*International Journal of Biomedicine 1(3) (2011) 128-131*

INTERNATIONAL JOURNAL OF BIOMEDICINE

Clinical Research

# A Prospective Observational Study of the Correlation between Obesity and the Autonomic Nervous System

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## Abstract

**Background:** As the ANS is involved in energy metabolism and the regulation of the cardiovascular system, people with idiopathic obesity can be considered to suffer an alteration in their autonomic nervous system. **Aim:** To correlate the BMI with cardiovascular autonomic functions. **Objectives:** To correlate three different sample groups of BMI in relation to their Cardiovascular Sympathetic and Parasympathetic functions. **Study design:** This was a prospective observational study. **Type of data:** This was the Primary Data. **Population:** Data has been collected from Pravara Rural Hospital Loni, Maharashtra, India. **Sample size:** 69 completely healthy male subjects (not having any major illness or chronic addiction) were selected for the study. All the subjects were evaluated using «CANWIN» cardiac autonomic neuropathy analyzer; windows based cardiac autonomic neuropathy analysis system with interpretation. **Statistical analysis:** Descriptive statistics was done and presented using tables and graphs, including mean values for continuous data to discuss the results. Correlations of outcome parameters were calculated with significance test. Statistical software SPSS, version16.0 was used for analysis. **Results:** Orthostatic hypotension and Handgrip test (increase in DBP) were more pronounced in Group III. Valsalva ratio test showed very weak negative correlation in group I and very weak positive correlation in Group II and III. **Conclusion:** Increased sympathetic activity and to some degree decreased Parasympathetic activity were found in correlation with the obesity. The study concluded that a more marked influence of obesity on activation of the sympathetic nervous system was observed. IJBM 2011; 1(3):128—131. 
***Key words:*** *autonomic, cardiovascular, obesity, parasympathetic, sympathetic.*

## Introduction

Although there is a wide fluctuation regarding calorie intake and the physical activity level of an individual, body weight has been observed to remain nearly constant, in animals as well as in humans. This suggests the presence of an integrative control mechanism that balances the energy expenditure and food intake [1-3].

A regulatory system that maintains constant energy storage is likely to involve complex interactions among humoral, neural, metabolic, and psychological factors; therefore, the autonomic nervous system (ANS) has been suggested as being the center, which coordinates this system [1-3]. Several experimental observations lend support to this viewpoint. For example, experimentally induced ventromedial hypothalamic lesions have been found to produce a combination of decreased sympathetic activity, increased parasympathetic activity and obesity [4].

Parasympathetic blockade using pharmacological means has been shown to increase thermogenesis induced by food intake; therefore, a relationship between parasympathetic activity and total energy storage in humans has been observed. An exogenously administered sympathetic agonist has been shown to increase caloric expenditure [1]. As the ANS is involved in energy metabolism and the cardiovascular system regulation [5-7], people with idiopathic obesity can be considered to have an alteration in their autonomic nervous systems that could promote obesity, ultimately leading to several clinical consequences of obesity, such as sudden death, hypertension or other cardiovascular abnormalities.

Therefore, ANS will be more carefully studied in future studies on the causes and the results of human obesity.

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# Material and methods

This study was carried out in the Department of Physiology, Rural Medical College Loni, Maharashtra, India, from Feb. 2009 to Dec. 2009.

***Inclusion criteria***

Totally, 69 healthy volunteer subjects (not having any major illness or chronic addiction) were selected for the study from among those visiting the OPD of Pravara Rural Hospital, Loni, Maharashtra, India. Based in the BMI they were divided into three groups, as follows: Group I – 22 males with BMI <25, Group II – 23 males with BMI 25 – 30 and Group III – 24 males with BMI >30.

***Obesity***

Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health [8].

Weight in kilograms and height in centimeters were measured and recorded for all the subjects to calculate the body mass index (BMI) as follows:

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

A person with a BMI of 30 or more is generally considered obese. A person with a BMI equal to or more than 25 is considered overweight [9].

