A Comparative Study of Pre Prandial and Post Prandial Heart Rate Variability between Obese and Non Obese Young Women Aged 18-25 Years

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Abstract— Background: In obesity, as excessive adipose tissue accumulates, an altered metabolic profile occurs along with a variety of adaptations/alterations in the cardiac structure and function even in the absence of co-morbidities. Objective: To compare pre prandial and post prandial heart rate variability between young healthy non obese and obese females aged 18-25 years. Methods: 50 obese and 50 non obese young healthy females aged 18-25 years were selected based on body mass index (BMI). Heart rate variability was recorded in both pre prandial and post prandial state using ECG V: 52 (HRV) power spectral analysis to identify separate frequency components, i.e., total power (TP), low-frequency power (LF), high frequency power (HF) and low frequency to high frequency ratio. Results: All the statistical methods were carried out using the SPSS software for Windows (version 16.0) and Minitab (version 11.0). The Paired-Samples T Test procedure was done to compare the means of two variables for a single group. The Independent-Samples T Test procedure was done to compare the means for two groups of cases. P value less than or equal to 0.001 was considered statistically significant. HRV analysis found significantly lower (p<0.001) values of TP, LF(ms2), HF(ms2) and HF (n.u.) and significantly higher values (p<0.001) of LF (n.u.) and LF/HF ratio among the obese group in both pre prandial and post prandial state respectively compared to non obese group. Conclusion: Our data indicate that obese subjects have decreased parasympathetic activity as evidenced by decrease in TP, LF(ms2), HF(ms2) and HF (n.u.) and increase in sympathetic activity as evidenced by increase in LF (n.u.) and LF/HF in both pre prandial and post prandial state.

Keywords: Autonomic nervous system, Food intake, Heart rate variability, obese young women

I. INTRODUCTION

Obesity is an emerging global health problem. Nutritional problem in India is gradually shifting from undernourishment to obesity. It is a disease, which has evolved with the advent of civilization, sedentary lifestyle and high calorie diet. Obesity is one of the causative factors for multiple co-morbid conditions leading to metabolic and cardiac disorders.

The incidence of overweight and obesity are increasing around the world especially in young adults and middle-aged people. With obesity, there are increased chances of acquiring endocrinal diseases, genetic and metabolic disorders.

Obesity is one of the risk factors attributed for the development of lipid abnormalities, insulin resistance, hypertension etc. Growing number of evidences indicate association of obesity and sudden cardiac deaths.

Imbalance in cardiac autonomic activity might be a predisposing factor for arrhythmogenesis and subsequently sudden cardiac deaths.

Obesity is accompanied with varied combination of abnormalities in the autonomic nervous system imbalance.

There is some difference of opinion on pattern of abnormal cardiac autonomic activity in obese humans.

One view is that obese people have a higher sympathetic tone, but it has been proved in some studies correlating with the catecholamine levels, but there is also evidence of reduced cardiac sympathetic tone in some studies. This controversy was partly explained on the duration of obesity.

There are several tests to determine the autonomic activity. Recently the most accepted tool is determining Heart Rate Variability.

Heart Rate Variability is a specific and sensitive noninvasive tool to evaluate cardiac autonomic activity. HRV is the degree of variation of the heart rate during the day under the balanced influence of sympathetic and parasympathetic component of the cardiac autonomic nervous system. It expresses the total amount of variation of both instantaneous heart rate and RR intervals. HRV also indicate the extent of neuronal damage to autonomic nervous system. HRV has been shown to be a good tool to quantify the tone of autonomic nervous system to the myocardium. It has also been associated with high predictive value in many diseases where a disturbance in the autonomic activity is likely, for example in conditions like cardiovascular diseases, metabolic diseases like diabetes mellitus, neurological diseases like Parkinson’s disease, trauma, gastrointestinal disease like irritable bowel syndrome, chronic respiratory disease like asthma, certain infections, neoplasia, surgeries like vagotomy and prognosis of disease.

