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### Original article

## Effect of obesity on autonomic nervous system

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#### ABSTRACT

The present study was carried out on 100 volunteers of which 50 subjects with BMI > 30kg/m<sup>2</sup> were included in study group and 50 subjects with BMI < 30kg/m<sup>2</sup> (non obese) were included in control group. The functioning of Autonomic nervous system was evaluated by six non-invasive tests- four of which were based mainly on parasympathetic control (30:15 ratio, standing to lying ratio (S/L ratio), expiration/inspiration ratio (E/I ratio) and valsalva maneuver) and two on sympathetic control (Blood pressure response to standing and cold pressor test). The results of the present study showed significantly low (p=0.001) S/L ratio in study group (1.04 ± 0.12) when compared to controls (1.12 ± 0.11) indicating impaired parasympathetic function. The mean change in systolic blood pressure before and after cold pressor test (CPT) was less in study group (7.12 ± 5.28) as compared to control group (10.38 ± 6.35) and this was statistically significant (p=0.006) indicating impaired sympathetic function. Thus, in obese both division of ANS are affected which may be the cause of various cardiovascular complications.

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### 1. Introduction

With continued rise in standards of living, obesity is emerging as a global epidemic in both children and adults. This has been called "New world syndrome" and is a reflection of massive social, economic and cultural problems currently facing developing and developed countries. Obesity is regarded as a complex disease because it arises from multifaceted interactions of genetic and environmental factors. [1] Obesity occurs when caloric intake exceeds energy expenditure and the excess calories are stored in an adipose tissue. Therefore, the ultimate cause of obesity is suggested to be an imbalance between energy intake and expenditure resulting from complex interaction of genetic, physiological, behavioural and environmental factors. [2] Since ANS is involved in energy metabolism and regulation of

cardiovascular system [3,4], It is conceivable that one or more sub groups of obesity have an alteration in their autonomic nervous system that may promote obesity and account for several clinical consequences of obesity.

Several studies in literature suggest that ANS of obese individuals is chronically altered.[5,6] The activity of sympathetic nervous system is a determinant of energy expenditure. It has been observed that Individuals with low resting muscle sympathetic nerve activity may be at risk for body weight gain resulting from a lower metabolic rate.[7] Obesity was found to be associated with decreased sympathetic activity in animal models.[8] Experimentally induced ventro-medial hypothalamic lesions resulted in decreased sympathetic activity, increased parasympathetic activity and obesity.[4] Overfeeding is found to be associated with sympathetic activation and there is evidence that adrenergic mechanisms contribute to cardiovascular complications.[9].

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Since there is dearth of literature for comprehensive studies in relation to autonomic disturbances in obesity in India. So, the present study has been attempted to assess relationship between ANS and obesity using autonomic function tests as diagnostic tools.

## 2. Materials and methods

The present study was carried out on 100 volunteers of which 50 subjects with BMI > 30kg/m<sup>2</sup> were included in study group and 50 subjects with BMI < 30kg/m<sup>2</sup> (non obese) were included in control group.

The mean age of subjects in study group was 33.38 ± 8.73 years and in control group was 34.86 ± 10.24 years. The subjects of study group were taken from local obesity centers – Personal point and Jannat. All the tests were performed in department of Physiology, DMC & Hospital, Ludhiana.

### 2.1. Exclusion Criteria

1. Patients of malignant hypertension, diabetes, psychological diseases, head trauma, cardiac abnormalities, IHD, cardiac failure, chronic obstructive lung diseases, psychological disorder etc.
2. Patients suffering from any clinical diseases likely to affect ANS.
3. Patients with history of smoking/alcohol/drug abuse.
4. Patients with abnormal random blood sugar (tested with glucometer). Patients taking medication e.g. Vasodilators,  $\alpha$  blocker,  $\beta$  blockers, barbiturates, opiates, tricyclic antidepressants and phenothiazines that could affect autonomic functions were also excluded from the study.

Details of proceedings and need of history was described to each volunteer so that subject is in state of calmness and without any anxiety at the time of the test. Consent was taken from volunteers before starting the tests. Following autonomic function tests were employed to evaluate the integrity of both parasympathetic and sympathetic innervation of heart (i.e. integrity of entire autonomic reflex arc) in both the groups.

