



HEART RATE VARIABILITY OF ONE MINUTE CONTROLLED DEEP BREATHING IN OBESE INDIVIDUALS

ARCHANA

Department of Physiology, STANLEY MEDICAL COLLEGE AND HOSPITAL

Abstract : BACKGROUND Obesity, an evolving metabolic disease, has become the most common risk factor for Non-communicable diseases. The increased morbidity and mortality in these diseases are due to autonomic imbalance¹. Heart Rate Variability (HRV) analysis is a tool to assess the cardiovascular autonomic function. Although, many methods are there to study the relationship between obesity and autonomic function, HRV plays an easy, simple, noninvasive and effective method. AIM OBJECTIVE To evaluate and compare cardio-respiratory interactions using HRV in respiratory maneuvers among Obese and non-obese individuals. MATERIALS AND METHODOLOGY After twenty minutes of supine rest, ECG (Lead II) was acquired during one minute controlled deep breathing at 6 breathes per minute and instantaneous RR intervals were analysed using Finland software for HRV. Time domain measures of obese were compared with age and gender matched non-obese group. Time domain measures SDNN (Standard Deviation of NN interval in ms), RMSSD (Root Mean Square of Standard Deviation in ms), NN50, p NN50 () and Deep breathing maximum (DB max) were taken for analysis. RESULT In Obese, Mean HR increased and Mean RR decreased significantly. There is a significant decrease in SDNN, RMSSD, NN50, pNN50() in Obese compared to Non obese. Deep breathing ratio is also significantly decreased in obese. CONCLUSION Obese individuals found to have impaired cardiovagagal dysfunction characterized by depressed parasympathetic activity.

Keyword : Obesity, Body Mass Index, Autonomic function, Heart Rate Variability

INTRODUCTION:

Obesity, an excessive accumulation of adipose tissue, is an important risk factor for developing chronic disease¹. With continued rise in standards of living, Obesity has become global epidemic² and “New world syndrome”. Consequently, this has lead to massive social, economic and cultural problems across developing and developed countries. This complex disease arises from multifaceted interactions of genetic and environmental factors^{2,3}. Of these the prime cause for obesity has been found to be the imbalance between energy intake and energy expenditure⁴.

The ANS is an Integrated Nervous System known to play a role in the interaction between circulation and respiration. Respiratory Sinus Arrhythmia is a rhythmical fluctuations in RR periods

(interval between successive QRS complex) at the respiratory frequency which has been characterized by the shortening and lengthening of RR interval in relationship with inspiration and expiration⁵. RSA is used as a selective non-invasive index of vagal control of heart. Respiratory mediated heart rate changes are small during quiet breathing but more pronounced respiratory sinus arrhythmia during deep breathing. RSA magnitude is inversely related to respiratory rate and directly related to tidal volume. Increase in tidal volume at slower respiration rates will cause larger RSA elevations than the same tidal volume increase at more rapid respiration rates⁶. These respiratory maneuvers, associated with reflex cardiovascular changes, provide an important information about the appropriate function of ANS and functional capacities of effectors (heart and vessels).

It is well known that Autonomic Nervous System plays pivotal role in regulation of energy metabolism and cardiovascular System¹. Thus, whether Obesity results from ANS dysfunction or Obesity itself a mounting factor for ANS dysfunction is unclear. We have attempted to study autonomic function in obese using Heart Rate Variability of One minute Controlled Deep Breathing as a diagnostic tool.

AIM: To test whether evaluation of the cardio- respiratory interactions using Heart Rate Variability can reveal cardiovagagal dysfunction in Obese individuals.

OBJECTIVE: To compare HRV during one minute controlled deep breathing in Obese with Non obese adults.

MATERIALS AND METHODS:

The study was done in thirty age and gender matched obese and non-obese individuals (Obese: 17:13, female: male; aged 28.13 ± 5.45 ; non-obese aged 26.33 ± 4.15)⁷. These participants were drawn from Master Health check-up at out-patient department. The study was conducted at the Neurophysiology Laboratory, Department of Physiology, after obtaining an informed and written consent from all the participants and was approved by the Institutional Ethics Committee.

Detailed history and clinical examination was done to exclude conditions interfering with autonomic modulation like smoking, alcohol intake, caffeine intake, meals, hypertension, diabetes mellitus and drug intake. All these examination were carried out between 8.00 AM and 1.00 PM under comparable ambient conditions, after having made the participants relaxed.

After cleaning the skin surface with the spirit, ECG electrodes were placed properly and lead II ECG was recorded for five minutes after a resting period of 15 minutes at supine rest position with normal breath rate of 12 – 16 / minute, using RMS polyrite D Hardware 2.2 (India). Then, participants were trained to breath at 6 breaths per minute, with inspiration for 5 sec and expiration for 5 seconds and ECG was recorded. The instantaneous RR intervals were plotted using Finland1.1 version, University of Kuopio, and Software for HRV analysis, with the interpolation frequency of 4Hz and smoothness prior method as per recommendations of Task Force⁸ (Circulation 1996).