The gastrointestinal tract (GIT) is supplied by the enteric nervous system comprising the myenteric plexus of Auerbach, the external and the internal submucous plexuses. This apart, the GIT comes under the influence of the autonomic nervous system that has the sympathetic and parasympathetic components. Ingestion of food is a visceral stimulus that leads to various physiological adjustments which include metabolic and cardiovascular changes such as increased blood flow to GIT and a decreased skeletal muscle blood flow.

Food intake causes peptides to be released in the GIT, which leads to vasodilatation locally. This leads to...
The enteric nervous system that controls the pacemaker and motor activity of GIT, communicates with the CNS, interacting with the heart through the ANS. The measured heart rate is modulated by the two main components of ANS. These are parasympathetic and sympathetic nervous system. An increase in sympathetic activity increases the heart rate and increased parasympathetic activity decreases the heart rate, thus balancing each other. Vagal activity is dominant in resting conditions.

This study is an effort to assess the effect of obesity on cardiac autonomic activity using Heart Rate Variability in young females.

II. MATERIAL AND METHODS

This study consists of 100 healthy female subjects in the age group of 18-25 years comprising 50 non-obese subjects and 50 obese subjects.

This study was conducted in the department of Physiology, Adichunchanagiri Institute of Medical Sciences, BG Nagar, Mandya district.

The selected students had regular, 30±3 days menstrual cycles for at least 6 months prior to this study. After detailed enquiry of the medical history of the subjects, those with history of smoking, alcoholism, medical illness were excluded. Subjects on oral contraceptive pill, hormonal replacement therapy and also subjects with noticeable weight gain or weight loss over the preceding 3 months were excluded from the study. Informed written consent was obtained from all participants, and the experiment protocol was approved by Ethical Committee of the college. Subjects selected for study were on 10th -12th day of their menstrual cycle.

A. Assessment of Body mass index

The body weight of the subjects was measured using a pedestal type of weighing scale with a maximum capacity of 150 kg. The body weight was considered to the nearest of 0.1 kg. Height without footwear was measured using a vertical scale (Avery, India) with an accuracy 0.5 cm and was rounded to the nearest 0.01 m.

B. The BMI was calculated as

\[
BMI = \frac{\text{Weight in kg}}{\text{(height in meters)}^2}
\]

Subjects were classified into 2 groups based on BMI as follows:

Normal weight – BMI < 18.5 – 24.99 kg/m²

Obese – BMI > 30 kg/m²

C. Procedure

Each of the subject was allowed to have breakfast in the morning at 8.30 AM. Standard lead II ECG was recorded with subject in supine position using ECG V: 52 (Heart Rate Variability analysis software) manufactured by NIVIQUR Meditech Pvt Ltd, Bengaluru and marketed by Inco Medicals; Ambala. Three chest lead electrodes were applied- one to the right second intercostal space, one to the left second intercostal space and one to the left fifth intercostal space near the apex. The recordings were taken at controlled room temperature (22±10°C) in the research lab. For each subject all recordings were taken in one day with calm and quiet surroundings. The subjects were restricted from active movements and they rested in sitting position in between the recordings. HRV was recorded for a period of 5 minutes (300s) once before the subjects had meals (lunch) and the second recording was taken 15 minutes after the lunch. The composition of food in lunch was similar in all subjects in quantity and quality. HRV was recorded in each subject between 12.30 and 18.00 hours to minimize the circadian effects.

D. Heart rate variability analysis

Beat-to-beat variations in instantaneous HR were derived offline using a rate-detector algorithm. For computing HRV indices during supine rest and 15 minutes after the lunch, recommendations of the Task Force on HRV were followed. Briefly, a 5-min ECG was acquired at a sampling rate of 1000 Hz during supine rest and 15 minutes after the lunch, with the subjects breathing normally at 12–18 per min. RR intervals were plotted using the ECG V: 52 software. An RR series was extracted using a rate-detector algorithm after exclusion of artifacts and ectopics. A stationary 256 second RR series was chosen for analysis. Low frequency (LF) and high frequency (HF) spectral powers were determined by integrating the power spectrum between 0.04 and 0.15 Hz and 0.15 and 0.4 Hz respectively. Total power was calculated by integrating the spectrum between 0.004 and 0.4 Hz and includes very low frequency, LF and HF components. Spectral powers are expressed in absolute units of milliseconds squared, LF and HF powers are also expressed in normalized units as described.

