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# A COMPARATIVE STUDY OF PRE PRANDIAL AND POST PRANDIAL AUTONOMIC NERVOUS SYSTEM RESPONSE BETWEEN OBESE AND NON OBESE YOUNG WOMEN AGED 18-25 YEARS.

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## ABSTRACT

Background; In obesity as excessive 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 comorbidities.

Objectives; 1.To study ANS parameters in preprandial and postprandial state in obese and nonobese young healthy females aged 18-25 years.

2.To compare within the groups the ANS parameters in the preprandial and post prandial state.

METHODS;50 obese and 50 nonobese young healthy females aged 18-25 years were selected based on body mass index.Systolic blood pressure and diastolic blood pressure was recorded using digital electronic blood pressure monitor in both pre and post prandial state.Heart rate and heart rate variability was recorded in both pre and postprandial state using ECG V:52HRV power spectral analysis to identify separate frequency components, i.e,total power, low frequency power and high frequency power.

RESULTS: 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 ≤0.001 was considered statistically significant.HRV analysis found significantly lower values( p=0.001) of TP,LF,HF and HF(nu) ( p=0.001) of LF(nu) and LF/HF ratio, and also increased HR and BP ( 0.001) among the obese group in both pre and post prandial respectively compare to non obese group.

CONCLUSION: Our data indicate that obese subjects have decreased parasympathetic activity as evidenced by decrease in TP (ms2), LF (ms2), HF(ms2) and HF( nu) and increase in sympathetic activity as evidenced by increase in HR,BP,LF( nu) and LF/HF ratio in both pre and post prandial state.

**KEYWORDS:Autonomic nervous system, pre prandial and post prandial,heart rate variability,obese young women.**

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# INTRODUCTION

Obesity is an emerging global health problem. [1-4] Nutritional problem in India is gradually shifting from undernourishment to obesity. [5] It is a disease, which has evolved with the advent of civilization, sedentary lifestyle and high calorie diet. [6] Obesity is one of the causative factors for multiple co-morbid conditions leading to metabolic and cardiac disorders. [7] The incidence of overweight and obesity are increasing around the world especially in young adults and middle-aged people. [6]With obesity, there are increased chances of acquiring endocrinal diseases, genetic and metabolic disorders. [7]Obesity is one of the risk factors attributed for the development of lipid abnormalities, insulin resistance, hypertension etc. [2] Growing number of evidences indicate association of obesity and sudden cardiac deaths. [8-11] Imbalance in cardiac autonomic activity might be a predisposing factor for arrhythmogenesis and subsequently sudden cardiac deaths. [3-5, 7]Obesity is accompanied with varied combinations of abnormalities in the autonomic nervous system imbalance. [12] There is some difference of opinion on pattern of abnormal cardiac autonomic activity in obese humans. [13] One view is that obese people have a higher sympathetic tone, [3] that 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. [14]There are several tests to determine the autonomic activity. Recently the most accepted tool is determining Heart Rate Variability. [15]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 indicates 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. [16-19]The clinical relevance of HRV was first appreciated in 1965 when Hen and Lee noted that fetal distress was preceded by alterations in inter beat intervals before any appreciable change occurred in heart rate itself. The last two decades have witnessed the recognition of a significant relationship between the ANS and cardiovascular mortality, including sudden cardiac death. Experimental evidence for an association between propensity for lethal arrhythmias and signs of either increased sympathetic or reduced vagal activity has spurred efforts for the development of quantitative markers of autonomic activity. HRV represents one of the most promising such markers. The apparently easy derivation of this measure has popularized its use. HRV refers to the beat–to–beat alterations in HR. [23]This study is an effort to assess the effect of obesity on cardiac autonomic activity using Heart Rate Variability in young females.

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. [20] 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. [21]Food intake causes

peptides to be released in the GIT, which leads to vasodilatation locally. This leads to redistribution of blood ie, more blood being supplied to GIT. [22]

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.

## MATERIALS AND METHODS

Source of data: This is a comparative study having 50 non obese and 50 obese females with 100 healthy female subjects and was conducted in the Research lab, Department of Physiology, Navodaya Medical College, Raichur. Ethical clearance: Ethical clearance was obtained from 'Navodaya Medical College Ethical Committee for Research' to conduct the study. Method of collection of data:Subjects were examined for their general physical health. Subject's clinical history and details were taken according to the standard proforma. Young health females of age 18-25 years were recruited for the study. Informed written consent was taken from all subjects in the study.

The BMI was calculated as

$$BMI = \frac{\text{Weight in kg}}{(\text{Height in meters})^2}$$

***Then they were categorized as below***

***Categorization of body mass index : [24,25]***

| Category | BMI ($kg/m^2$) |
|---|---|
| Underweight | <18.5 |
| Normal weight | 18.5-24.9 |
| Over weight | 25-29.9 |
| Obesity (class 1) | 30-34.9 |
| Obesity (class2) | 35-39.9 |
| Extreme obesity (class 3) | >40 |

***Inclusion criteria***

- Young obese females (n=50) in the age group of 18-25 years.
- Young non obese females (n=50) in the age group of 18-25 years
- Subjects selected for study were on $10^{th}$ - $12^{th}$ day of their menstrual cycle.