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**2.2. Heart rate response to immediate standing (30:15 ratio)**- was calculated when the subject rose to erect posture from supine position. The 30:15 ratio was calculated by taking ratio of longest R-R interval at beat 30 and shortest R-R interval at beat 15 after standing. A 30:15 ratio of 1.00 was taken as normal and value of <1.00 was considered as abnormal.[10]

**3. Standing to lying ratio (S/L ratio)** – In this test ECG was recorded from 20 beats before to 60 beats after lying down. The point at which subject started to lie down was marked. S/L ratio was calculated as longest R-R interval during 5 beats before lying down to shortest R-R interval during 10 beats after lying down.[11] S/L ratio of >1 was taken as normal and <1 as abnormal.[12]

### 2.4. Heart rate during deep breathing

**(Expiration/inspiration ratio)** – Subject was asked to breathe deeply at rate of six breaths per minute. A standard ECG recording was taken during deep inspiration and expiration. Variation in heart rate was calculated as rate of longest R-R interval during expiration to shortest R-R interval during inspiration.[13] A value of 1.20 or higher was taken as normal.[14]

### 2.5. The Heart rate response to Valsalva manoeuvre

**(Valsalva ratio)**- Subject was made to perform Valsalva manoeuvre for 15 seconds by blowing against closed glottis through a mouth piece attached to manometer and maintained an expiratory pressure of 40mm of Hg for 15 sec. ECG was recorded during the manoeuvre (Strain period, 15 sec) and for fifteen seconds after release of pressure. The Valsalva ratio was calculated as the ratio of longest R-R interval after manoeuvre to shortest R-R interval during manoeuvre. Value >1.21 was taken as normal and value <1.21 was considered as abnormal. [15]

### 2.6. Blood pressure response to standing

– After BP of the subject was recorded in supine position, he was made to stand within 3-4 sec. and to remain motionless. Blood pressure (both SBP & DBP) was recorded in 30 second interval. Difference between readings of SBP & DBP in lying position and then after standing were calculated. Normal response was taken as <10mmHg fall in blood pressure. A fall in BP >20-30 mm Hg was taken as abnormal.[16]

### 2.7. Cold Pressor test

– Subject was asked to immerse his hand in cold water and temperature was maintained at 4-6 °C throughout procedure. Blood Pressure measurement was made from other arm at pain threshold time, which is defined as time between immersion of hand and subjective feeling of pain. Maximum increase in systolic and diastolic pressures were recorded. A rise of diastolic BP >15 mmHg was taken as normal and less than this was considered as abnormal.[12] Failure of systolic BP to rise by 16-20 mmHg and diastolic BP by 12-15 mmHg was indication of autonomic neuropathy.[17]

The results of study were expressed in mean S.D. and were compared with the control group. The data was analyzed using ANOVA test.

## 3. Results

The mean BMI of study group was 34.25 ± 3.13 kg/m<sup>2</sup> and that of control group was 23.78 ± 2.06 kg/m<sup>2</sup>. The difference between groups was statistically significant (p = 0.000).

The study group showed a decrease in mean 30:15 ratio (1.10 ± 0.20) when compared to control group (1.14 ± 0.11) but this was not significant statistically (p=0.225).

The standing/lying ratio (1.04 ± 0.12) in the study group decreased significantly (p=0.001) as compared to that in the control group (1.12 ± 0.11).

The expiration/inspiration ratio was low (1.26 ± 0.18) in the study group than in the control group (1.31 ± 0.15) but it was non significant statistically (p=0.226).

Valsalva ratio in the study group was (1.41 ± 0.38) low as compared to control group (1.40 ± 0.26) but was non significant statistically (p=0.892).

The difference in mean systolic blood pressure (2.60 3.70 mmHg) on change from lying to standing in study group was less as compared to that in control group (4.14 4.49mmHg) but this was statistically non-significant (p=0.065).