E. The analysis of HRV

I) Frequency Domain analysis:

The frequency Domain components of HRV were analyzed by using Fast Fourier Transform method. The power spectrum so got is subsequently divided into bands of frequencies.

- TP ms² : Total power, variance of all NN intervals.
- LF ms² : Power in low frequency range.
- HF ms² : Power in high frequency range.
- LF nu : Low Frequency component, where nu means statistically normalized units. This mainly signify sympathetic component.
- HF nu : High Frequency component, where nu means statistically normalized units. This signify parasympathetic component.
- LF/HF : Ratio of Low Frequency component to High Frequency component, which signify the sympathovagal balance

F. Statistical methods applied

All the statistical methods were carried out through the SPSS for Windows (version 16.0) and Minitab (version 11.0). The Paired-Samples T Test procedure was done to compare the means of two variables for a single group. The Independent-Samples T Test procedure was done to compare the means for two groups of cases. P value less than or equal to 0.001 were considered statistically significant.

III. RESULTS

50 non-obese females and 50 obese females were analyzed for the results. The results obtained were expressed as mean ± standard deviation.
A. Physical characteristics of the subjects
On analysis of the physical characteristics of the 50 non-obese females, the mean age (years) was 19.46 ± 1.77; the mean weight (kg) was 53.54 ± 6.32; the mean height (m) was 1.584 ± 0.055, the mean BMI (kg/m²) was 21.27 ± 1.61. (Table: 1)

On analysis of the physical characteristics of the 50 obese females, the mean age (years) was 20.58 ± 1.97; the mean weight (kg) was 83.36 ± 5.93; the mean height (m) was 1.60 ± 0.06; the mean BMI (kg/m²) was 32.47 ± 1.87. (Table: 1)

B. Heart Rate Variability (Frequency Domain) Parameters
1) Comparison of pre prandial and post prandial TP (ms²) between non-obese and obese females.
In the pre testing, non-obese group had a mean TP value of 1659.9000, which has been decreased to 748.0400 in the post test which was found to be significant (P=0.005). Similarly, the obese group had a mean TP value of 1550.8600 which has been decreased to 1150.0000, again the decrease was found to be statistically significant (P=0.001). (Table: 2)

In the pre testing, a significant difference was observed (P=0.175) between non-obese (mean 1659.9000) and obese groups (mean 1550.8600). Also, in the post test, non-obese and obese (means 1476.3800 and 1150.0000 respectively) differed significantly (P=0.001). Further, repeated measure ANOVA revealed a significant (P=0.020) differential decrease for obese and non-obese groups, where obese group had higher rate of TP decrease compared to non-obese group. (Table: 2)

2) Comparison of pre prandial and post prandial LF (ms²) between non-obese and obese females.
In the pre testing, non-obese group had a mean LF value of 809.7800 which has been decreased to 665.8400 in the post test which was found to be significant (P=0.001). Similarly, the obese group had a mean LF value of 780.9600 which has been decreased to 665.8400, again the decrease was found to be statistically significant (P=0.001). (Table: 3)

In the pre testing, a significant difference was observed (P=0.050) between non-obese (mean 809.7800) and obese groups (mean 780.9600). Also, in the post test, non-obese and obese (means 748.0400 and 665.8400 respectively) differed significantly (P=0.001). Further, repeated measure ANOVA revealed a significant (P=0.021) differential decrease for obese and non-obese groups, where obese group had higher rate of LF decrease compared to non-obese group. (Table: 3)

3) Comparison of pre prandial and post prandial HF (ms²) between non-obese and obese females.
In the pre testing, non-obese group had a mean HF value of 884.5200, which has been decreased to 585.7600 in the post test which was found to be significant (P=0.001). Similarly, the obese group had a mean HF value of 850.9200 which has been decreased to 384.3600, again the decrease was found to be statistically significant (P=0.001). (Table: 4)