BMI: Body weight was measured (to the nearest 0.5 kg) with the individuals in position standing motionless on the weighing scale and with the weight distributed equally on each leg. Height was measured (to the nearest 0.5cm) with the individual standing in an erect position against a vertical scale and with the head positioned so that top of the external auditory meatus was level with the inferior margin of the bony orbit (Frankfurt's plane). WHO Asian Pacific Standards⁷ were taken for BMI classification.

Heart Rate Variability Analysis: HRV analysis was performed with linear methods in the time domain. In the time domain, SDNN [Standard Deviation of NN interval and in the frequency domain], RMSSD [Root Mean Square of Standard Deviation], NN50 [number of differences between adjacent NN intervals greater than 50msec] and pNN50 [the percentage of differences between adjacent normal to-normal intervals greater than 50 msec] were used. The HRV analysis software Finland1.1v of Kuopio Lab was used to analyse the recording. Deep breathing maximum (DB max), a mean ratio of maximum / minimum in RR interval during one minute of controlled deep breathing was also used.

STATISTICAL ANALYSIS:

SPSS version 15 was used for statistical analysis. The Data are expressed as mean \pm Standard Deviation. The variation in parameters between the two groups was tested using student's independent t-test. P value less than 0.05 was considered significant.

RESULTS:

In Table I, there shows no significant difference in age between obese and non obese. Weight and BMI is significantly ($p < 0.01^{**}$) higher in obese compared to Non obese.

Table II shows significant increase in Mean HR and decrease in RR in obese than Non obese. SDNN decreased significantly in Obese compared to Non Obese. Also RMSSD, NN50, pNN50 significantly decreased in obese compared to Non Obese. There is also significant decrease in DBmax in Obese compared to Non obese.

DISCUSSION:

In the present study, there is an increase in mean heart rate during paced breathing in obese individuals. Normally the chronotropicity of heart is under the parasympathetic control⁸. In Hugh. Peterson et al¹⁰ and Hirsch et al¹¹ it is showed that resting heart rate is increased with increase in body fat and 10 % increase in this body fat is associated with decline in parasympathetic tone. It is well documented that heart rate is inversely related to RR interval⁹ (young et al). HRV shows the variations between consecutive heart beat and therefore beat to beat control mechanisms. Hence we hypothesized autonomic dysfunction in obese individuals and studied HRV in one minute controlled deep breathing in obese individuals.

It is well known that during deep breathing there is an increase in parasympathetic activity at SA node. Hence, the increase in heart rate observed in obese during deep breathing suggests that there is definite parasympathetic deactivation. Thus SDNN reflecting parasympathetic activity⁸ is observed to be reduced in the obese individuals when compared to non obese. Also RMSSD, NN50 and pNN50 were also reduced in obese.

Deep breathing maximum is decreased in Obese compared to Non obese. The normal value for deep breathing for age group 18 – 25 years was 1.21 (Smith et al 1995). But in our study, DB max in obese is 1.17. This indicates a reduced respiratory sinus arrhythmia in obese which strongly reinforces decreased parasympathetic activity in obese¹². The decrease in the DB in the study group may be due to reduced baroreflex sensitivity or impaired vagal afferents to the brain or an impaired ability of the brain stem to recognize the different signals resulting in an altered efferent signal to the heart and produce decreased cardiac parasympathetic activity¹¹.

This parasympathetic withdrawal explain a subclinical autonomic dysfunction in obese, which could be due to leptin resistance, inflammatory factors like TNF alpha, Interleukins and sedentary lifestyle^{13,14}. This vagal restraint could be the reason for sudden death happening in the busy world.

CONCLUSION:

Our study indicates there is a reduction in the HRV. Cardiac autonomic function is impaired by decreased parasympathetic activity. The result suggest there is a need for monitoring increase in their weight and to prevent future complications.

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N= 30	OBESSE		NON OBESSE		p value
	Mean	Standard Deviation	Mean	Standard Deviation	
Age (years)	28.13	5.45	28.33	4.15	0.158
Weight (kilograms)	75.53	12.25	58.20	8.01	*0.01**

Height (centimetres)	155.05	5.55	150.45	4.75	0.125
BMI (Kg/m ²)	30.61	4.45	21.81	1.89	*0.01**

*p<0.05 - significant **p<0.01 - highly significant

N= 30	OBESSE		NON OBESSE		p value
	MEAN	STANDARD DEVIATION	MEAN	STANDARD DEVIATION	
Mean HR (beats per min)	84	8	71	12	*0.01**
Mean RR (milliseconds)	713	72	555	132	*0.01**
SDNN (milliseconds)	62.07	27.45	100.22	19.15	*0.01**
RMSSD (milliseconds)	65.57	25.12	94.43	17.43	*0.01**
NN50	74.57	51.50	110.53	57.23	0.037*
pNN50 (%)	15.13	15.75	31.22	20.15	0.014*
SD max	1.17	0.03	1.25	0.05	*0.01**

*p<0.05 - significant ** p<0.01 - highly significant