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ORIGINAL ARTICLE

# Effect of Short-Term Mental Stress on Cardiovascular Reactivity and Heart Rate Variability in Children of Hypertensive Parents

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## ABSTRACT

***Background:*** Hypertension accounts for 13.5 million deaths worldwide every year and is one of the most prevalent risk factors for cardiovascular disease. Numerous studies suggest a significant link between cardiovascular reactivity to stress and the development of future hypertension. Most of the previous research has focused on hypertensive patients directly, aiming to predict their stress reactivity rather than exploring their offspring. So, in the present study, we attempted to find out whether there is an effect of acute mental stress on cardiovascular reactivity and changes in heart rate variability (HRV) in children of hypertensive parents.

***Material and Methods:*** This case-control study includes sixty participants from the general population, adhering to specific inclusion and exclusion criteria. Among these, 30 participants were young adults aged 18 to 22 years, with at least one parent diagnosed with essential hypertension according to JNC VII guidelines. Additionally, thirty control participants were selected, consisting of healthy young adults matched for age and sex, who are children of parents with no family history of hypertension. Acute mental stress was induced using problem-solving questions from the MENSA workout questionnaire. Cardiovascular reactivity and heart rate variability were assessed in resting, stress, and recovery stages. Continuous variables were expressed as mean ± Standard Deviation (S.D.). An unpaired t-test was employed to compare the means between the two groups. A p-value of less than 0.05 is considered significant.

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***Results:*** Following acute mental stress, children in the hypertensive group exhibited a notable rise in cardiovascular reactivity (p-value≤0.005). In the heart rate variability test, a significant reduction in Total power (p-value≤0.005) was noted during the stress phase, while a substantial increase in low frequency (p-value≤0.005) occurred in the recovery phase.

***Conclusion:*** This study observed increased cardiovascular reactivity and decreased overall heart rate variability, indicating heightened sympathetic activity and diminished vagal tone in subjects prior to hypertension onset. The rising rates of childhood hypertension, coupled with increased cardiovascular reactivity and reduced heart rate variability, underscore the urgent need for primary preventive measures and targeted intervention strategies for this at-risk group.

**KEYWORDS**

• Hypertension • Cardiovascular reactivity • Heart rate variability • Stress

## INTRODUCTION

Hypertension accounts for 13.5 million deaths worldwide every year and is one of the most prevalent risk factors for cardiovascular disease (CVD).[1] Exaggerated physiological reactions to acute stress (AMS), or more precisely, acute mental stress, have been proposed to be involved in mediating the CVD risk with hypertension.[2] Specifically, several lines of evidence suggest that exaggerated mental stress-induced cardiovascular and neuroendocrine reactions predict not only the premature development of hypertension and other precursors of coronary heart disease but also an accelerated progression of atherosclerosis and the likelihood of having a future acute coronary syndrome such as myocardial infarction. [3]

Hypertension clusters in families and a positive family history of hypertension represent a significant risk factor for future hypertension in the non-hypertensive offspring.[4] Childhood hypertension has become a widely investigated topic within the last decade due to its rising prevalence and associated sequelae.[5] Hypertension in childhood and adolescence is associated with adult cardiovascular morbidity and mortality.[6]

Heart rate (HR) and heart rate variability (HRV) are two standard indices of peripheral physiological arousal mediated by the autonomic nervous system. HR, the number of heartbeats per minute, typically increases under stress; HRV, the variation in heartbeats within a specific timeframe, typically decreases under stress. Thus, HRV can be conceptualized as a measure of autonomic flexibility, with some researchers suggesting it indexes an individual's ability to regulate emotional arousal in response to environmental demands.[7]

Numerous studies suggest a significant link between cardiovascular reactivity to stress and the development of future hypertension. This supports the reactivity hypothesis, which posits that heightened physical and psychological responses to stress indicate a subgroup at greater risk for cardiovascular disease.[8] Most studies conducted so far indicate that heightened cardiovascular reactivity (CVR) to stress could contribute to future hypertension.[9] Most of the previous research has focused on hypertensive patients directly, aiming to predict their stress reactivity rather than exploring their offspring 10. So, in the present study, we attempted to find out whether there is an effect of acute mental stress on cardiovascular reactivity and changes in heart rate variability (HRV) in children of hypertensive parents.

## MATERIAL AND METHODS

This case-control study was conducted in the Department of Physiology, Hamdard Institute of Medical Research Institute, Jamia Hamdard, New Delhi, after institutional ethical committee approval and informed consent was obtained from all the participating subjects explaining the study's objectives.

