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Original Research Article

Association of Cardiovascular Autonomic Function with Psychological Distress in Young Adults

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Abstract:

Background: Psychological distress has been increasingly recognized as a contributing factor to cardiovascular dysfunction. Alterations in cardiac autonomic regulation may represent early physiological changes linking psychological stress to future cardiovascular risk.

Objective: To evaluate the association between psychological distress and cardiovascular autonomic function in young adults using resting cardiovascular parameters and short-term heart rate variability (HRV) analysis.

Methods: This cross-sectional study included 100 young adults aged 18–25 years, comprising 50 healthy controls and 50 psychologically distressed participants categorized using a validated self-reported stress scale. Resting heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded under standardized laboratory conditions. Short-term HRV was assessed from 360-second R–R interval recordings using Kubios software. Time-domain (Mean RR, SDNN, RMSSD, NN50, pNN50) and frequency-domain (Total Power, LF, HF, LF/HF ratio) parameters were analyzed. Pearson correlation was used to assess associations between stress scores and HRV indices.

Results: Psychologically distressed participants demonstrated significantly higher resting HR, SBP, and DBP compared to controls ($p < 0.01$). Time-domain HRV parameters were significantly reduced in the distressed group, including Mean RR, SDNN, RMSSD, NN50, and pNN50 ($p < 0.001$). Frequency-domain analysis revealed significantly lower Total Power and HF power, with significantly higher LF power and LF/HF ratio ($p < 0.001$), indicating sympathetic predominance and reduced parasympathetic modulation. Stress scores showed strong negative correlations with parasympathetic indices (r ranging from –0.55 to –0.65) and positive correlations with LF power and LF/HF ratio ($r = 0.48$ and 0.66, respectively; $p < 0.001$).

Conclusion: Psychological distress in young adults is significantly associated with cardiovascular autonomic imbalance characterized by reduced vagal activity and increased sympathetic dominance. These findings suggest that psychological distress may contribute to early autonomic dysregulation and potential long-term cardiovascular risk.

Keywords: Psychological Distress, Heart Rate Variability, Autonomic Nervous System.

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Introduction

Cardiovascular disease (CVD) remains prevalent and risk factors for its progression to adverse outcomes are incompletely understood. [1,2] anxiety disorders are an important mood disorder commonly associated with a higher risk of fatal cardiovascular diseases (CVD), including coronary heart disease, sudden cardiac death and cerebrovascular diseases, with a global prevalence of 0.9% to 28.3% [3]. The combination of a stressful trigger and pathological cardiac substrate underlies such mortality events, and evaluating the components of this paradigm may help explain this excess risk. The autonomic nervous system (ANS) serves as a key mediator, with possible downstream consequences including mental stress–induced myocardial ischaemia and electrical instability that may precede CVD mortality events. [4] The underappreciation of the role of psychosocial factors in the development and progression of cardiac diseases may partly explain why these diseases remain the leading cause of death

in most developed countries. Although several studies investigating the relationship between CVD and negative emotions have focused on depression. [5]

We can study ANS changes in response to stress with heart rate variability (HRV), an ambulatory electrocardiographic (ECG) digital biomarker that measures beat-to-beat changes in heart rate over time [6]. Frequency-based HRV measures reflect the sympathetic and parasympathetic response to respiration, baroreflex activity, and hormonal activity, allowing these measures to serve as surrogate markers of ANS function. High-frequency (HF) HRV is a known measure of parasympathetic function and low-frequency (LF) HRV has been associated with baroreflex sensitivity. The LF band in particular may reflect maladaptive stress responses underlying increased CVD risk, with lower levels of HRV associating with ventricular tachyarrhythmias, abnormal myocardial perfusion, and sudden cardiac death. While decreased long-term HRV is known to predict all-cause mortality, the role of acute psychological stress in leading to cardiovascular events, as demonstrated by the earthquake study and others, [7] suggests the acute autonomic response to stress may also play a role. We know little of how stress, and the neurobiological flexi bility to respond to psychological stress, contributes additive risk for CVD mortality. [8] Understanding autonomic reactivity may inform CVD risk reduction interventions aimed at mechanisms involving cardiovascular adaptation to acute psychological stress. In this study, we have determined the Association between Cardiovascular Autonomic Function with Psychological Distress in Young Adults.

