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Physiology

HEART RATE VARIABILITY AS AN INDICATOR OF CARDIAC AUTONOMIC MODULATION IN OBESE ADULTS

Dr. Mamta Vinay Rode Associate Professor , department of Physiology Government Medical College, Nagpur

KEYWORDS :

INTRODUCTION

Obesity has reached global epidemic proportion in adults and children. (1) Obesity has evolved with the advent of civilization, sedentary lifestyle, high caloric diet.(2) Obesity is one of the causative factor for its morbid conditions leading to metabolic and cardiac disorder (3) Obesity is associated with numerous comorbidities including hypertension, dyslipidemia , cardiovascular diseases because of its maladaptive effects on various cardiovascular risk factors and its adverse effects on cardiovascular structure and function.(4)

Heart rate variability is used as a non invasive measurement of cardiac autonomic modulation.(5) The balance of sympathetic and parasympathetic modulation regulates HRV. Lower HRV has been associated cardiovascular diseases, morbidity and mortality.(6) HRV also indicate the extent of neuronal damage to autonomic nervous system .

HRV represents continuous fluctuation in heart rate. RR interval variations in ECG represent beat to beat control of heart rate mainly by the autonomic nerve supply to heart. Thus HRV measured by power spectral analysis provides a quantitative marker of autonomic neural control of heart rate and has been shown to reflect cardiovascular health. Time domain analysis of HRV uses statistical methods to quantify the variation of standard deviation or the differences between successive RR intervals. Frequency domain analysis of HRV enables us to calculate the respiratory dependent high frequency (HF) and the low frequency (LF) powers. High frequency power is mediated by vagal activity (8) while low frequency power represents both sympathetic and parasympathetic activity but predominently sympathetic modulation where as LF/HF ratio reflect sympathovagal balance or the sympathetic modulation.(9).

The study is designed to evaluate the effect of obesity on cardiac autonomic activity in young normal weight and obese adults.

MATERIAL AND METHODS

This cross sectional study includes 100 participants in the age group of 18-20 years. Subjects were divided into two groups depending on their BMI. Obese group with BMI ≥ 30 kg/m^2 and control group with BMI $\leq$ 25 kg/m^2. They were requested to complete a questionnaire that include specific information on age, drugs intake, physical activity, tobacco, alcohol consumption. Written informed consent was taken. Clinical examination was conducted of all subjects to rule out any systemic disease. Weight to nearest .5kg, height, and waist circumference and hip circumference to nearest 0.1cm were measured according to standard procedure. Body mass index was calculated by dividing weight in kgs by the square of height in meters. The study was carried out in human physiology laboratory. After reporting subjects were asked to relax for 15 min. Resting heart rate was recorded. Blood pressure was measured. ECG was recorded for 15 minutes using physiopac pp-4 Medicaid system.

HRV analysis included time and frequency domain indices. Time domain indices in milliseconds includes SDNN standard deviation of the all NN interval SDANN standard deviation of the average NN interval, RMSSD square root of the mean of the sum of the squares of differences between adjacent NN interval. NN50-adjacent NN interval that are greater than 50 ms PNN 50% percentage of difference between adjacent NN interval that are greater than 50ms.

Frequency domain includes Total power in ms^2, VLF (ms^2) very low frequency band variation LF absolute values in msec, LF/HF ratio. The study was approved by institutional ethics committee. Statistical analysis were performed using SPSS graph pad software. In order to compare physical characteristics, time domain and frequency domain of HRV indices between two groups were tested by applying students 't' test

Table-Anthropometric characteristics of two group

Variables	Control group	Cases	P value
Age years	20.98 ± 2.18	20.62 ± 2.51	P > 0.05
Ht. cms.	163.27 ± 7.78	162.35 ± 5.55	P > 0.05
Wt. Kg	60.25 ± 5.25	80.45 ± 4.5	P < 0.05
BMI %	22.45 ± 1.32	32.6 ± 3.95	P < 0.05
Waist circumference (cm)	82.05 ± 5.50	98.25 ± 6.80	P < 0.05
Hip circumference (cm)	96.04 ± 3.20	105.75 ± 8.30	P < 0.05
WHR	0.85 ± 0.05	0.93 ± 0.05	P < 0.05

Resting Heart Rate & BP in two groups

Variables	Control	Cases	P Value
Heart rate beats/min	78.57 ± 10.48	86.05 ± 12.09	P < 0.05
Systolic BP mm of Hg	120.75 ± 6.98	128.60 ± 8.69	P < 0.05
Diastolic BP mm of Hg	80.12 ± 3.48	86.00 ± 4.25	P < 0.05