In the pre testing, a significant difference was observed (P=0.654) between non-obese (mean 884.5200) and obese groups (mean 850.9200). Also, in the post test, non-obese and obese (means 585.7600 and 384.3600 respectively) differed significantly (P=0.001). Further, repeated measure ANOVA revealed a significant (P=0.051) differential decrease for obese and non-obese groups, where obese group had higher rate of HF decrease compared to non-obese group. (Table: 4)

4) Comparison of pre prandial and post prandial LF (nu) between non-obese and obese females.
In the pre testing, non-obese group had a mean LFnu value of 40.4400, which has been increased to 53.6400 in the post test which was found to be significant (P=0.001). Similarly, the obese group had a mean LFnu value of 45.4000 which has been increased to 64.9200, again the increase was found to be statistically significant (P=0.001). (Table: 5)

In the pre testing, a significant difference was observed (P=0.003) between non-obese (mean 40.4400) and obese groups (mean 45.4000). Also, in the post test non-obese and obese (means 53.6400 and 64.9200 respectively) differed significantly (P=0.001). Further, repeated measure ANOVA revealed a significant (P=0.022) differential increase for obese and non-obese groups, where obese group had higher rate of LFnu increase compared to non-obese group. (Table: 5)

5) Comparison of pre prandial and post prandial HF (nu) between non-obese and obese females.
In the pre testing, non-obese group had a mean HF nu value of 39.9200, which has been decreased to 33.4800 in the post test which was found to be significant (P=0.001). Similarly, the obese group had a mean HF nu value of 33.5000 which has been decreased to 20.2200, again the decrease was found to be statistically significant (P=0.001). (Table: 6)

In the pre testing, a significant difference was observed (P=0.001) between non-obese (mean 39.9200) and obese groups (mean 33.5000). Also, in the post test non-obese and obese (means 33.4800 and 20.2200 respectively) differed significantly (P=0.001). Further, repeated measure ANOVA revealed a significant (P=0.001) differential decrease for obese and non-obese groups, where obese group had higher rate of HF nu decrease compared to non-obese group. (Table: 6)

6) Comparison of pre prandial and post prandial LF: HF ratio between non-obese and obese females.
In the pre testing, non-obese group had a mean LF: HF value of 0.9180, which has been increased to 1.4820 in the post test which was found to be significant (P=0.001). Similarly, the obese group had a mean LF: HF value of 1.4820 which has been increased to 2.1640, again the increase was found to be statistically significant (P=0.001). (Table: 7)

In the pre testing, a significant difference was observed (P=0.075) between non-obese (mean 0.9180) and obese groups (mean 1.4820). Also, in the post test, non-obese and obese (means 1.4820 and 2.1640 respectively) differed significantly (P=0.001). Further, repeated measure ANOVA revealed a significant (P=0.001) differential increase for obese and non-obese groups, where obese group had higher rate of LF: HF increase compared to non-obese group. (Table: 7).

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>MEAN</th>
<th>SD</th>
<th>T Value</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
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<tr>
<td>Non Obese</td>
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<td>19.46</td>
<td>1.77</td>
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<td>0.004</td>
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<tr>
<td>Obese</td>
<td>50</td>
<td>20.58</td>
<td>1.97</td>
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</table>
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Table 1: Comparison Of Pre Prandial And Post Prandial Lf (Ms²) Between Non Obese And Obese Females.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-obese</td>
<td>884.5200</td>
<td>349.75188</td>
<td>T=7.12</td>
<td>P=0.001</td>
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<tr>
<td>Obese</td>
<td>850.9200</td>
<td>397.06252</td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>867.7200</td>
<td>372.64398</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HF PRE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-obese</td>
<td>585.7600</td>
<td>285.49889</td>
<td>T=298.7</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Obese</td>
<td>384.3600</td>
<td>121.11182</td>
<td>F (Change)=8.130; P=0.001</td>
<td>F (change X groups)=3.91; P=0.051</td>
</tr>
<tr>
<td>Total</td>
<td>485.0600</td>
<td>240.51199</td>
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<td></td>
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</tbody>
</table>

