a modifiable risk factor for cardiovascular disease stresses the importance of incorporating routine stress assessment into primary care and lifestyle medicine practices. Evidence-based interventions, including cognitive-behavioural therapy, mindfulness-based approaches, meditation, controlled breathing techniques, relaxation training, guided imagery, yoga, and regular physical exercise, have demonstrated benefits for psychological well-being and cardiovascular outcomes.

In the context of increasing psychosocial demands in contemporary societies, integrating structured stress management strategies into cardiovascular prevention and clinical care is essential for complete, integrated patient management.

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