***Exclusion criteria***

- Age below 18 years and above 25 years.
- Subjects with history of Asthma, diabetes mellitus, hypertension, other cardiovascular diseases, endocrine disease or surgery.
- Subjects on chronic medication.
- Subjects with noticeable weight gain or weight loss over the preceding 3 months.
- Subjects having any neuro-muscular disorders.
- Subjects on any drugs affecting the functioning of Autonomic Nervous System-adrenergic blockers, calcium channel blockers, anxiolytics, anesthetics, narcotics, chemotherapeutics were also excluded.

***Assessment of Body mass index: [24,25]***

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.

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

Normal weight – BMI – 18.5 – 24.99 kg/$m^2$
Obese – BMI > 30 kg/$m^2$

***Investigations and interventions conducted***

This study involved non invasive procedures. Procedures were conducted in the presence of another person who was either the teaching/non technical staff of the department of physiology, Navodaya Medical College and Research Centre, Raichur. Following tests were performed in supine position 2-3 hours after a light breakfast in sequence after familiarizing the subjects with the testing procedures.

***Autonomic Function Tests : [26]***

1. Heart Rate (HR)
2. Systolic Blood Pressure (SBP)
3. Diastolic Blood Pressure (DBP)
4. Heart rate variability (HRV)

***Instruments***

- Digital Physiograph
- ECG V: 52 [HRV analysis software]- was used in the computer to detect the peak to peak intervals and further mathematical and analytical calculations was done in order to get the values of the parameter
- Digital electronic blood pressure monitor
- Weighing Machine

***ECG V: 52 (Heart Rate Variability analysis software)***

Manufactured by NIVIQURE Meditech pvt. Ltd. Bengaluru and marketed by Inco Medicals; Ambala. Manufactured year-2006; computerized software for ambulatory ECG recording. Recordings were standardized and instructions followed as per the guidelines of Task Force of the European Society of Cardiology as HRV, standards of measurement, physiological interpretation and clinical use. The software has an inbuilt analysis and interpretation of five minute uninterrupted recording of standard Lead II recording of ECG in supine resting eyes closed relaxed state.

***Digital Electronic Blood Pressure Monitor***

Manufactured by Kawamoto Corporation, Osaka – Japan. Model No: KBM-125
Specifications of the instrument are as follows:

***Measurement range***

Cuff pressure: 0 to 300mm of Hg Pulse: 30 to 220pulse/min

***Accuracy***

Cuff pressure: ±3mmHg Pulse: ±5% of reading
Operating temperature: +10$^0$C to +40$^0$C
Humidity: 85%Rh or below
Power supply: 9 Volt battery
Exhaust Valve: Manual
Cuff: Standard Adult size (cover range arm circumference 23 to 32cm)

***PROCEDURE***

Each of the subjects was allowed to have breakfast in the morning at 8.30 AM. No dietary restrictions were imposed before the study. The procedure was explained to each subject before the rest. The HRV was recorded with subject in supine position using Niviqure Software. 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. A non reactive electrode gel was applied to the skin where electrodes were placed for better conduction. The recordings were taken at controlled room temperature (22±1$^0$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) in both the recordings. HR, SBP, DBP and HRV were recorded 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. The timings confirmed to their normal lunch timings.

***Heart Rate***

Prior to the test the subject was made to lie down in supine position on a couch. ECG leads were connected using electrodes from the subject to the ECG V: 52. The resting heart rate was recorded on a computerized ECG from lead two, at a speed of 30mm/sec. The second reading was taken 15 minutes after the lunch.

***Systolic Blood Pressure (SBP) & Diastolic Blood Pressure (DBP)***

BP was measured with digital electronic blood pressure monitor in supine position after a period of rest for 5 minutes. The second reading was taken 15 minutes after the lunch.

***Heart rate variability analysis***

A chest lead ECG was recorded throughout supine rest using the ECG V: 52 system (Niviqure Meditech pvt. Ltd). 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. [27] 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. 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.

***The analysis of HRV : [15]***

***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 $ms^2$ : Total power, variance of all NN intervals.
LF $ms^2$ : Power in low frequency range.
HF $ms^2$ : 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.

***Statistical methods applied***

***Descriptives***

The Descriptive procedure displays univariate summary statistics for several variables in a single table and calculates standardized values (z scores). Variables can be ordered by the size of their means (in ascending or descending order), alphabetically, or by the order in which the variables (the default) are selected.

***Paired-Samples T Test***

The Paired-Samples T Test procedure compares the means of two variables for a single group. It computes the differences between values of the two variables for each case and tests whether the average differs from 0.