The difference in mean diastolic blood pressure on change from lying to standing in study group (3.96 3.64 mmHg) was more as compared to control group (2.92 4.96 mm Hg) but this increase was non significant.(p=0.236)

The difference in mean systolic blood pressure recorded before and after cold pressor test was low in the study group (7.12 5.28 mmHg) as compared to the control group (10.38 6.35 mmHg) and was statistically significant.(p=0.006) The difference in mean diastolic blood pressure recorded before and after CPT was non-significantly (p=0.500) lower in study group (7.32 3.95 mmHg) as compared to control group (7.84 3.72 mmHg).

**Table shows comparison of parameters in study and control group.**

Study group	Study group n=50	Control group n=50	F value	pvalue
Age (Years)	33.38 ± 8.73	34.86 ± 10.24	0.604	0.439
BMI (Kg/m <sup>2</sup> )	34.25 ± 3.13	23.78 ± 2.06	389.258	0.000*
30:15 Ratio	1.10 ± 0.20	1.14 ± 0.11	1.491	0.225 NS
S/L Ratio	1.04 ± 0.12	1.12 ± 0.11	11.385	0.001*
E/l Ratio	1.26 ± 0.18	1.31 ± 0.15	1.482	0.226 NS
Valsalva Ratio	1.41 ± 0.38	1.40 ± 0.26	.081	0.892 NS
BP change on standing (SBP mm of Hg)	2.60 ± 3.70	4.14 ± 4.49	3.496	0.065 NS
BP change on standing (DBP mm of Hg)	3.96 ± 3.64	2.92 ± 4.96	1.423	0.236 NS
CPT(SBP mm f Hg)	7.12 ± 5.28	10.38 ± 6.35	7.779	0.006*
CPT(DBP mm of Hg)	7.32 ± 3.95	7.84 ± 3.72	0.459	0.500 NS

NS = Non Significant \* = Significant

Abbreviations :

- 30:15 Ratio – Heart rate response to immediate standing
- S/L Ratio – Standing to lying ratio
- E/l Ratio – Heart rate during deep breathing
- Valsalva ratio – Heart rate response to Valsalva manoeuver
- CPT – Cold pressor test
- SBP – Systolic blood pressure
- DBP – Diastolic blood pressure

#### 4. Discussion

Obesity is a common and significant health hazard and is associated with an increased incidence of hypertension, congestive heart failure and unexplained sudden death as well as an overall increase in mortality rate.

In the present study, standing to lying ratio was significantly lower in study group (1.04 0.12) as compared to control group (1.21 0.11) (p=0.001). This decrease indicates impaired vagal function in the study group. Similar results have been reported by other workers[18] who observed decreased parasympathetic response in terms of HRV (Heart Rate Variability analysis) of 5 minute R-R interval before and after head up tilt manoeuver. In another study it has been seen that Cardiac parasympathetic

dysfunction present in obese subjects could be associated with higher carbohydrate intake and lower fat and protein intake which results in parasympathetic abnormality. [19] It is also demonstrated that parasympathetic activity increased with weight loss in obese.[20]

In the present study, the mean change in systolic blood pressure before and after CPT was statistically significant (p= 0.006) in study group as compared to control group. The impaired CPT in obese could possibly be because of hypofunctional sympathetic nervous system. Results of our study correlate with observations made by other workers [21] that showed increase in blood pressure during exposure to cold pressor test on loss of around 30% of excess weight following a

period of hypocaloric diet. In another study, there was reduced sympathetic responsiveness associated with thermoregulation demonstrated by abnormal heart rate variability on cold exposure. [22]

Hence finding of our study suggested impaired autonomic nervous system function (both sympathetic and parasympathetic) in obese. The results of present study are similar to other human studies in respect to cardiac autonomic dysfunction in obese. [6,23] In another study obese children possessed a decreased sympathetic and parasympathetic activity [24,25] and parasympathetic activity increased with weight loss in obese women. [26] It is documented that low sympathetic activity and a low activity of adrenal medulla leads to development of central adiposity. [27]

## 5. Conclusions

Obesity is associated with ANS dysfunction which may be cause of various cardiovascular complications. So, if autonomic nervous system dysfunction is diagnosed early by doing autonomic function tests, it may prove an important aid in identification of those prone to weight gain and are at higher risk of cardiovascular complications resulting for autonomic dysfunction.

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