Harsukh Educational Charitable Society
International Journal of Community Health and Medical Research
Journal home page:www.ijchmr.com doi: 10.21276/ijchmr

ISSN E: 2457-0117 **ISSN P:**2581-5040 **Index Copernicus** ICV 2018=62.61

Original Research

Assessment Of Effect Of Stress On Heart Rate Variability In Obese Subjects

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ABSTRACT

Background: Heart rate variability (HRV) is the physiological phenomenon of variation in the time interval between heartbeats. The present study was conducted to assess effect of stress on heart rate variability in obese subjects. **Materials & Methods:** The present study was conducted on subjects (25 obese, 25 non- obese) of both genders. In all subjects, a baseline blood pressure was recorded using mercury sphygmomanometer. The baseline ECG was recorded for 5 minutes in both groups. The frequency domain of HRV analysis such as LFnu, HFnu and LF/HF ratio recordings was carried out. The mental stress test was applied as a mental stressor. **Results:** SBP in group I was 112.6 mm Hg and in group II was 134.8 mm Hg, DBP in group I was 68.4 mm Hg and in group II was 76.6 mm Hg. The difference was significant (P< 0.05).LFnuin group I at rest was 31.5 and during mental stress was 36.7, HFnu in group I at rest was 46.4 and during mental stress was 49 and LF/HF ratio in group I at rest was 0.8 and during mental stress was 0.9. The difference was significant (P< 0.05). LFnuin group I at rest was 47.5 and during mental stress was 39.2, HFnu in group I at rest was 38.4 and during mental stress was 32.4 and LF/HF ratio in group I at rest was 1.3 and during mental stress was 1.2. The difference was significant (P< 0.05). **Conclusion:** Author found reduced autonomic neuronal activity to mental stress in obese subjects as compared to non- obese.

Key words: Autonomic neuronal activity, Obese, Stress

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This article may be cited as: Porwal NB. Assessment Of Effect Of Stress On Heart Rate Variability In Obese Subjects. HECS Int J Comm Health Med Res 2019; 5(2):48-50.

INTRODUCTION

Modern day is the age of stress. Evidence is accumulating that specific 'triggers' such as psychological and physical stress may precipitate myocardial infarction and sudden cardiac death. Many studies have demonstrated an association of high body mass index (BMI) with greater risk for cardiovascular disease.[1]

Heart rate variability (HRV) is the physiological phenomenon of variation in the time interval between heartbeats. It is measured by the variation in the beat-to-beat interval. Other terms used include: "cycle length variability", "RR variability" and "heart period variability".[2]Physiologically, coping with stress imposes modulation in neuroendocrine and autonomic nervous system such that there is minimal organ damage. The major system that is going to be affected in the long run, in response to stress is cardiovascular system. There is interest in HRV in the field of psychophysiology.[3] For example, HRV is related to emotional arousal. High-frequency (HF) activity has been found to decrease under conditions of acute time pressure and emotional strain and elevated state anxiety, presumably related to focused attention and motor inhibition. HRV has been shown to be reduced in individuals reporting to worry more. In individuals with post-traumatic stress disorder (PTSD), HRV and its HF component (see below) is reduced whilst the low-frequency (LF) component is elevated. Furthermore, PTSD patients demonstrated no LF or HF reactivity to recalling a traumatic event. Many studies have shown a higher sympathetic activity to mental stress in obese subjects.[4]The present study was conducted to assess effect of stress on heart rate variability in obese subjects.

MATERIALS & METHODS

The present study was conducted in the department of Physiology. It comprised of age matched 50 subjects (25 obese, 25 non- obese) of both genders. All were informed regarding the study and written consent was obtained. Ethical clearance was obtained prior to the study.

General information such as name, age, gender etc. was recorded. Subjects were divided into 2 groups. Group I were non- obese and group II were obese patients. In all subjects, a baseline blood

pressure was recorded using mercury sphygmomanometer. The baseline ECG was recorded for 5 minutes in both groups. The frequency domain of HRV analysis such as LFnu, HFnu and LF/HF ratio recordings was carried out. The mental stress test was applied as a mental stressor. The ECG was recorded for 5 minutes during mental stress in both groups and HRV analysis was done. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Assessment of blood pressure in both groups

parameters	Group I	Group II	P value
SBP	112.6	134.8	0.01
DBP	68.4	76.6	0.05

Table I, Graph I shows that SBP in group I was 112.6 mm Hg and in group II was 134.8 mm Hg, DBP in group I was 68.4 mm Hg and in group II was 76.6 mm Hg. The difference was significant (P< 0.05)

