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Original Research Article

Study of the Effect of Acute Mental Stress on Heart Rate Variability in Obese Adults in Darbhanga District

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Abstract:

Background: Obesity and stress are two lifestyle risk factors that have an impact on a variety of physiological aspects of the human body. Autonomic neural activity determines how well a person can handle stress. In the current study, we compared the frequency domain indices of HRV in obese and non-obese adults in Darbhanga District before and during a mental arithmetic stress test.

Methods: The current study was carried out at Department of Physiology, Darbhanga Medical College, Laheriasarai, Bihar from April 2024 to June 2024. Prior to the study start, approval from the institute ethics committee was acquired.

Results: Similar in age for five minutes, 60 male subjects, 30 obese and 30 non-obese performed a mental arithmetic stress test. Prior to the stress test, the rise in LFnu and LF/HF ratio in the obese group was statistically significant ($p < 0.001$). Obese patients showed decreased LFnu, HFnu, and LF/HF ratio during the mental arithmetic test, with the LFnu reduction statistically significant ($p<0.01$). In contrast, there was a statistically significant decrease in HFnu ($p>0.05$) and an increase among LFnu ($p<0.001$) and LF/HF ratio ($p<0.01$) in non-obese patients.

Conclusion: The findings imply that the obese group has lower autonomic neural activity in response to mental stress.

Keywords: HRV, Mental stress, LFnu, HFnu, LF/HF ratio.

Introduction

The present is an era of stress. There is growing proof that certain "triggers" including emotional and physical stress can cause myocardial infarction and abrupt cardiac death [1,2]. Numerous studies have shown that having a high body mass index (BMI) increases your chance of developing cardiovascular disease [3,4]. Physiologically, managing stress requires modifying the neuroendocrine and autonomic nerve systems so that organ damage is kept to a minimum. The cardiovascular system is the main system that stress will have a long-term negative impact on. The balance between two opposing forces, i) central nervous system arousal, which is sympathoexcitatory, and ii) arterial baroreflex activation, which is sympathoinhibitory is assumed to be the cause of the rise in sympathetic nerve activity in humans during mental stress. In the past two decades, there has been a considerable correlation between cardiovascular mortality, especially sudden cardiac death, and the autonomic nervous system [5,6]. Lethal arrhythmia symptoms, such as elevated sympathetic activity, decreased vagal tone, or both, have prompted the development of tests to assess the autonomic nervous system. Heart rate variability is a promising quantitative indicator of the autonomic nervous system (HRV). HRV's frequency domain analysis provides insightful data on the autonomic nervous system's sympathetic and parasympathetic components (ANS).Even if the autonomic neural activity is normal in the basal condition, stress testing reveals autonomic malfunction through HRV. The Stroop test, general knowledge questions, reaction time tasks, memory tests, simulated speech tasks, mental arithmetic tests, and other tests can all be used to create mental stress. The most typical, simple to give, and accurately controlled by the investigator is the mental arithmetic test. Numerous studies have demonstrated that healthy, non-obese persons had increased sympathetic activity to mental stress [7,8]. However, there are few studies that use frequency analysis of HRV under mental stress to

evaluate ANS in obese individuals. So, the current investigation was started.

Material and Methods

The present study was conducted in the Department of Physiology, Darbhanga Medical College, Laheriasarai, Bihar from April 2024 to June 2024. Permission from the institute's ethics committee was obtained prior to the commencement of the study.

A total of 30 healthy male volunteers in the Darbhanga District were used for the study. They ranged in age from 20 to 23 and had a BMI of less than 25 kg/m^2.

A study group of 30 obese people (BMI > 30 kg/m2) who were age and sex matched was also selected from the same district. None of the participants had ever received a cardiovascular disease diagnosis, a psychiatric condition diagnosis, or a medication-related heart rate-altering finding. Following a thorough explanation of the non-invasive technique's detailed method in their native language, each participant voluntarily provided their informed consent. The subjects were instructed to stop all exercise, eating, and drinking two hours before to the start of the trial. The study was conducted early in the day. After 10 minutes of rest, a baseline blood pressure was taken using a mercury sphygmomanometer. Then, using an AD Instruments power lab, a baseline ECG in lead II was recorded for 5 minutes in both the non-obese and the obese group. Computers were used for signal gathering, storage, and processing. On a detachable hard drive, the digitalized ECG signals were saved for analysis. Using the HRV module software for Lab Chart, the frequency domain of HRV analysis, including LFnu, HFnu, and LF/HF ratio records, was performed. By identifying the R waves from each ECG waveform, the HRV module analyses the beat-to-beat interval fluctuation in ECG records. R wave fiducial locations that were stable, noise-free, and ectopic-free were found, and power spectra of these R waves were obtained using the Fast Fourier Transform. The sampling rate was 500 Hz. A mental stressor was delivered using the mental stress test that Holly R. [8] utilised and reported in her work. The subjects were instructed to continually subtract three or four numbers from a three-digit figure as part of the test, and they were then asked to speak their responses out.

The test-takers were instructed to work swiftly and were gently reprimanded for giving incorrect answers. To boost the sympathetic response to perceived mental stress, this was done. Both the control group and the obese group had their ECGs recorded for 5 minutes during a mental stress test, and the above-described HRV analysis was performed. Calculating the arithmetic mean and standard deviation allowed us to compare the numbers between the two groups (obese and non obese). To evaluate the significance of variations in autonomic function across the groups, an unpaired t test was used. Also utilising a paired t test within the group itself. The statistical analysis was performed using SPSS version 16.