***Independent-Samples T Test***

The Independent-Samples T Test procedure compares means for two groups of cases. Ideally, for this test, the subjects should be randomly assigned to two groups, so that any difference in response is due to the treatment (or lack of treatment) and not to other factors.

***GLM Repeated Measures Define Factors***

GLM Repeated Measures analyzes groups of related dependent variables that represent different measurements of the same attribute. This dialog box defines one or more within-subjects factors for use in GLM Repeated

Measures. The order in which the within-subjects factors is specified is important. Each factor constitutes a level within the previous factor. All the statistical methods were carried out through the SPSS for Windows (version 16.0) and Minitab (version 11.0)

***Presentation of data***

Master charts (Annexure IV) showing the indices of autonomic functions and heart rate variability parameters were tabulated separately for non obese and obese females. 50 non obese females and 50 obese females were analyzed for the results. The results obtained were expressed as mean ± standard deviation.

***Statistical evaluation***

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.

***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/$m^2$) 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 /$m^2$) was 32.47 ± 1.87. (Table: 1)

**TABLE 1**

***Physical characteristics of the subjects***

| | Group | N | MEAN | SD | T value | P value |
|---|---|---|---|---|---|---|
| AGE | Non obese<br>Obese | 50<br>50 | 19.46<br>20.58 | 1.77<br>1.97 | 2.986 | 0.004 |
| WEIGHT | Non obese<br>Obese | 50<br>50 | 53.54<br>83.36 | 6.32<br>5.93 | 24.312 | 0.001 |
| HEIGHT | Non obese<br>Obese | 50<br>50 | 1.58<br>1.60 | 0.05<br>0.06 | 1.562 | 0.122 |
| BMI | Non obese<br>Obese | 50<br>50 | 21.27<br>32.47 | 1.61<br>1.87 | 32.034 | 0.001 |

***Autonomic function test parameters***

***Comparison of pre prandial and post prandial HR between non obese and obese females.***

In the pre testing, non-obese group had a mean HR value of 83.20, which has been increased to 90.76 in the post test, which was found to be significant (P=0.001). Similarly, the obese group had a mean HR value of 86.64 which has been increased to 96.60, again the increase was found to be statistically significant (P=0.001). (Table: 2, Graph:1) In the pre testing, a significant difference was observed (P=0.001) between non-obese (mean 83.20) and obese groups (mean 86.64). Also, in the post test, non-obese and obese groups (means 90.76 and 96.60 respectively) differed significantly (P=0.001). Further repeated measure ANOVA revealed a significant (P=0.026) differential increase for obese and non-obese groups, where the obese group had higher rate of HR increase compared to non-obese group. (Table: 2, Graph:1)

***Comparison of pre prandial and post prandial SBP between non obese and obese females.***

In the pre testing, non-obese group had a mean SBP value of 111.2600, which has been increased to 123.8400 in the post test which was found to be significant (P=0.001). Similarly, the obese group had a mean SBP value of

123.8400 which has been increased to 140.8400, again the increase was found to be statistically significant (P=0.001). (Table: 3, Graph: 2) In the pre testing, a significant difference was observed (P=0.001) between non-obese (mean111.2600) and obese groups (mean123.8400). Also, in the post test, non-obese and obese groups (means 123.8400 and 140.8400 respectively) differed significantly (P=0.001). Further repeated measure ANOVA revealed a significant (P=0.001) differential increase for obese and non-obese groups, wherethe obese group had higher rate of SBP increase compared to non-obese group. (Table: 3, Graph: 2)

***Comparison of pre prandial and post prandial DBP between non obese and obese females.***

In the pre testing, non-obese group had a mean DBP value of 77.7600, which has been decreased to 73.6000 in the post test which was found to be significant (P=0.001). However, the obese group had a mean DBP value of 78.3600 which has been increased to 80.7200, again the increase was found to be statistically significant (P=0.003). (Table: 4, Graph: 3) In the pre testing, a non-significant difference was observed (P=0.463) between non-obese (mean 77.7600) and obese groups (mean 78.3600). However, in the post test non-obese and obese groups (means 73.6000 and 80.7200 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 DBP increase compared to non-obese group. (Table: 4, Graph: 3)

***Heart Rate Variability (Frequency Domain) Parameters***

***Comparison of pre prandial and post prandial TP ($ms^2$) 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 1476.3800 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: 5, Graph: 4). 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: 5, Graph: 4).

***Comparison of pre prandial and post prandial LF ($ms^2$) 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 748.0400 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: 6, Graph: 5). 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: 6, Graph: 5).

***Comparison of pre prandial and post prandial HF ($ms^2$) 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:7 , Graph: 6). In the pre testing, a significant difference was observed (P=0.654) between non-obese (mean884.5200) 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: 7, Graph: 6).

***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 be statistically significant (P=0.001). (Table: 8, Graph: 7). 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 (means53.6400 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 the obese group had higher rate of LFnu increase compared to non-obese group. (Table: 8, Graph: 7).