Materials and Methods

Study Design and Participants: The study was undertaken following approval from the Institutional Ethics Committee for human research. It was conducted in the Autonomic Function Testing (AFT) Laboratory of the Department of Physiology, Government Medical College, Ongole. This cross-sectional observational study enrolled apparently healthy young adults aged 18–25 years.

Exclusion criteria: Patients with comorbidities like diabetes, hypertension or cardiac diseases, renal disease, other psychiatric illnesses, autonomic dysfunction and pulmonary disorder were excluded from this study.

Methodology

After obtaining written and informed consent, participants were advised to attend the Physiology Department's autonomic function test (AFT) laboratory between 9:30 a.m. and 10:00 a.m., after a light breakfast, for autonomic function testing. They were instructed to refrain from vigorous physical activity for 12 hours; to avoid tea, caffeine products, tobacco, and alcohol for 24 hours; and to withhold anticholinergics, antihistaminics, sympathomimetic and parasympathomimetic agents, if any, for 12 hours before the recording. Height in centimeters was measured using a wall-mounted stadiometer and body weight was recorded using an automated weighing machine (BMI was calculated using Quetelet's index. Heart rate (HR), Systolic Blood Pressure (SBP), and Diastolic Blood Pressure (DBP) were recorded after 10 min of rest in the supine position on a couch in the lab (room temperature maintained at 250C) using an automated sphygmomanometer. Psychological distress was evaluated using a validated self-reported scale, namely the Perceived Stress Scale (PSS). Participants were classified according to established cutoff scores.

Short-term Heart Rate Variability (HRV): Participants were instructed to empty their bladder before the recording. They then rested in a supine position in the laboratory for 15 minutes. ECG electrodes were attached and connected to record Lead II ECG for 5–10 minutes in a quiet room maintained at 25°C for HRV assessment. The R–R intervals from the entire recording were extracted and stored in text format. HRV analysis was performed using Kubios HRV software (version 2.0; Bio-Signal Analysis Group, Kuopio, Finland). The software, originally based on MATLAB, was compiled into a standalone C program. Time-domain parameters included mean RR, RMSSD, SDNN, NN50, and pNN50. Frequency-domain parameters included Total Power (TP, ms^2), Low-Frequency power (LF, ms^2 and normalized units), High-Frequency power (HF, ms^2 and normalized units), and the LF/HF ratio.

Statistical Analysis: Data were analyzed using statistical software. Continuous variables were expressed as mean ± standard deviation. Group comparisons and correlation analyses were performed, with $p < 0.05$ considered statistically significant.

Results

A total of **100 participants** (50 controls and 50 psychologically distressed young adults) completed cardiovascular autonomic function testing and short-term HRV analysis.

Table 1: Comparison of Cardiovascular Autonomic Parameters Between Groups

Parameter	Controls (n=50) Mean ± SD	Psychological Distress(n=50) Mean ± SD	p-value
Resting HR (bpm)	72 ± 6	80 ± 7	<0.001
SBP (mmHg)	114 ± 8	122 ± 9	<0.01
DBP (mmHg)	74 ± 6	82 ± 7	<0.01
Mean RR (ms)	820 ± 75	740 ± 80	<0.001
SDNN (ms)	55 ± 12	38 ± 10	<0.001
RMSSD (ms)	45 ± 10	28 ± 8	<0.001
NN50	38 ± 9	18 ± 6	<0.001
pNN50 (%)	22 ± 6	10 ± 4	<0.001
Total Power (ms²)	3200 ± 450	2100 ± 400	<0.001
LF (ms²)	1200 ± 200	1500 ± 250	<0.001
HF (ms²)	1500 ± 250	800 ± 200	<0.001
LF/HF Ratio	1.6 ± 0.4	2.5 ± 0.6	<0.001

Table no 1 show that psychologically distressed participants demonstrated significantly higher resting heart rate and blood pressure compared to controls. Resting HR was significantly elevated in the distress group ($p < 0.001$). Both SBP and DBP were significantly higher in distressed participants ($p < 0.01$). These findings indicate increased basal sympathetic activity in psychologically distressed young adults.