***Exclusion criteria***

Subjects suffering from any major illness like diabetes mellitus, cardiac failure, cardiac arrhythmias and chronic obstructive lung disease or with any chronic addiction were excluded from the study.

Data comprising the clinical history, including name, age, sex, occupation were obtained from all the subjects and recorded. Special importance was paid in the clinical history to any symptoms suggestive of autonomic neuropathy.

The following tests were performed on all the subjects for evaluation of the Autonomic nervous system, by using the computerized device named «CANWIN» Cardiac Autonomic Neuropathy Analyzer Genesis medical system Pvt. Ltd., Hyderabad. The need for manual recordings, readings and calculation was thus eliminated. Inbuilt time domain waveform analysis and BP measurements made the task of conducting all the following six ANS tests very easy.

For parasympathetic functions:

- Resting heart rate / min
- Heart rate response to deep breathing (E: I ratio)
- Immediate heart rate response to standing (30:15 ratio)
- Valsalva ratio.

For sympathetic functions:

- Orthostatic variation in BP.
- Isometric handgrip test.

***Statistical analysis***

Descriptive statistics was performed and presented as tables and graphs, including mean values for continuous data to discuss the results more easily. Correlations of outcome parameters were calculated using the significance test. Statistical software SPSS, version 16.0 was used for analysis.

# Results

A total of 69 volunteer males were included in the present study.

The statistical analysis for sympathetic as well as parasympathetic tests was carried out separately on each subject. After analysis, the cardiovascular autonomic functions were correlated with their BMI (Tab. 1, 2).

***Table 1.***
*ANOVA for parasympathetic and sympathetic tests.*

| BMI | Parasympathetic tests | | | | Sympathetic tests | |
|---|---|---|---|---|---|---|
| | **Resting HR** | **E: I ratio** | **30:15 ratio** | **Valsalva ratio** | **Orthostatic hypotension** | **Handgrip test** |
| <25 (n=22) | 73.55±6.65 | 1.17±0.127 | 1.72±2.89 | 1.55±0.396 | 1.41±8.96 | 10.27±11.69 |
| *p-value* | *p>0.05* | *p>0.05* | *p>0.05* | *p<0.05* | *p<0.05* | *p<0.05* |
| 25-30 (n=23) | 74.43±7.79 | 1.20±0.124 | 1.033±0.250 | 4.44±9.04 | 5.17±6.62 | 9.04±7.81 |
| *p-value* | *p>0.05* | *p>0.05* | *p<0.05* | *p<0.05* | *p<0.05* | *p<0.05* |
| >30 (n=24) | 76.04±9.09 | 1.16±0.24 | 1.64±2.78 | 2.98±3.92 | 2.17±10.10 | 8.00±9.16 |
| *p-value* | *p>0.05* | *p<0.05* | *p>0.05* | *p<0.05* | *p<0.05* | *p<0.05* |

***Table 2.***
*Descriptive statistics.*

| BMI | | Range | Minimum | Maximum | Mean | Standard Deviation | Variance |
|---|---|---|---|---|---|---|---|
| < 25 | Age | 25 | 32 | 57 | 37.36 | 5.53 | 30.62 |
| (n=22) | BMI | 7 | 17 | 24 | 21.02 | 1.99 | 3.97 |
| 25-30 | Age | 33 | 22 | 55 | 41.39 | 9.60 | 92.25 |
| (n=23) | BMI | 4.09 | 25.30 | 29.39 | 26.96 | 1.20 | 1.43 |
| > 30 | Age | 29 | 21 | 50 | 34.50 | 9.63 | 92.78 |
| (n=24) | BMI | 5.22 | 30.49 | 35.71 | 33.39 | 1.59 | 2.51 |

In all the three groups, it was observed that the Parasympathetic tests, the Resting Heart Rate, E: I ratio tests and 30:15 ratio test were statistically not significant ($p>0.05$), whereas the Valsalva ratio tests showed statistical significance ($p<0.05$).

From among the sympathetic tests, the orthostatic hypotension test and handgrip test were statistically significant ($p<0.05$). Therefore, the BMI was correlated with the Valsalva ratio test for parasympathetic functions and with the orthostatic hypotension test and handgrip test for sympathetic functions.