***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: 9, Graph: 8). In the pre testing, a significant difference was observed (P=0.001) between non-obese (mean39.9200) and obese groups (mean33.5000). 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: 9, Graph: 8).

***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: 10, Graph: 9). 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: 10, Graph: 9).

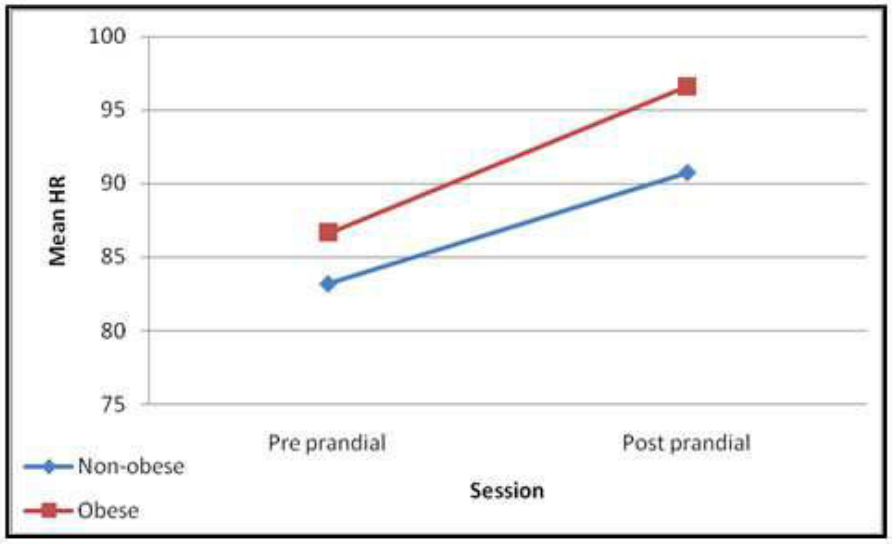
## TABLE 2

***Comparison of pre prandial and post prandial HR between non obese and obese females.***

| | Group | Mean | S.D | T value | P value |
|---|---|---|---|---|---|
| HR PRE | Non-obese | 83.20 | 4.06 | 4.320 | 0.001 |
| | Obese | 86.64 | 3.90 | | |
| | Total | 84.92 | 4.32 | | |
| HR POST | Non-obese | 90.76 | 3.15 | 3.613 | 0.001 |
| | Obese | 96.60 | 4.39 | | |
| | Total | 93.68 | 4.80 | | |
| Paired t value & P (NO) | T=12.612; P=0.001 | | | F(Change) =273.84; P=0.001 | F (change X groups) =5.139; P=0.026) |
| Paired t value & P (O) | T=5.45; P=0.001 | | | | |

## GRAPH 1

***Comparison of pre prandial and post prandial HR between non obese and obese females.***



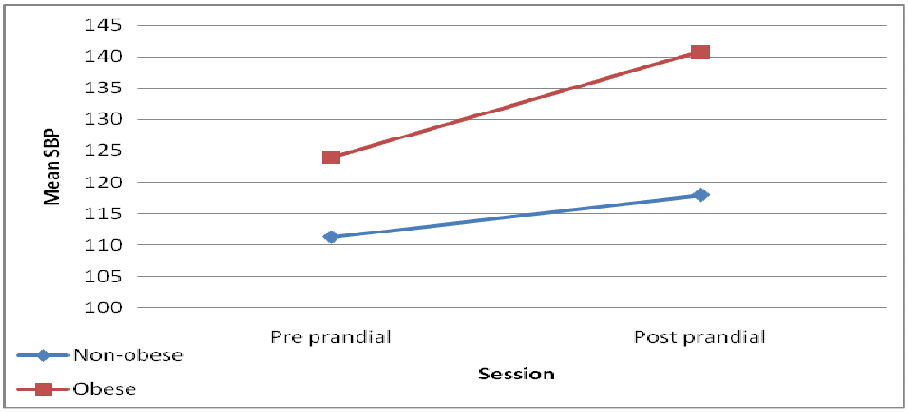

## TABLE 3

***Comparison of pre prandial and post prandial SBP between non obese and obese females.***

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| SBP PPRE | Non-obese | 111.2600 | 4.86894 | 13.447 | 0.001 |
| | Obese | 123.8400 | 4.47834 | | |
| | Total | 117.5500 | 7.85008 | | |
| SBP PPOST | Non-obese | 123.8400 | 4.61126 | 29.511 | 0.001 |
| | Obese | 140.8400 | 2.96483 | | |
| | Total | 129.4000 | 12.12727 | | |
| Paired t value & P (NO) | T=6.70; P=0.001 | | | F (Change) =667.867; P=0.001 | F (change X groups)=126.14;P=0.001) |
| Paired t value & P (O) | T=22.299; P=0.001 | | | | |