### Participant screening and analysis

Sixty participants were recruited from the general population, adhering to specific

inclusion and exclusion criteria. Among these, 30 participants were young adults aged 18 to 22 years, with at least one parent diagnosed with essential hypertension according to JNC VII guidelines. The exclusion criteria included individuals with a BMI of 19 kg/m² or lower and over 30 kg/m², those who engage in regular exercise, individuals with comorbidities such as hypertension, diabetes mellitus, bronchial asthma, hypothyroidism, hyperthyroidism, renal diseases, and endocrine disorders, as well as those taking any medications, smoking, chewing tobacco, or having a recent illness.

Additionally, thirty control participants were selected, consisting of healthy young adults matched for age and sex, who are children of parents with no family history of hypertension. These controls were chosen to establish a baseline for comparison with the study participants.

## STUDY PROTOCOL

The subject reported at the Department of Physiology, HIMSR, New Delhi, at 8:00 am. Informed consent is taken. Before recording the heart rate variability and cardiovascular reactivity on acute mental stress, the subject should abstain from tea, coffee, and ice cream at least 12 hours before testing. Clinical data recording includes detailed history: Name, age, sex, any history of relevant present or past illnesses (as per our inclusion and exclusion criteria), any relevant personal history (like smoking, exercise training, etc.), family, and treatment history. The anthropometric measurements of weight in kg and height in meters (m) were recorded using a balance beam scale and stadiometer. Body Mass Index (BMI) was calculated by dividing weight in kg by height in square meters $(m)^2$. Upon arrival, participants were given a 15-minute rest period, after which their resting blood pressure (BP) and heart rate (HR) were recorded while seated, along with heart rate variability (HRV). Subsequently, a 5-minute acute stress test was administered using the MENSA WORKOUT Questionnaire. During this stress test, BP was recorded at the 4-minute mark, and HR at the 5-minute mark. Short-term HRV was continuously monitored throughout the stress duration. Following the stress test, a 5-minute recovery period was allowed, during which BP, HR, and HRV were recorded again.

## MENSA QUESTIONNAIRE

In this study, acute mental stress was induced using problem-solving questions from the MENSA workout questionnaire. Participants experienced mental stress through three tasks: 1) reverse counting from 100, 2) subtracting 7 from 1000, and 3) completing the computer-based MENSA workout questionnaire. These tasks were conducted within a five-minute timeframe. Randomly selected questions from the MENSA workout questionnaire, which contains 30 questions, effectively produced the desired cardiovascular response, and completing the task took five minutes. Therefore, the MENSA workout questionnaire was chosen as the mental stressor for our study.

## CARDIOVASCULAR REACTIVITY

Cardiovascular reactivity refers to the difference in BP and HR measurements taken before and after the intervention, or the difference between baseline and the higher value observed.

Cardiovascular reactivity is represented as Δ HR, Δ SBP, Δ DBP.

### Heart Rate Variability

AD instruments lab Chart pro version 8.1.13 was used as data acquisition software for recording heart rate variability.

### Test Procedure:

All participants must refrain from consuming caffeine, alcohol, and tobacco products for at least 12 hours before the test. Subjects are asked to lie down and relax for 5 minutes before the start of the recording. They are also instructed to close their eyes and avoid any movements or conversation during the recording. Lead II Electrocardiographic (ECG) recording was done for 5 minutes in the supine position. After recording, all data were stored and analyzed offline. The time and frequency domain parameters of HRV were analyzed by detecting the R waves.

## STATISTICAL EVALUATION

Statistical analysis was conducted using SPSS. Continuous variables were expressed as mean ± Standard Deviation (S.D.). An unpaired t-test was employed to compare the means between

the two groups. A p-value of less than 0.05 is considered significant.

## RESULTS

This case-control study involved 30 healthy young adults aged 18 to 30, children of hypertensive parents (BMI < 29.9 kg/m²) as the case group. A matched control group of 30 healthy individuals with no family history of hypertension was also included.

The anthropometric data showed no significant difference between the control (offspring of normotensive) and cases (offspring of hypertensive parents) as shown in Table 1.