The correlation of BMI with the orthostatic hypotension test showed a very weak negative correlation in Groups I and III, and a very weak positive correlation in Group II. In all the three Groups, this correlation was not statistically significant ($p>0.05$), (Tab. 3).

***Table 3.***
*Correlation of BMI with Orthostatic hypotension test, Handgrip test and Valsalva ratio test*

| Correlation (Pearson) | BMI and orthostatic hypotension test | BMI and handgrip test | BMI and Valsalva ratio |
|---|---|---|---|
| <25 (n=22) | -0.23 | 0.36 | -0.02 |
| p-value | p> 0.05 | p>0.05 | p>0.05 |
| Statistical interpretation | Very weak negative correlation | Moderate positive correlation | Very weak negative correlation |
| 25-30 (n=23) | 0.11 | -0.17 | 0.18 |
| p-value | p>0.05 | p>0.05 | p>0.05 |
| Statistical interpretation | Very weak positive correlation | Very weak negative correlation | Very weak positive correlation |
| >30 (n=24) | -0.011 | 0.07 | 0.08 |
| p-value | p>0.05 | p>0.05 | p>0.05 |
| Statistical interpretation | Very weak negative correlation | Very weak positive correlation | Very weak positive correlation |

However, clinically, in Groups II and III, orthostatic hypotension was more severe when compared with Group I. Correlation of BMI with the Handgrip test showed Moderate positive correlation in Group I, Very weak negative correlation in Group II and Very weak positive correlation in Group III. In all the three Groups, this correlation was not statistically significant ($p>0.05$), although clinically in Group III the increase in diastolic blood pressure was much higher than in Groups I and II, which indicated sympathetic over-activity in Group III. Correlation of BMI with Valsalva ratio test showed a very weak negative correlation in Group I and Very weak positive correlation in Groups II and III. In all the three Groups, this correlation was not statistically significant ($p>0.05$); however, clinically mild parasympathetic dysfunction was observed in Groups II and III.

## Discussion

These results indicate the presence of overactive Sympathetic functions and decreased parasympathetic activation to some degree, as the BMI increased.

These changes can be explained based on the Sympathetic-adrenergic and baroreflex functions in relation to obesity. Leptin is an adipocyte-derived protein hormone that performs the primary task of regulating food intake and energy homeostasis (via increased sympathetic nervous system (SNS) outflow) by binding to specific Leptin receptors in the hypothalamus [8, 9]. Numerous isoforms of the Leptin receptor are present in humans, but the soluble form of the Leptin receptor (sOB-R) has been most studied and shown to be low in concentration in obese patients [10]. Leptin resistance is a physiological state observed in human obesity that is characterized by high leptin and low sOB-R concentrations. In fact, leptin has been postulated to increase the sympathetic nervous system activity by increasing the concentrations of circulating norepinephrine, as evidenced in animal models [9, 11]. A recent study in children indicated that leptin resistance decreases when the adiposity level decreases and this reduction is associated with improved cardiovascular function (decreased blood pressure) [12].

The findings of this study are consistent with those of Christopher L. Kaufman [13] who studied the relationships of cardiac autonomic function with metabolic abnormalities in childhood obesity. In addition, Riva P. [14] who studied obesity and autonomic function in adolescence, observed a similar increase in sympathetic tone coupled with a drop in the vagal tone.

However, our findings are inconsistent with those of K Laederach-Hofmann [15], Ramis Çolak [1], who found no overstimulation with increase in weight, but on the contrary observed a depression in the sympathetic and parasympathetic activity.

In conclusion, the study showed an increase in the Sympathetic activity and to some degree a decrease in the Parasympathetic activity in males, as the BMI increased. These Autonomic dysfunctions in obesity could be due to the leptin resistance and diminished baroreceptor function with increase in weight. Finally, the important changes in ANS function dependent on weight appear to be a promising avenue to conduct long-term studies on weight loss and weight gain in the future.

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