Time domain parameters of HRV in two groups

Variables	Control	Cases	P Value
SDNN (msec^2)	79.3 ± 18.36	42.96 ± 15.42	P < 0.05
SDANN (msec^2)	57.1 ± 23.35	40.40 ± 18.32	P < 0.05
RMSSD (msec^2)	55.0 ± 21.45	43.0 ± 4.25	P < 0.05
NN50 (msec^2)	76.0 ± 41.53	60.50 ± 42.29	P < 0.05

Frequency domain parameters of HRV in two groups

Variables	Control	Cases	P Value
Total power	4824.6 ± 1864.56	3487 ± 1555.50	P < 0.05
VLF(msec^2)	1333.8± 916.2	979.2± 576.6	P < 0.05
LF (ms^2)	1862.8 ± 919.75	1975 ± 660.49	P > 0.05
LF (nu)	85.01 ± 3.31	93.32 ± 5.51	P > 0.05
HF (ms^2)	1533.7 ± 916.2	841.2 ± 576.6	P < 0.05
HF(nu)	41.4	32.2	P < 0.05
LF / HF	12 ± 0.64	2.4 ± 1.69	P < 0.05

DISCUSSION

The present study shows that the heart rate was significantly higher in obese young adults. Higher heart rate is marker of relative sympathetic dominance and is an independent marker of mortality in a wide spectrum of conditions (8). Systolic and diastolic blood pressure was significantly higher in obese group. Obesity associated hypertension might be attributed to production of several vasoactive factors from adipose tissue. Increased sympathetic nervous system activity owing to the central nervous system action of leptin and low atrial natriuretic factor level leading to sodium retention and volume expansion (10).

The study also shows that in obese young adults SDNN, SDANN and TP was significantly lower indicating decrease in HRV in young obese adults. Thus the study confirms the high prevalence of alteration in HRV in obese individuals as shown in earlier studies on obese children, adolescent (12) and adults (13,14).

Reduced heat rate variability is a marker of sympathovagal imbalance. Time domain measures of heart rate variability, SDNN, SDANN reflect both sympathetic and parasympathetic modulation of heart rate and reduced SDANN and SDNN values usually indicate relative sympathetic dominance (15). Further more in prospective study reduced heart rate variability has been shown to be the strongest independent predictor of progression of focal coronary atherosclerosis (16) decreased HRV could partly account for the higher cardio vascular risk and incidence of sudden death in obese person.

Our result suggests that in obese young adults there was decrease in RMSSD, NN50, pNN50 of time domain indices and HF component of frequency domain indices which mainly assess the parasympathetic activity of heart indicating lower parasympathetic activity in obese young adults. These findings are similar to other studies which demonstrated that obese persons have decreased parasympathetic activity, 7, 17.

The exact mechanism that may cause impairment of parasympathetic nerve function is not yet been established. Obesity is a state of impaired glucose, tolerance, hyperinsulinemia and insulin resistance. Acute insulin administration has been shown to reduce high frequency power and measure of respiratory sinus arrhythmia during euglycemic hyperinsulinemia in normal weight and obese subjects. The hyper insulinemia may contribute to low cardiac vagal activity (18).

The present study shows that obese young adults have higher LF and LF/HF ratio components of frequency domain indices which mainly measures the sympathovagal balance to heart reflecting an increase in sympathetic nerve activity in these persons. The LF / HF ratio has been proposed to be an accurate measure of the overall sympathovagal balance of the autonomic nervous system in which higher values indicate a more sympathetically driven cardio vascular system (19).

Earlier studies on sympathetic nerve activity in obese persons have conflicting results. Some studies have shown decrease (20,12) and some increase in sympathetic activity in obesity (22).

Measurements of plasma and urinary catecholamine concentration as indices of sympathetic nervous system activity have ranged from low to through normal to high 23, 24.

Insulin and leptin levels are elevated in obesity. Thus increased insulin and leptin levels are thought to increase sympathetic nervous system activity, 12, 25,26.

Obese persons may suffer from an increased mortality risk due to cardio vascular disorder related to either continuously lowered Parasympathetic activity or altered sympathetic activation. HRV analysis can detect changes even before clinical signs appear 6. Thus regular assessment of HRV measures can be used as a biomarker for early detection and subsequent management of cardio vascular disease in obese individual.

CONCLUSION

The present finding demonstrate decrease HRV, higher sympathetic and lower parasympathetic activity in obese subjects.

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