Results

In the current study, 30 patients aged 17 to 20 who were obese and 30 who were not were included. Table I displays the physical characteristics of the two groups.

Table 2 displays the systolic and diastolic blood pressure readings as well as a frequency domain study of HRV prior to mental stress. In comparison to non-obese patients, there was a statistically significant increase in SBP ($p<0.001$) and DBP ($p<0.001$).

Table 1: Comparison of physical parameters between Non Obese and Obese subjects

	Non obese (Mean±SD)	Obese (Mean±SD)	p-value
Age(years)	19±0.7	19±0.4	0.07
Height(cm)	171±6	171±4	0.7
Weight(kg)	62±9	93±7	<0.001***
BMI(kg/m^2)	21±2	32±2	<0.001***

*****p value<0.001 suggests very highly significant.**

Age and height did not significantly differ between obese and non-obese people. However, there was a substantial (p value <0.001) difference in weight and body mass index between the two groups.

Table 2: Systolic blood pressure, diastolic blood pressure and frequency domain parameters of HRV at rest in non-obese and obese subjects

	Non obese (Mean±SD)	Obese (Mean±SD)	p-value
SBP(mmHg)	113±6	126±8	<0.001***
DBP(mmHg)	66±4	77±6	<0.001***
LFnu	30±14	49±13	<0.001***
HFnu	48±12	37±7	<0.001***
LF/HF	0.7±0.4	1.4±0.5	<0.001***

*****p value <0.001 suggests very highly significant.**

When compared to non-obese people, there was a substantial increase in LF nu-Low frequency power in normalised unit ($p < 0.001$) and LF/HF ratio ($p < 0.001$). But as compared to non-obese individuals, there was a substantial decrease in HFnu-High frequency power in normalised unit ($p < 0.001$).

Table 3: Comparison of Frequency domain parameters of HRV at rest and during mental stress in Non-obese subjects

	At Rest (Mean±SD)	During mental stress (Mean±SD)	p-value
LFnu	30±14	35±13	<0.001***
HFnu	48±12	46±8	0.4
LF/HF	0.7±0.4	0.8±0.4	0.003**

*****p value <0.001 suggests very highly significant. **p value <0.01 suggests highly significant.**

In non-obese persons, LFnu and LF/HF ratio increased statistically significantly with mental stress.

Table 4: Comparison of Frequency domain parameters of HRV at rest and during mental stress in obese subjects

	At Rest (Mean±SD)	During mental stress (Mean±SD)	p-value
LFnu	49±13	38±18	0.002**
HFnu	37±7	34±10	0.2
LF/HF	1.4±0.5	1.2±0.7	0.12

****p value <0.01 suggests highly significant.**

During times of mental stress, LFnu decreased statistically significantly in obese people. Although HF nu and the LF/HF ratio decreased, these changes were not statistically significant.

Discussion

In our study, obese people had significantly higher LFnu and LF/HF ratios at rest compared to non-obese people. An rise in the LF/HF ratio, which reflects sympathovagal balance, denotes sympathetic dominance [9]. In addition, the obese group's blood pressure was substantially higher than that of the control group. Our research is in line with that of Louis J. Aronne et al, Khwaja Sarwari et al, and Chethan HA et al [10,11,12], which also revealed that obese people have greater sympathetic activity under resting conditions.

The rise in LFnu and LF/HF ratio after mental stress in non-obese people suggested increased sympathetic activity because the sympathovagal balance was tilted toward sympathetic predominance. However, detecting changes in autonomic regulation to mental stress in obese individuals was our primary goal. With obesity, we also anticipated a similar sympathetic overdrive in response to stress. On the other hand, in obese people, stress caused a drop in LFnu, HFnu, and LF/HF ratio. Although not statistically significant in our investigation, the drop in HFnu, a component that supports parasympathetic activity [9], suggests a decline in parasympathetic activity.

Additionally, LFnu revealed a statistically significant ($p=0.002$) decrease in obese people under stressful conditions. The combined decrease in HFnu, LFnu, and LF/HF ratio may likely imply that there is vagal withdrawal in obese individuals to mental stress, which is a risk factor for developing cardiac problem. This is because LF nu signals both sympathetic activity and parasympathetic activity [9].

Higher morbidity and death are also believed to be caused by the lower stress response. Although LFnu and LF/HF ratio in obese people remained higher under mental stress compared to non-obese people, obese people nevertheless reacted to mental stress by withdrawing their parasympathetic nervous system. Our research is in line with that of K Laederach-Hofmann [13].

Therefore, most evidence, including that from our study, indicates that obesity is characterised by a predominance of the sympathetic nervous system (SNS) in the baseline state and a reduction in the ANS's reactivity to diverse sympathetic stimuli [14,15,16].

We get to the conclusion that both the non-obese and the obese group's HRV exhibits minute yet significant changes in autonomic control to mental stress. However, during mental stress, there is decreased autonomic neural activity in the obese group, indicating that being obese may be a risk factor for the early onset of many stress-related illnesses.

As a result, measuring HRV under mental stress can be a useful method for identifying early subclinical autonomic impairment in obese people. We did not assess the subjective level of stress in either group, which may have provided some information on the reasons why the autonomic regulation of the body alters during stress. Plasma norepinephrine levels might have also been measured, which might have provided more information. The gender differences in stress reactivity could likewise be the subject of more research.

Conclusion

The findings imply that the obese group has lower autonomic neural activity in response omental stress.

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