**Table 1:** Comparison of means of anthropometric data between control and cases group

| Variables | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) |
|---|---|---|
| Age (years) | 20.46 ± 0.83 | 20.37 ± 1.40 |
| Weight (Kg) | 57.54 ± 8.61 | 55.96 ± 7.36 |
| Height (cm) | 165.99 ± 9.13 | 162.81 ± 7.67 |
| BMI (kg/m2) | 20.78 ± 1.56 | 21.03 ± 1.46 |

### Cardiovascular Parameters at different stages

#### *At Resting Stage*

When cardiovascular parameters Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Heart Rate (HR) of the cases group were compared with the control group at Resting Stage we observed significantly increased resting SBP, DBP, and HR in cases as shown in Table 2.

**Table 2:** Comparison of means of SBP, DBP, and HR at Resting Stage

| | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) | p-value |
|---|---|---|---|
| SBP (mmHg) | 113.9 ± 6.7 | 117.3 ± 7.3 | 0.0159* |
| DBP (mmHg) | 74.22 ± 4.2 | 75.22 ± 5.4 | 0.0063* |
| HR (bpm) | 68.33 ± 7.3 | 71.33 ± 7.8 | 0.001* |

**Note:** Analysis was done using an unpaired t-test. *p-value ≤ 0.05 was considered statistically significant.

#### *At Stress Stage*

When cardiovascular parameters SBP, DBP and HR of cases were compared with the control group at stress stage we observed significant increase in SBP and HR in cases as compared to the control group. Whereas there was no significant increase in DBP in cases when compared to the control group as shown in Table 3.

**Table 3:** Comparison of means of SBP, DBP & HR at Stress Stage

| | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) | P - value |
|---|---|---|---|
| SBP (mmHg) | 118.8 ± 3.4 | 123.7 ± 5.4 | 0.011* |
| DBP (mmHg) | 81.22 ± 2.1 | 85.23 ± 2.4 | 0.09049 |
| HR (bpm) | 78.33 ± 7.2 | 84.20 ± 8.2 | 0.011* |

**Note:** Analysis was done by unpaired t-test. *p-value ≤ 0.05 was considered statistically significant.

#### *At Recovery Stage*

When cardiovascular parameters SBP, DBP, and HR of the cases were compared with the control group at the recovery stage, we observed significantly raised SBP, DBP, and HR values in the cases as compared to the control group, as shown in Table 4.

**Table 4:** Comparison of means of SBP, DBP & HR at Recovery Stage

| | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) | P-Value |
|---|---|---|---|
| SBP (mmHg) | 115.2 ± 3.4 | 119.2 ± 5.7 | 0.001* |
| DBP (mmHg) | 74.67 ± 4.5 | 79.33 ± 6.1 | 0.001* |
| HR (bpm) | 73.93 ± 3.8 | 77.33 ± 5.9 | 0.001* |

**Note:** Analysis was done by unpaired t test. *p-value ≤ 0.05 was considered statiscally significant.

#### *Cardiovascular reactivity*

When Δ HR, Δ SBP, Δ DBP, denoting the cardiovascular reactivity of the cases, are compared with the control group, we observe significantly increased cardiovascular reactivity in the cases group compared to the control group, as shown in Table 5.

**Table 5:** Comparison of means of cardiovascular reactivity in control and cases group

| | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) | P-value |
|---|---|---|---|
| Δ SBP (mmHg) | 5.267 ± 3.3 | 8.933 ± 4.5 | 0.0131* |
| Δ DBP (mmHg) | 5.867 ± 3.5 | 10.73 ± 5.3 | 0.0056* |
| Δ HR (bpm) | 8.733 ± 4.1 | 15.93 ± 6.1 | 0.009* |

**Note:** Analysis was done by unpaired t test. *p-value ≤ 0.05 was considered statistically significant.

## Heart Rate Variability (HRV) test

### *At Resting Stage*

**Table 6:** Comparison of means of HRV Parameters between cases and control group at Resting Stage

| | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) | p-value |
|---|---|---|---|
| SDRR | 58.59 ± 4.3 | 43.36 ± 6.7 | 0.00196* |
| RMSSD | 72.58 ± 3.6 | 60.18 ± 7.9 | 0.0026* |
| PNN50 | 90.30 ± 2.6 | 90.86 ± 3.5 | 0.2302 |
| VLF | 1.956 ± 1.9 | 2.764 ± 1.7 | 0.4402 |
| LF | 3.802 ± 2.1 | 3.984 ± 1.9 | 0.5671 |
| HF | 2.873 ± 1.1 | 0.7302 ±1.3 | 0.3075 |
| LF/HF | 0.2779 ± 1.08 | 0.3603 ± 1.06 | 0.0266 |
| TP | 2789 ± 6.3 | 20132 ± 8.7 | 0.0125* |