Table 2: Comparison Of Pre Prandial And Post Prandial Lf (Nu) Between Non Obese And Obese Females.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-obese</td>
<td>40.4400</td>
<td>8.50248</td>
<td>T=6.354</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Obese</td>
<td>45.4000</td>
<td>7.65053</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>42.9200</td>
<td>8.42397</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Comparison Of Pre Prandial And Post Prandial Hf (Ms²) Between Non Obese And Obese Females.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-obese</td>
<td>40.4400</td>
<td>8.50248</td>
<td>T=6.354</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Obese</td>
<td>45.4000</td>
<td>7.65053</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>42.9200</td>
<td>8.42397</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4: Comparison Of Pre Prandial And Post Prandial Hf (Ms²) Between Non Obese And Obese Females.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-obese</td>
<td>53.6400</td>
<td>8.29251</td>
<td>T=13.2</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Obese</td>
<td>64.9200</td>
<td>9.42389</td>
<td>F (Change)=1 45.927; P=0.001</td>
<td>F (change X groups)=5.51 4;P=0.022</td>
</tr>
<tr>
<td>Total</td>
<td>59.2800</td>
<td>10.49394</td>
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<td></td>
</tr>
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</table>

Table 5: Comparison Of Pre Prandial And Post Prandial Lf (Nu) Between Non Obese And Obese Females.
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Table 6: Comparison of Pre Prandial and Post Prandial HF (Nu) Between Non Obese and Obese Females.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HF nu PRE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-obese</td>
<td>39.9200</td>
<td>5.87225</td>
<td>6.415</td>
<td>0.001</td>
</tr>
<tr>
<td>Obese</td>
<td>33.5000</td>
<td>3.94994</td>
<td></td>
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</tr>
<tr>
<td>Total</td>
<td>36.7100</td>
<td>5.93278</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>HF nu POST</strong></td>
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<td></td>
<td>16.291</td>
<td>0.001</td>
</tr>
<tr>
<td>Non-obese</td>
<td>33.4800</td>
<td>4.52314</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>20.2200</td>
<td>3.55878</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>26.8500</td>
<td>7.79714</td>
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</tr>
</tbody>
</table>

Table 7: Comparison of Pre Prandial and Post Prandial Lf:HF Ratio Between Non Obese and Obese Females.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LF: HF PRE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-obese</td>
<td>0.9180</td>
<td>0.38305</td>
<td>1.803</td>
<td>0.075</td>
</tr>
<tr>
<td>Obese</td>
<td>1.4820</td>
<td>0.35995</td>
<td></td>
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</tr>
<tr>
<td>Total</td>
<td>0.9850</td>
<td>0.37588</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>LF: HF POST</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Non-obese</td>
<td>1.4820</td>
<td>0.50616</td>
<td>7.902</td>
<td>0.001</td>
</tr>
<tr>
<td>Obese</td>
<td>2.1640</td>
<td>0.34092</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1.8230</td>
<td>0.54935</td>
<td></td>
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</tr>
</tbody>
</table>

IV. DISCUSSION

In the present study the following HRV (frequency domain analysis) parameters like TP (ms²), LF (ms²), HF (ms²), LF (nu), HF (nu) and LF/HF ratio were estimated in pre prandial and post prandial state between obese and non-obese young adult females.

The major findings of this study were that the obese group showed significant reduction (p=0.001) in TP, LF, HF, HF nu and showed a significant increase (p=0.001) in the values of LF nu and LF/HF in post prandial state when compared to non obese group. These finding indicate the presence of impaired parasympathetic activity in obese individuals. In addition, this study also indicated an elevated level of sympathetic activity in obese group. Thus, it showed a definite shift in the sympathovagal balance towards sympathetic component.