R–R interval data revealed significant reductions in vagally mediated time-domain parameters in the psychologically distressed group. Mean RR interval was significantly lower ($p < 0.001$). SDNN, RMSSD, NN50, and pNN50 were significantly reduced ($p < 0.001$). Reduced time-domain indices indicate diminished overall HRV and impaired parasympathetic modulation in distressed individuals. Frequency domain analysis using Fast Fourier Transformation demonstrated significant alterations in autonomic balance. Total Power was significantly reduced in distressed participants ($p < 0.001$), HF power was significantly lower ($p < 0.001$), LF power was significantly higher ($p < 0.001$) LF/HF ratio was significantly elevated ($p < 0.001$). These findings indicate sympathetic predominance and reduced parasympathetic tone in psychologically distressed young adults.

Table no 2 Correlation between Psychological Stress Scores and HRV Parameters

HRV Parameter	Correlation with Stress Score (r)	p-value
Mean RR (ms)	-0.62	<0.001
SDNN (ms)	-0.58	<0.001
RMSSD (ms)	-0.65	<0.001
NN50	-0.60	<0.001
pNN50 (%)	-0.63	<0.001
Total Power (ms²)	-0.55	<0.001
LF (ms²)	+0.48	<0.01
HF (ms²)	-0.59	<0.001
LF/HF Ratio	+0.66	<0.001

Table no 2 shows that Pearson correlation analysis revealed a significant negative correlation between psychological stress scores and parasympathetic HRV indices, including Mean RR, SDNN, RMSSD, NN50, pNN50, HF power, and Total Power ($p < 0.001$). Conversely, LF power and LF/HF ratio demonstrated significant positive correlations with stress scores, indicating increasing sympathetic dominance with higher levels of psychological distress. These findings further support the association between psychological distress and autonomic imbalance in young adults.