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Graph I: Assessment of blood pressure and Frequency domain parameters of HRV at rest in both groups

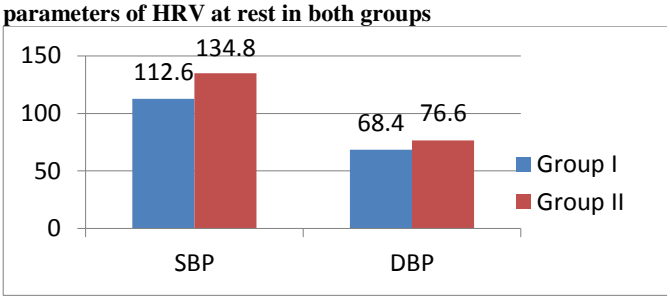

Table II Frequency domain parameters of HRV at rest and during mental stress in both groups

Group I

	Rest	Mental Stress	P value
LFnu	31.5	36.7	0.01
HFnu	46.4	49.0	0.5
LF/HF ratio	0.8	0.9	0.01
Group II			
	Rest	**Mental Stress**	**P value**
LFnu	47.5	39.2	0.001
HFnu	38.4	32.4	0.42
LF/HF ratio	1.3	1.2	0.57

Table II shows that LFnu in group I at rest was 31.5 and during mental stress was 36.7, HFnu in group I at rest was 46.4 and during mental stress was 49 and LF/HF ratio in group I at rest was 0.8 and during mental stress was 0.9. The difference was significant (P< 0.05).

LFnuin group I at rest was 47.5 and during mental stress was 39.2, HFnu in group I at rest was 38.4 and during mental stress was 32.4 and LF/HF ratio in group I at rest was 1.3 and during mental stress was 1.2. The difference was significant (P< 0.05).

DISCUSSION

The increase in sympathetic nerve activity in humans during mental stress is thought to reflect the balance between two opposing forces i) central nervous system arousal, which is sympathoexcitatory, and ii) arterial baroreflex activation, which is sympathoinhibitory. Last two decades have witnessed significant relationship between the autonomic nervous system and cardiovascular mortality, including sudden cardiac death.[5]Decreased parasympathetic nervous system (PSNS) activity or increased SNS activity will result in reduced HRV. High frequency (HF) activity (0.15 to 0.40 Hz), especially, has been linked to PSNS activity. Activity in this range is associated with the respiratory sinus arrhythmia (RSA), a vagally mediated modulation of heart rate such that it increases during inspiration and decreases during expiration. Less is known about the physiological inputs of the low frequency (LF) activity (0.04 to 0.15 Hz). Though previously thought to reflect SNS activity, it is now widely accepted that it reflects a mixture of both the SNS and PSNS.[6] The present study was conducted to assess effect of stress on heart rate variability in obese subjects.In presents study, subjects were divided into 2 groups. Group I were non- obese and group II were obese patients. We found that systolic blood pressure (SBP) in group I was 112.6 mm Hg and in group II was 134.8 mm Hg, diastolic blood pressure (DBP) in group I was 68.4 mm Hg and in group II was 76.6 mm Hg.

We observed that LFnuin group I at rest was 31.5 and during mental stress was 36.7, HFnu in group I at rest was 46.4 and during mental stress was 49 and LF/HF ratio in group I at rest was 0.8 and during mental stress was 0.9. Similarly, LFnuin group I at rest was 47.5 and during mental stress was 39.2, HFnu in group I at rest was 38.4 and during mental stress was 32.4 and LF/HF ratio in group I at rest was 1.3 and during mental stress was 1.2.

Khwaja et al[7] found that before stress test LFnu and LF/HF ratio showed statistically significant increase in obese group. During mental arithmetic test LFnu, HFnu and LF/ HF ratio was reduced in obese subjects with LFnu reduction being statistically significant whereas in non obese subjects, there was statistically increase in LFnu and LF/HF ratio with reduction in HFnu which was statistically not significant.Studies revealed that LF nu indicates both sympathetic activity and parasympathetic activity, the combined decrease in HFnu, LFnu and LF/HF ratio may probably indicate that there is vagal withdrawal in obese individuals to mental stress which is a risk factor in developing cardiac disorder. The decreased stress reactivity is also thought to play a role as a factor for higher morbidity and mortality. Although LFnu and LF/HF ratio in obese individuals remained elevated during mental stress compared to non obese individuals, yet obese individuals responded to mental stress by parasympathetic withdrawl.[8]

CONCLUSION

We found reduced autonomic neuronal activity to mental stress in obese subjects as compared to non- obese.

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