The cardiac autonomic activity can be assessed by several methods like valsalva maneuver, deep breathing test, handgrip test, cold pressor test, lying to standing test etc. But HRV has evolved as a specific and sensitive noninvasive tool to evaluate cardiac autonomic activity, which expresses the total amount of variation of both instantaneous heart rate and RR intervals. HRV indicates the extent of neuronal damage to autonomic nervous system. \[15\] The LF nu values are considered as a measure of sympathetic activity. \[15-19\] In this study the pre and post prandial LF nu values were significantly increased (p=0.001) in the obese group thus suggesting the presence of elevated cardiac sympathetic activity in obese individuals. \[31\]

The HF nu values are considered as a measure of parasympathetic activity. \[5-19\] In this study the pre and post prandial HF nu values were significantly reduced (p=0.001) in the obese individuals, which further adds on to the earlier finding of decreased cardiac parasympathetic activity. \[32\]

The LF/HF values reveal the global sympathovagal balance. \[15-19\] In this study the pre prandial and post prandial LF/HF values in the obese group was significantly higher (p=0.001) when compared to non obese group thus suggesting the alteration in the sympathovagal balance towards the sympathetic component in obese group. \[32\]

Similar findings were reported by multiple studies Laederach-Hofmann K et al, \[33\] Nagai N et al, \[34\] Muscelli E et al, \[35\] Hirsch J et al, \[36\] Porrier P et al. \[37\] Karason K et al, \[36\] Amano M et al, \[37\] Sekine M et al, \[37\] Rabbia F et al, \[28\] Gulzar JM et al, \[27\] Fu CC et al, \[28\] Kaufman CL et al. \[38\]

As far as the parasympathetic activity is considered there is not much difference among the studies, as almost all of the studies showed a significant reduction in the parasympathetic activity with increasing body weight. \[32\]

The mechanism underlying these changes of parasympathetic and sympathetic nervous activities in overweight is unknown. Several hormonal signals have been postulated. These include insulin, which has been shown in humans to increase muscle sympathetic nerve activity during euglycemic insulin clamp; free fatty acids, which have been shown to increase BP in rats by stimulation of excitatory hepatic afferent nerves; and leptin, the ob gene product, which has been shown to increase sympathetic discharge to several tissues in rats, and has been found elevated during rapid weight gain in humans. \[26\]

In contrast to the increase in the sympathetic activity found in the present study some studies have showed a significant reduction in the sympathetic activity. \[13\] This variation among the studies was partially explained...
on the basis of the duration of obesity. It has been said that duration of the obesity has a major role to play in determining the level of cardiac sympathetic activity. The present study demonstrated that the LF-to-HF ratio, the cardiovascular sympathovagal balance was increased after the meal. This finding supports the physiological phenomena, such as increased heart rate, blood pressure, and cardiac output, commonly observed after food ingestion. In one study, where spectral analysis of the HRV was applied to evaluate the postprandial sympathovagal balance, has shown an insignificant increase of the LF-to-HF ratio throughout the 30-min postprandial period. The content of the test meal might account for the difference in the results. In some study, a low calorie liquid diet (250 kcal) was used as the test meal, while we selected the solid meal of 500 kcal. Young et al. and Jansen and Hoe flagels, noted that a low-calorie meal would not produce any significant change in the heart rate or mean arterial blood pressure. Meals with high energy content (over 450 kcal), however, would cause a significant increase in the heart rate.

Traditionally, sympathetic tone was thought to be activated over the entire human body after ingestion of food. It is responsible for approximately 30% of the thermic effect of food and also helps maintain blood pressure postprandially. However, in the current study, we noticed that the increased LF to HF ratio was actually attributed to vagal withdrawal, instead of sympathetic activation in the heart.

Few studies attempted to use spectral analysis of the HRV to evaluate the sympathetic and parasympathetic function after a meal, and the results were conflicting. Kaneco et al. demonstrated a slight, but significant increase of HF amplitude (vagal tone) during the first 5 min after the test meal. However, the vagal activation disappeared thereafter. In contrast, Lipszt et al. showed an increased LF power, suggesting heightened sympathetic nervous control of the heart in healthy young volunteers after food intake. Furthermore, Vaz et al found a similar postprandial autonomic response as ours. They noted that the HF component was unchanged, while the LF component tended to decline postprandially, although not statistically significant. Sympathetic activity has been reported to be activated after food ingestion in humans and animals, which is evidenced by an increased postprandial plasma noradrenaline spillover response of whole-body, fore arm, kidney, and skeletal muscle. Increased postprandial sympathetic nervous activity, and increased postprandial muscle nerve activity measured by microneurography.