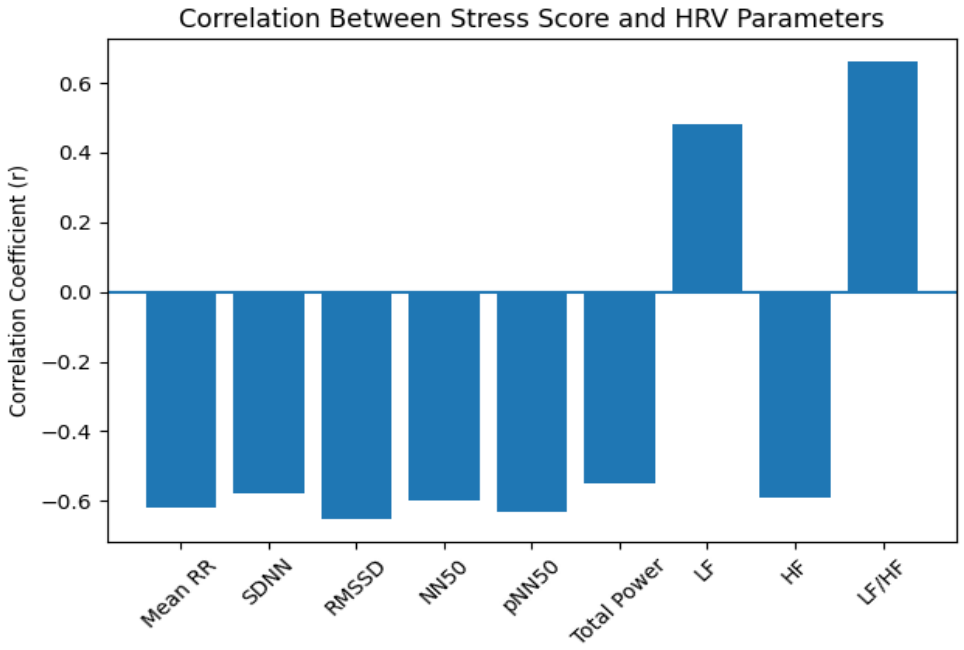

Figure 1: Correlation Between Stress Score and HRV Parameters

Figure 1. Shows that Negative correlations were observed between stress scores and Mean RR, SDNN, RMSSD, NN50, pNN50, Total Power, and HF power, indicating reduced parasympathetic modulation with increasing stress levels. Positive correlations were noted for LF power and LF/HF ratio, reflecting increased sympathetic dominance in psychologically distressed young adults.

Discussion

The present study investigated the association between psychological distress and cardiovascular autonomic function in young adults using standardized autonomic function testing and short-term heart rate variability (HRV) analysis. The findings demonstrate significant autonomic imbalance in psychologically distressed individuals, characterized by elevated resting cardiovascular parameters, reduced overall HRV, diminished parasympathetic modulation, and increased sympathetic dominance. Furthermore, stress scores showed strong correlations with HRV indices, reinforcing the link between psychological distress and autonomic dysregulation.

Psychologically distressed participants exhibited significantly higher resting heart rate and blood pressure compared to controls. Elevated resting cardiovascular parameters are well-recognized indicators of increased sympathetic tone and reduced vagal restraint. Similar observations have been reported in young populations experiencing psychosocial stress, suggesting early cardiovascular activation in distressed individuals [9].

Time-domain HRV analysis revealed significant reductions in Mean RR interval, SDNN, RMSSD, NN50, and pNN50 in the psychologically distressed group. These parameters primarily reflect vagal modulation of heart rate. Reduced vagal tone is associated with impaired autonomic adaptability and decreased cardiovascular resilience to stress. Previous studies in young adults with depression and psychological distress have demonstrated similar reductions in parasympathetic indices [10].

Frequency-domain analysis further confirmed autonomic imbalance. Total Power and HF power were significantly reduced in distressed individuals, whereas LF power and LF/HF ratio were significantly elevated. Reduced HF power reflects diminished parasympathetic activity, while increased LF/HF ratio indicates sympathetic predominance. These findings are consistent with evidence that chronic psychological distress shifts autonomic balance toward sympathetic dominance [11].

Correlation analysis strengthened these observations. Stress scores showed strong negative correlations with parasympathetic-related HRV indices (Mean RR, SDNN, RMSSD, NN50, pNN50, HF power, and Total Power) and positive correlations with LF power and LF/HF ratio. This dose–response relationship suggests that increasing levels of psychological distress are progressively associated with autonomic dysregulation. The magnitude of correlation observed in this study aligns with previous reports linking psychological distress to cardiac autonomic imbalance [12].

Autonomic dysregulation associated with psychological distress may contribute to long-term cardiovascular vulnerability. Sustained sympathetic activation and reduced vagal modulation have been

implicated in endothelial dysfunction, arrhythmogenesis, and hypertension. Large epidemiological studies have demonstrated that psychological distress is independently associated with increased cardiovascular morbidity and mortality [13].

The present findings emphasize that autonomic alterations occur even in young and apparently healthy individuals experiencing psychological distress. Early identification of such physiological changes may allow preventive interventions before the development of overt cardiovascular disease.

Conclusion

The present study demonstrates a significant association between psychological distress and cardiovascular autonomic dysfunction in young adults. Psychologically distressed individuals exhibited elevated resting cardiovascular parameters, reduced overall heart rate variability, diminished parasympathetic modulation, and increased sympathetic dominance. Stress scores showed strong correlations with HRV indices, indicating a progressive relationship between psychological distress and autonomic imbalance.

These findings suggest that psychological distress in young adulthood is accompanied by measurable physiological alterations that may predispose individuals to future cardiovascular risk. Routine assessment of psychological stress along with non-invasive autonomic evaluation may be valuable for early cardiovascular risk stratification and preventive strategies.

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