## GRAPH 2

***Comparison of pre prandial and post prandial SBP between non obese and obese females.***



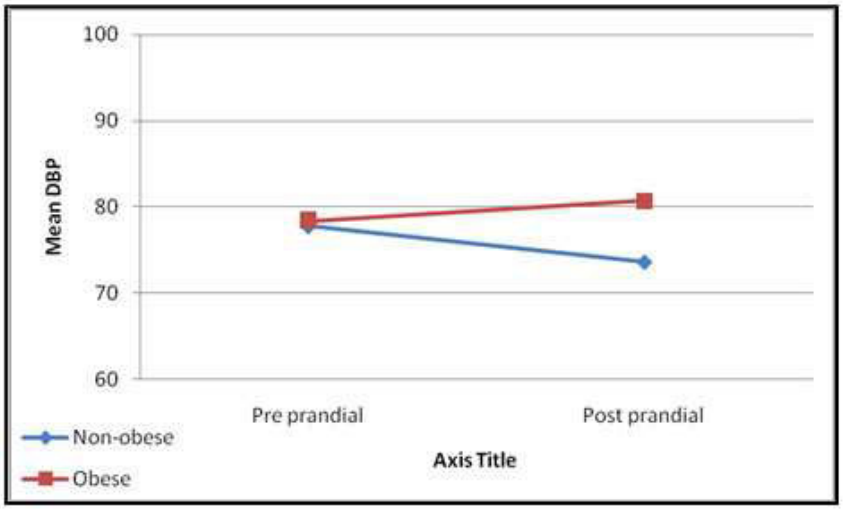

## TABLE 4

***Comparison of pre prandial and post prandial DBP between non obese and obese females.***

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| DBP PRE | Non-obese | 77.7600 | 3.62846 | 0.736 | 0.463 |
| | Obese | 78.3600 | 4.47560 | | |
| | Total | 78.0600 | 4.06468 | | |
| DBP POST | Non-obese | 73.6000 | 3.28261 | 12.352 | 0.001 |
| | Obese | 80.7200 | 2.41627 | | |
| | Total | 77.1600 | 4.58526 | | |
| Paired t value & P (NO) | T=4.16; P=0.001 | | F (Change)=4.074; P=0.046 | F (change X groups) =53.459; P=0.001) | |
| Paired t value & P (O) | T=3.134; P=0.003 | | | | |

## GRAPH 3

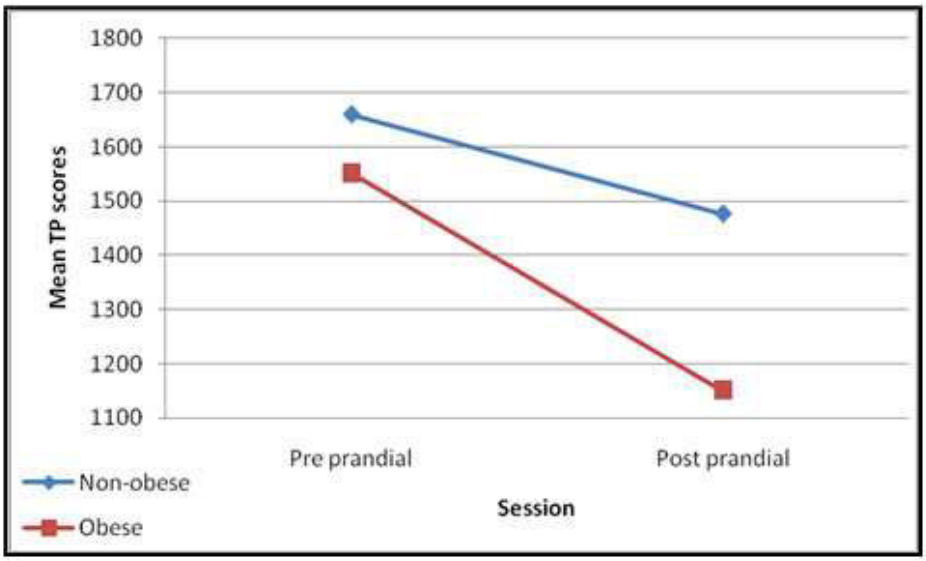
***Comparison of pre prandial and post prandial DBP between non obese and obese females.***



## TABLE 5
### *Comparison of pre prandial and post prandial TP ($ms^2$) between non obese and obese females.*

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| TP PRE | Non-obese | 1659.9000 | 374.94953 | 1.367 | 0.175 |
| | Obese | 1550.8600 | 421.20367 | | |
| | Total | 1605.3800 | 400.49499 | | |
| TP POST | Non-obese | 1476.3800 | 316.34345 | 6.615 | 0.001 |
| | Obese | 1150.0000 | 147.06503 | | |
| | Total | 1313.1900 | 295.18803 | | |
| Paired t value & P (NO) | T=183.52; P=0.005 | | | F(Change)=40.729; P=0.001 | F (change X groups)=5.634; P=0.020) |
| Paired t value & P (O) | T=5.990; P=0.001 | | | | |