**Note:** Analysis was done using an unpaired t-test. *p-value ≤ 0.05 was considered statistically significant.

### *At stress stage*

**Table 7:** Comparison of means of HRV Parameters between cases and control group at stress stage

| | Control N=30 (Mean ± SD) | Cases N=30 (Mean ± SD) | p-value |
|---|---|---|---|
| **SDRR** | 50.56 ± 2.3 | 43.32 3.1± | 0.1295 |
| **RMSSD** | 61.26 2.8± | 59.41± 2.9 | 0.2808 |
| **PNN50** | 90.07± 4.7 | 90.13± 4.2 | 0.3305 |
| **VLF** | 3.938± 1.8 | 3.063±1.6 | 0.4914 |
| **LF** | 3.984± 1.6 | 4.034±2.1 | 0.3274 |
| **HF** | 3.326± 1.09 | 2.747± 1.05 | 0.5670 |
| **LF/HF** | 0.3337± 1.05 | 0.3092± 1.06 | 0.3868 |
| **TP** | 2927± 5.3 | 2188± 6.4 | 0.0001* |

**Note:** Analysis was done using an unpaired t-test. *p-value ≤ 0.05 was considered statistically significant.

### *At Recovery Stage*

**Table 8:** Comparison of means of HRV Parameters between cases and control group at recovery Stage

| | Control N=30 (Mean ± SD) | Cases =30 (Mean ± SD) | P-value |
|---|---|---|---|
| SDRR | 50.91± 2.1 | 57.56±3.4 | 0.0805 |
| RMSSD | 70.93± 3.2 | 77.64± 3.4 | 0.133 |
| PNN50 | 128.1± 4.2 | 92.47±6.5 | 0.0001* |
| VLF | 1.638± 1.2 | 1.190± 1.4 | 0.6195 |
| LF | 4.152± 1.4 | 8.1± 2.5 | 0.0001* |
| HF | 2.996± 1.4 | 3.196± 1.7 | 0.4355 |
| LF/HF | 0.2974± 1.07 | 0.2854± 1.04 | 0.7817 |
| TP | 2858± 8.9 | 2533± 7.5 | 0.0667 |

**Note:** Analysis was done using an unpaired t-test. *p-value ≤ 0.05 was considered statistically significant.

## DISCUSSION

As the prevalence of hypertension and cardiovascular complications are increasing in the younger age group, this study holds importance to study the effect of short-term mental stress on cardiovascular study and heart rate variability in children of hypertensive parents.

In this study, 30 cases of children of hypertensive parents and 30 age sex and BMI controls were taken (Table 1). It was observed that the resting SBP, DBP, and HR were significantly increased in the cases (Table 2). This suggests increased sympathetic activity even at the resting stage in children of hypertensives.

A marked increase in systolic blood pressure (SBP) and heart rate (HR) was noted during the stress phase in children of hypertensive parents (Table 3). This swift elevation in blood pressure is fueled by the sympathetic-adrenal medullary axis, which prompts catecholamine release, consequently raising both blood pressure (BP) and heart rate (HR).[11] The study reveals delayed responses, evident from the significant increase in SBP, diastolic blood pressure (DBP), and HR during the recovery phase in cases, compared to the control group (Table 4). This elevation is linked to the activation of the hypothalamopituitary-adrenal axis, which amplifies catecholamines' impact on vascular reactivity.[12] The slow response stems from the HPA axis activation, leading to the release of corticotropin-releasing hormone (CRH) from the paraventricular nucleus of the hypothalamus into the bloodstream.[13]

Acute stress causes an elevation in heart rate, improves heart muscle contractions, expands the heart, and reallocates blood flow to larger muscles. In contrast, chronic stress results

in sustained activation of the sympathetic nervous system and the HPA axis, leading to heightened levels of stress hormones, such as cortisol and epinephrine.[14]

Furthermore, it has been suggested that the rise in BP and HR responses is due to a decrease in vagal tone or an increase in the stimulation of the afferent sympathetic nervous system.[15]

This study also noted a marked increase in cardiovascular reactivity among children with hypertension (Table 5). Regardless of the underlying mechanisms, researchers have concluded that heightened reactivity or delayed recovery contributes to the development of cardiovascular disease risk due to stress and other psychosocial factors.[16]

