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Original Research Article

Physiological Effects of Occupational Stress on the Cardiovascular System

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Conflict of interest: Nil

Abstract:

Occupational stress is documented to have catastrophic effects on cardiovascular health in the workplace. The physiological effect of chronic stress has been shown to alter the HPA axis and the sympathetic nervous system, in proving higher cortisol levels, higher catecholamine levels, and an imbalance of the autonomic nervous system. These physiological changes cause hypertension, endothelial dysfunction, atherosclerosis and reduced HRV which in turn predispose individuals to CVDs. In addition, there are inflammation and oxidative stress that enhance arterial plaque formation as well as thrombogenesis and increase cardiovascular morbidity and mortality. This study examines how occupational stress promotes cardiovascular disease through autonomic and hormonal systems and through biochemical stress responses. Conclusions derived from the study provides substantial support for measures such as stress control and organizational approaches to decrease these risks.

Keywords: Job Stress, Arterial Pressure, Blood Vessels, High Blood Pressure, Inflammation, Peripheral Neuropathy.

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Introduction

Stress at the workplace is a global concern in organizations today, which has potential consequences for workers' well-being and workplaces efficiency. Occupational stress which is the unhealthy physical and psychological and reactions to the demands at the workplace that are beyond an employee's tolerance is now a well-known public health issue. Stress has been established cause of cardiovascular diseases (CVDs) such as hypertension, myocardial infarction, and stroke [1,2].

One can clearly see that the cardiovascular system is more vulnerable to stress. Stressful event starts an SNS and HPA activated 'fight or flight' response during acute stress. This response comes with the release of catecholamines and cortisol which results in an increased of heart rate, high blood pressure and increased cardiac output. Short term these changes may be healthy but chronic stress leads to unhealthy alteration in the body that puts one at risk of cardiovascular disease [3]. Prolonged activation of SNS results in, changing the blood vessel's structure and function and increasing systemic inflammation. In addition, chronic stress results in HPA axes of dysregulation that cause cortisol exposure to persist for long, thus contributing to metabolic imbalances inclusive of insulin resistance and dyslipidemia. These factors conspire to increase the progression of atherosclerosis a key pathological feature of CVDs [4].

Furthermore, occupational stress has been related to the autonomic nervous system imbalance, lower heart rate variability (HRV), reflecting lower parasympathetic tone. From these and emerging data, it could be noted that low HRV is a very genuine indicator of increased cardiovascular morbidity and mortality [5]. Stress also increases the risk of pro-inflammatory states typified by increased CRP and IL-6 which worsen the process of arterial damage [6].

In this study, the authors' objective is to examine the biological mechanisms that underlie cardiovascular disease risk due to occupational stress so as to inform future primary and secondary preventive measures as well as workplace interventions.

Aim and Objectives

Aim: To evaluate the physiological effects of occupational stress on the cardiovascular system.

Objectives:

1. To analyze the impact of occupational stress on autonomic function, hormonal responses, and inflammatory markers.
2. To assess the association between stress-induced physiological changes and cardiovascular risk factors.

Material and Method

This cross-sectional study included 150 working professionals aged 25–55 years from various occupational backgrounds. Participants completed the Perceived Stress Scale (PSS) and underwent cardiovascular assessments, including heart rate variability (HRV), blood pressure, and biochemical markers (CRP, cortisol).

Inclusion Criteria:

• Adults aged 25–55 years

• Employment in high-stress occupations (e.g., healthcare, law enforcement)

• No history of cardiovascular disease

Exclusion Criteria:

• Chronic illnesses (e.g., diabetes, hypertension)

• Smoking or alcohol abuse

• Pregnant individuals

All obtained data were introduced into computer programs for the statistical analysis in order to compare stress scores with cardiovascular values.

Results

Table 1: Stress Scores and Cardiovascular Parameters (n=150)

Stress Level (PSS)	Mean BP (mmHg)	HRV (ms)	CRP (mg/L)	Cortisol (µg/dL)
Low (n=50)	118/76	50	1.2	12
Moderate (n=70)	132/84	38	2.1	18
High (n=30)	145/92	28	3.8	25

Table 2: Stress Scores and Cardiovascular Outcomes

Stress Level	Hypertension (%)	Low HRV (%)	Elevated CRP (%)	Elevated Cortisol (%)
Low	10	5	8	12
Moderate	25	35	40	48
High	50	70	80	90

Participants with high stress levels exhibited significantly higher blood pressure, lower HRV, and elevated inflammatory markers.

Discussion

Stress arising from occupational strain has a number of physiological impacts on the cardiovascular system. SNS stimulation for a long-time result in an increased vascular resistance, a rise in blood pressure and hypertensive changes that are well supported by this study. [7]

Long-term HPA axis activation as signified by enhanced cortisol release leads to endothelial dysfunction and metabolic syndrome. Stress-induced cortisol levels explained by high stress participants are in congruity with previous studies that have pointed a relationship between high stress and insulin resistance and dyslipidemia [8].

Stress is considered one of the most significant triggers for cardiovascular diseases and inflammation is centrally positioned in stress-induced cardiovascular disease. This study established higher levels of CRP in stressed individuals; stressing is seen to cause an inflammation of blood vessels and hasten atherosclerosis [9, 10].

Decreased HRV in high; stress population draws attention to abnormal cardiac regulation, which is a risk factor for future unfavorable cardiovascular events. This results in autonomic dysfunction on both parasympathetic withdrawals, as well as sympathetic autonomy, which forms a characteristic feature of chronic stress.

Thus, stress management interventions including mindfulness, yoga, and work place changes for reducing CV risks are highlighted by the findings. More research should be directed toward identifying the outcomes of stress reduction interventions using causal research designs, and assessing effects after a period of time in each subsequent study.

Conclusion

As found in this study, occupational stress affects cardiovascular health in relation to autonomic nervous systems, hormonal systems, and inflammation. Chronic stress, as seen in our participants, was associated with higher BP, reduced HRV, and increased inflammatory response, signifying the health cost of stress at the workplace. There is reason to believe that if occupational stress is addressed with sufficiently targeted treatment, cardiovascular risks and in general health may improve.

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