## GRAPH 4
### *Comparison of pre prandial and post prandial TP ($ms^2$) between non obese and obese females.*

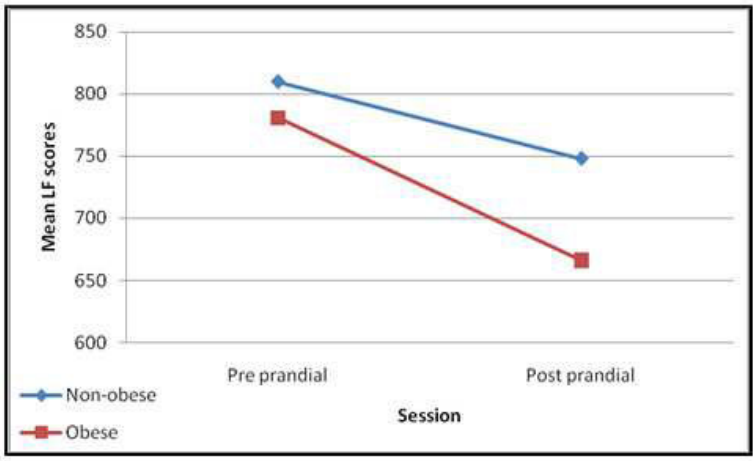



## TABLE 6
### *Comparison of pre prandial and post prandial LF ($ms^2$) between non obese and obese females.*

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| LFPRE | Non-obese | 809.7800 | 74.95013 | 1.982 | 0.050 |
| | Obese | 780.9600 | 70.39827 | | |
| | Total | 795.3700 | 73.77712 | | |
| LFPOST | Non-obese | 748.0400 | 70.67516 | 5.673 | 0.001 |
| | Obese | 665.8400 | 74.16814 | | |
| | Total | 706.9400 | 83.07351 | | |
| Paired t value & P (NO) | T=61.74; P=0.001 | | | F(Change)= 60.545;P=0.001 | F (change X groups)=5.515;P=0.021) |
| Paired t value & P (O) | T=7.124; P=0.001 | | | | |

## GRAPH 5

***Comparison of pre prandial and post prandial LF ($ms^2$) between non obese and obese females.***



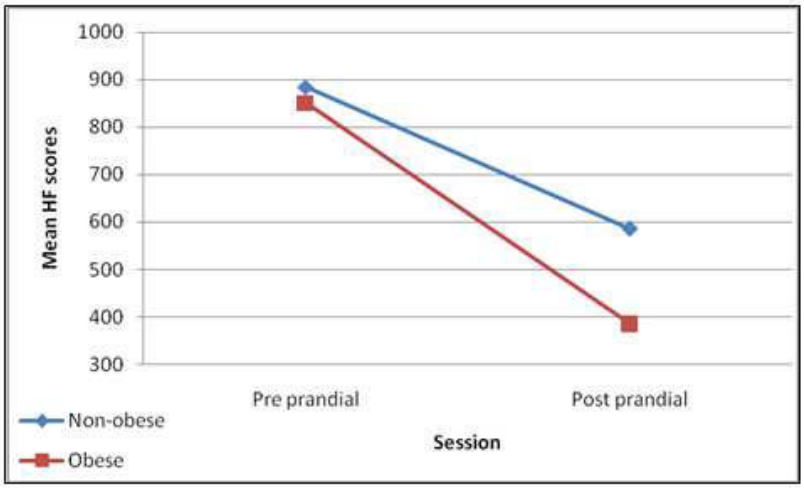

## TABLE 7

***Comparison of pre prandial and post prandial HF ($ms^2$) between non obese and obese females.***

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| HF PRE | Non-obese | 884.5200 | 349.75188 | 0.449 | 0.654 |
| | Obese | 850.9200 | 397.06252 | | |
| | Total | 867.7200 | 372.64398 | | |
| HF POST | Non-obese | 585.7600 | 285.49889 | 4.592 | 0.001 |
| | Obese | 384.3600 | 121.11182 | | |
| | Total | 485.0600 | 240.51199 | | |
| Paired t value & P (NO) | T=298.76; P=0.001 | | | F (Change)=81.30; P=0.001 | F (change X groups)=3.91;P=0.051) |
| Paired t value & P (O) | T=7.463; P=0.001 | | | | |