Heart rate variability (HRV) evaluates how acute mental stress affects cardiac autonomic function. This device is non-invasive and tracks changes in heart rate (HR) and fluctuations in the R–R (N-N) interval. The study observed a notable decrease in total power and SDNN during the resting stage in children of hypertensive parents (Table 6). This indicates reduced parasympathetic activity. Among the various methods for examining HRV, one of the most straightforward and commonly used is the standard deviation of the mean R–R interval (SDRR/SDNN).[17] Research indicates that healthy individuals exhibit an increase in heart rate (HR) in response to acute mental stress, alongside a decrease in standard deviation of normal-to-normal intervals (SDNN).[18] This study noted a notable reduction in total power among children of hypertensive parents (Table 7). This reduction in total power points to a significant decline in parasympathetic activity during stress when comparing children of hypertensives to those with non-hypertensive parents. The irregular patterns observed in immediate heartbeat fluctuations are believed to stem from multiple factors, including autonomic activity (both parasympathetic and sympathetic), hormones, neurochemical substances, end diastolic volume, and afterload.[19] Moreover, it has been proposed that even in normal conditions, the heart rate (HR) generation system tends to fluctuate between stable positions. This functional variability of the sinoatrial (SA) node not only allows the heart to transition between states but also aids in addressing various known and unknown challenges. From this viewpoint, the stability of the HR rhythm and the reduced responsiveness of the SA node to different situations are associated with an increased risk of future cardiovascular diseases.[20]

A variety of testing methods are employed to evaluate stress responses in humans. Heart rate variability examines the fluctuations in the time intervals between consecutive heartbeats, indicating the balance between the sympathetic (fight-or-flight) and parasympathetic (relaxation) nervous systems.[21] In this study, an increase in LF and a decrease in pNN50 were noted during the recovery phase in children of hypertensive parents (Table 8). Similar patterns observed in another study suggest that reduced heart rate variability correlates with sympathetic dominance and heightened stress levels, while increased heart rate variability indicates stress resilience and enhanced cardiovascular health.[22]

Other studies have observed a rise in the LF/HF ratio and LFnu, along with a decline in HFnu among children of hypertensive parents, indicating a sympathovagal imbalance. Heart Rate Variability (HRV) provides an accurate assessment of the autonomic nervous system and is valuable for early hypertension diagnosis. Utilizing HRV for early detection in high-risk individuals can help prevent additional cardiovascular and cerebrovascular issues.[23]

Our study reveals that incidence of prehypertension and the risk of cardiovascular dysfunction in relation to altered sympathovagal response is more in children of hypertensive parents than in the children of normotensive parents. Sympathovagal imbalance in the form of increased sympathetic drive and decreased parasympathetic drive can lead to prehypertension in these genetically predisposed individuals.[24]

Numerous studies have shown that lower HRV correlates with cardiovascular diseases and mortality.[25] Hypertension typically links to heightened sympathetic activity and diminished parasympathetic activity. Previous reports indicated reduced HRV in adults with hypertension.[26] Interestingly, this phenomenon is now also observed in children of hypertensive parents. Therefore, autonomic reactivity may serve as a predictive indicator for future development of cardiovascular issues hypertension.[27]

These findings emphasize the potential role of genetic predisposition in the early onset of autonomic dysregulation.

This study observed increased cardiovascular reactivity and decreased overall heart rate variability, indicating heightened sympathetic activity and diminished vagal tone in subjects prior to hypertension onset. The rising rates of childhood hypertension, coupled with increased cardiovascular reactivity and reduced heart rate variability, underscore the urgent need for primary preventive measures and targeted intervention strategies for this at-risk group. Engaging in acute exercise may help diminish the effects of cardiovascular hyper-reactivity on disease progression for those with a family history of hypertension, as demonstrated by previous research 28. Additionally, pharmacological treatments, weight loss, calorie restriction, and exercise should be implemented to enhance autonomic function and lower blood pressure and insulin resistance in this high-risk population, thereby reducing the likelihood of developing hypertension and cardiovascular diseases in the future.

The study's strength is its examination of acute stress effects on cardiovascular reactivity and heart rate variability across all three stages: resting, stress, and recovery. However, a limitation is the need for a larger sample size. Future research could also incorporate biochemical markers of stress and cardiovascular diseases for enhanced understanding.

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## AUTHOR CONTRIBUTIONS

All authors contributed equally. All authors read and approved the final manuscript and have no conflict of interest.

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