The sympathetic activity increases initially as one of the mechanism to oppose further weight gain especially in those individuals who have developed obesity recently. But if the obesity is of a longer duration, then it is likely to lead to reduction of the autonomic activity and hence a reduction in the sympathetic activity also. In one of the study, the recently obese individuals showed increase in the sympathetic component, which is a homeostatic mechanism to prevent weight increase, whereas the intermediate onset obesity and long standing obesity showed no significant difference in comparison to lean individuals. Further it showed a linear decrease in the sympathetic component in the obese individual with the duration of the obesity. In the present study, duration of obesity was not considered. As the subjects in the study were in the age group of 18-25 years, they probably belong to the recently onset obese.

This study showed significant inverse relationship between BMI and HRV parameters like TP, LF, HF, HF nu, but at the same it showed significant positive relation of BMI and LF nu, LF/HF. The present study shows that the derangement of the sympathovagal balance to sympathetic component is related to the amount of fat (BMI).

Thus this study showed reduced parasympathetic activity and increased sympathetic activity in obese females when compared to control group.

This sympathovagal imbalance can explain the increased incidence of sudden cardiac deaths associated with obesity. Thus early interventional programs like weight reduction, lifestyle changes and physical exercises, which reduce fat content of the individual, can be advised to reduce the chances of subsequent cardiac rhythm abnormalities.

From the above discussion, we come to the conclusion that vagal activity is the major contributor to the HF component. Disagreement exists in respect to the LF component some studies suggest that LF, when expressed in normalized units, it is a quantitative marker of sympathetic modulations; other studies view LF as reflecting both sympathetic activity and vagal activity. Consequently, the LF/HF ratio is considered by some investigators to mirror sympathovagal balance or to reflect the sympathetic modulations. It is important to note that HRV measures fluctuations in autonomic inputs to the heart rather than the mean level of autonomic inputs. Thus, both autonomic withdrawal and saturating high level of sympathetic input lead to diminished HRV.

Limitations of the present study are that the duration of obesity was not considered in this study, which could have helped in establishing the relation of duration & effects of obesity on cardiac autonomic activity. Serum levels of hormones like catecholamines, leptin etc. could have been considered for better understanding of relation of obesity and its effects on autonomic nervous system.

There is scope for further studies like study could be undertaken in various age groups and also in both genders for more clarity. Further, a prospective study can be undertaken in the same subjects to know the effect of weight loss on the cardiac autonomic activity.

V. CONCLUSION

In the present study group, obesity is accompanied with disturbances in the cardiac autonomic activity with sympathetic predominance. The following conclusions can be drawn from the results of the present study.

- Total power (TP) and Low frequency power (LF) expressed in absolute units (ms²) and High frequency power (HF) expressed in both absolute units (ms²) and in normalized units (nu) is decreased in obese females in pre and post prandial state when compared to non obese females.
indicating that there is a decrease in the cardiac parasympathetic activity in obese subjects.

- Low frequency power when expressed in normalized units (LFnu) is increased in obese females in pre and post prandial state when compared to non obese females indicating that there is an increase in the cardiac sympathetic activity in obese subjects.

- Low frequency to high frequency power (LF/HF) ratio is increased in obese females in pre and post prandial state when compared to non obese females indicating that there is a sympatho-vagal imbalance in obese subjects.

- Though our study is by no means exhaustive, it does provides a glimpse into the variety of adaptations / alterations in cardiac autonomic functions that occur as excessive adipose tissue accumulates, even in the absence of overt disease. Although we understand to some extent the pathophysiology of obesity, a number of scientific questions need to be addressed for us to have a more complete understanding of this condition. Further research is recommended to understand how genes and gene-environment interaction leads to obesity. A better understanding of ethnic / racial differences in the development and progression of obesity is needed. We need to evaluate the strategies, and efficacy of obesity treatment.

REFERENCES