## GRAPH 6

***Comparison of pre prandial and post prandial HF ($ms^2$)between non obese and obese females.***



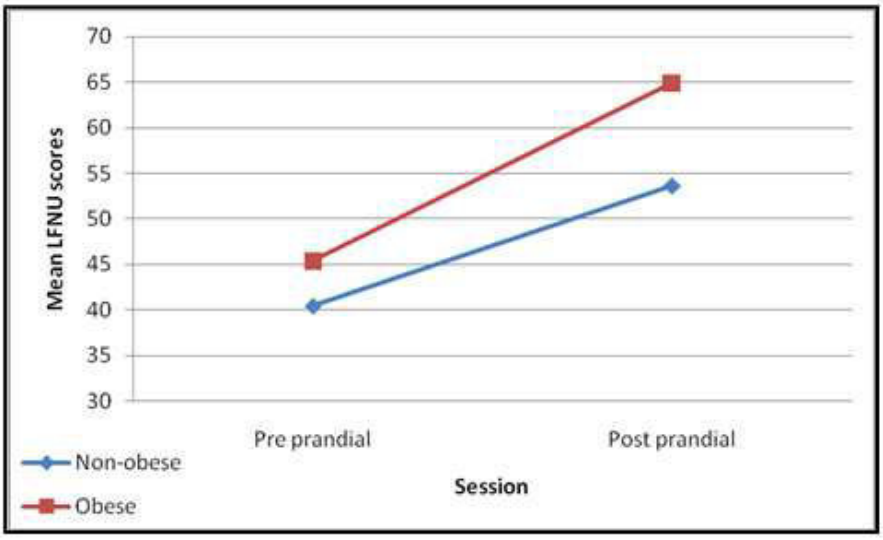
## TABLE 8

***Comparison of pre prandial and post prandial LF (nu) between non obese and obese females.***

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| LF nu PRE | Non-obese | 40.4400 | 8.50248 | 3.066 | 0.003 |
| | Obese | 45.4000 | 7.65053 | | |
| | Total | 42.9200 | 8.42397 | | |
| LF nu POST | Non-obese | 53.6400 | 8.29251 | 6.354 | 0.001 |
| | Obese | 64.9200 | 9.42389 | | |
| | Total | 59.2800 | 10.49394 | | |
| Paired t value & P (NO) | T=13.20; P=0.001 | | | F (Change)=145.927; P=0.001 | F (change X groups)=5.444;P=0.022) |
| Paired t value & P (O) | T=10.427; P=0.001 | | | | |

## GRAPH 7

***Comparison of pre prandial and post prandial LF (nu) between non obese and obese females.***




## TABLE 9

***Comparison of pre prandial and post prandial HF (nu) between non obese and obese females.***

| | Group | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| HF nu PRE | Non-obese | 39.9200 | 5.87225 | 6.415 | 0.001 |
| | Obese | 33.5000 | 3.94994 | | |
| | Total | 36.7100 | 5.93278 | | |
| HF nu POST | Non-obese | 33.4800 | 4.52314 | 16.291 | 0.001 |
| | Obese | 20.2200 | 3.55878 | | |
| | Total | 26.8500 | 7.79714 | | |
| Paired t value & P (NO) | T=6.44; P=0.001 | | | F (Change) = 225.322; P=0.001 | F (change X groups)=27.108;P=0.001) |
| Paired t value & P (O) | T=16.849; P=0.001 | | | | |

**GRAPH 8**

***Comparison of pre prandial and post prandial HF (nu) between non obese and obese females.***

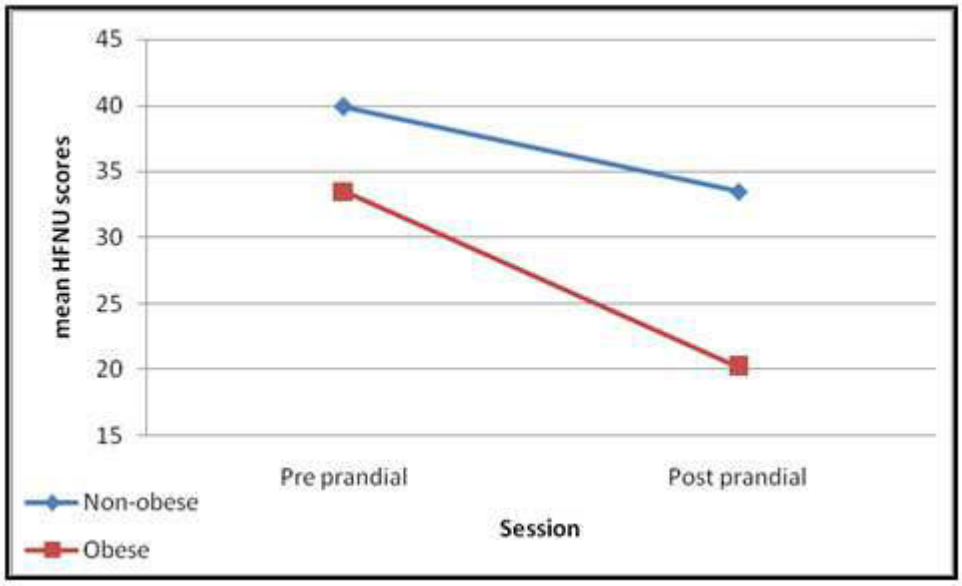



**TABLE 10**

***Comparison of pre prandial and post prandial LF:HF ratio between non obese and obese females.***

| | GROUP | Mean | SD | T value | P value |
|---|---|---|---|---|---|
| LF:HF PRE | Non-obese | 0.9180 | 0.38305 | 1.803 | 0.075 |
| | Obese | 1.4820 | 0.35995 | | |
| | Total | 0.9850 | 0.37588 | | |
| LF:HF POST | Non-obese | 1.4820 | 0.50616 | 7.902 | 0.001 |
| | Obese | 2.1640 | 0.34092 | | |
| | Total | 1.8230 | 0.54935 | | |
| Paired t value & P (NO) | T=0.564; P=0.001 | | F (Change)=235.394; P=0.001 | | F (change X groups)=25.166;P=0.001) |
| Paired t value & P (O) | T=15.587; P=0.001 | | | | |

# DISCUSSION

The main goal of the study was to detect changes of autonomic cardiovascular regulation in healthy obese young females in the age group 18-25 years and to compare the results with the healthy non-obese controls. The differences in the mean values of each parameter between obese and non obese females were analyzed and discussed. In the present study the following autonomic function test parameters like HR, SBP, DBP,HRV (frequency domain analysis) parameters like TP ($ms^2$), LF ($ms^2$), HF ($ms^2$), 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 HR, SBP, DBP, 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. [28] The HF nu values are considered as a measure of parasympathetic activity. [15-19] 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. [29]

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. [29] Similar findings were reported by multiple studies Laederach-Hofmann K et al, [30] Nagai N et al, [31] Muscelli E et al, [32] ,Poirier P et al , [33] Karason K et al, [34] Amano M et al, [35] Sekine M et al, [36] Rabbia F et al, [37] ,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. [29] 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. [39]

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. [14] It has been said that duration of the obesity has a major role to play in determining the level of cardiac sympathetic activity. [13, 14] Spectral analysis of the pre and postprandial HRV has been demonstrated as a simple, sensitive and noninvasive test to evaluate both the sympathetic and parasympathetic function as well as the balance between the two at the same time. While this noninvasive method assesses cardiac autonomic function, animal studies are required to investigate the relationship between cardiac vagal activity and gastric vagal activity. Once this relationship is known, noninvasive spectral analysis method of HRV can be applied to investigate gastric vagal activity and its association with gastrointestinal disorders.

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. [40] 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. [41] and Jansen and Hoe fnagels, [42] 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. [43,44]

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 [45,46] and also helps maintain blood pressure postprandially. [47] 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 [41] 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, Lipsitz et al [48] 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. [49] They noted that the LF component was unchanged, while the HF component tended to decline postprandially, although not statistically significant. The content of the test meal, the timing of measurement, the method in HRV measurement (with or without spectral analysis ) and analysis method in defining LF and HF (relative value or absolute value ) may be responsible for these. 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; [49,50] increased postprandial renal sympathetic nervous activity[51] and increased postprandial muscle nerve activity measured by microneurography. Many patients with gastrointestinal motility diseases, such as irritable bowel syndrome, functional dyspepsia, chronic pseudo obstruction, and chronic constipation, have been reported to have autonomic dysfunction. [52-55]

Number of evidences indicate that incidence of sudden cardiac deaths are more common in obese individuals. Also it has been shown that sympathovagal imbalance is one of the main predisposing factors for arrhythmogenesis. The negative relation of the protective parasympathetic activity and the positive relation of the sympathetic activity could very much predispose obese subjects to cardiac rhythm disorders. [56] 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 a reduction of the autonomic activity and hence a reduction in the sympathetic activity also. [13, 14] In one of the study, the recently obese individuals showed an 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. [14] 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 a 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 side and substantial reduction in parasympathetic 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, life style changes and physical exercises, which reduce fat content of the individual, can be advised to reduce the chances of subsequent cardiac rhythm abnormalities.

Adolescents and young adults with impaired autonomic responses to foods may be at high risk for obesity and stress-related diseases. Therefore, we tested the hypothesis that measurement of sympathovagal balance, performed in adolescent and young adult subjects before and after ingesting foods, may be indicators of their proportion of body fat and, therefore, risk for obesity and stress-related diseases. The RR interval variations present during resting conditions represent a fine tuning of the beat to beat control mechanisms. Vagal afferent stimulation leads to reflex excitation of vagal efferent activity and inhibition of sympathetic efferent activity.

The opposite reflex effects are mediated by the stimulation of sympathetic afferent activity. Efferent vagal activity also appears to be under “tonic” restraint by cardiac afferent sympathetic activity. Efferent sympathetic and vagal activities directed to the sinus node are characterized by discharge largely synchronous with each cardiac cycle that can be modulated by central (vasomotor and respiratory centers) and peripheral (oscillation in arterial pressure and respiratory movements) oscillators. These oscillators generate rhythmic fluctuations in efferent neural discharge that manifest as short and long term oscillations in the heart. Analysis of these rhythms may permit inferences on the state and function of (a) the central oscillators, (b) the sympathetic and vagal efferent activity, (c) humoral factors, and (d) the sinus node. 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 saturatingly high level of sympathetic input lead to diminished HRV. [23] 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.

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