RESEARCH ARTICLE

Influence of body fat on heart rate variability in healthy females

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Abstract

Objective: To evaluate the effect of central obesity indices in healthy females on cardiac autonomic activity, using heart rate variability as an index reflecting the cardiac autonomic balance.

Method: The cross-sectional study was conducted at the Department of Physiology, College of Medicine, Mustansiriyah University, Baghdad, Iraq, from September 2020 to April 2021, and comprised adult healthy women having a regular menstrual cycle who were not consumers of tobacco, alcohol or any medication. Waist circumference, waist-hip ratio, waist-height ratio and lipid accumulation product measurements were noted for each subject. Repeated blood pressure and heart rate measurements were taken on the brachial artery of the right arm until stable values were achieved. Heart rate variability was based on electrocardiogram data related to R-R interval. Data was analysed using GraphPad InStat version 3.06.

Results: There were 53 women with mean age 37.4±10.5 years. Low-frequency power normalised units at the highest two quintiles of waist circumference was significantly higher than the lowest two quintiles ($p<0.03$), and significantly and positively correlated with waist-hip ratio ($p<0.02$) and waist-height ratio ($p<0.05$). At the highest two quintiles of lipid accumulation product, low-frequency power normalised units value was significantly higher than at the lowest two quintiles ($p<0.02$).

Conclusion: Increased waist circumference, waist-height ratio, waist-hip ratio and lipid accumulation product values were significantly associated with high sympathetic tone.

Key Words: Abdominal, Circumference, Hip Ratio, Blood Pressure, Arm, Brachial Artery, Heart Rate, Lipid Accumulation, Electrocardiography, Menstrual Cycle

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Introduction

Obesity is one of the leading diseases that increase mortality from cardiovascular disease (CVD).[1] Central obesity is a visceral, metabolically active fat that surrounds the organs and is linked to metabolic dysregulation, predisposing individuals to CVD and other conditions that are represented by waist circumference (WC), waist-height ratio (WHtR) and waist-hip ratio (WHR). Insulin resistance (IR), hypertension (HTN) and decreased high-density lipoprotein (HDL) have been proposed as plausible reasons for this relationship. However, an increase in the incidence of CVD in obese people has been linked to a sympathovagal deficiency of autonomic function[2].

Heart rate variability (HRV) is defined as a beat-to-beat variation in the cardiac rhythm which possibly occurs because of periodic changes in parasympathetic and sympathetic inputs to the heart. HRV is affected by various factors, like respiration, circadian rhythm, environmental factors, yoga and exercises[3]. At rest, it is predicted that parasympathetic cardiac modulation will be predominant. Patients with heart disease have a more sympathetic tone and less parasympathetic tone of the heart. Sedentary activities, like sitting and watching television, as well as using the computer for an extended period contribute to weight-gain that influences the cardiac autonomic functions by increasing sympathetic and decreasing parasympathetic activity[4]. Several studies have found lower HRV in overweight and obese people, implying that autonomic abnormalities are involved in the pathways that promote arrhythmia and sudden death. As a result, autonomic processes of asymptomatic overweight people should be tested to see whether any abnormalities exist in a subclinical stage, which aids in early diagnosis and prevention of obesity-related complications [5].

The current study was planned to evaluate the effect of central obesity indices in healthy females on cardiac autonomic activity, using HRV as an index that reflects the cardiac autonomic balance.

Subjects and Methods

The cross-sectional study was conducted at the Department of Physiology, College of Medicine,

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Mustansiriyah University, Baghdad, Iraq, from September 2020 to April 2021. Healthy females having regular menstrual cycle who were not consumers of tobacco, alcohol or any medication were enrolled in the study. The sample was collected using the simple random sampling technique. Data, including medical history, was collected after taking verbal informed consent.

Height and weight of the subjects, who had been fasting overnight, were measured using a flexible, non-elastic measuring tape. WC was measured as the circumference of the abdomen at the umbilicus, and maximum hip circumference (HC) was measured with the legs closed.

Increased cardiovascular risk was defined as WC ≥94cm in men, and ≥80cm in women[6].

Blood samples were taken via venipuncture for analysis of triglycerides (TG). Lipid accumulation product (LAP) was calculated as: LAP = [(WC in cm) −58)] × TG concentration (mM). The cut-off value for LAP was set at 40.6-44. 0cm.mmol/l[7].

With the subject in a supine position, systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured after 5min of complete rest with the head slightly flexed and completely supported by the couch surface. Repeated BP and heart rate (HR) measurements were taken with an automated sphygmomanometer (Rossmax Swiss GmbH, Tramstrasse 16, CH-9442 Berneck, Switzerland) on the brachial artery of the right arm until stable values for both parameters were achieved. The subjects were asked to avoid Valsalva manoeuver, to maintain spontaneous respiration, and to remain quiet throughout the procedure.

HRV measurements were done using an analogue-to-digital converter (PowerLab Data Acquisition Unit 26T, AD Instruments Pty Ltd, New South Wales, Australia) and LabChart Pro version 7.2 software through which electrocardiogram (ECG) signals were converted and digitally recorded on a computer. For ECG recording, two electrodes were attached to the right arm and left leg (Lead II), and one electrode was attached to the right leg (serving as earth). ECG was recorded for 5 min sampled at 1kHz.

The R-R interval data from the ECG recording was carefully examined for abnormal beats and artifacts. The existence of artifacts or the presence of ectopic beats affects HRV analysis and compromises the veracity of the parameters, if not secluded. Removal of abnormal beats and artifacts was achieved by visual inspection of all R-R intervals throughout the 5 min period. The outlier data (abnormal beats and artifacts) was specified and removed using Rosner's Extreme Studentised Deviate test for multiple outliers. The probability was set to 0.05. The residual data was then processed by the free version of Kubios HRV standard software version 3.3.0 (Kuopio, Finland) which was used to calculate the frequency domain measures of HRV for HRV measurements without the application of filters. The analysis was done in terms of frequency domain measures from the R-R tachogram obtained with the same software. The software detects automatically R peaks to obtain the tachogram. The process identified domain parameters as: LF power (ms2) = Low-frequency band absolute power (0.04-0.15 Hz), and HF power (ms2) = High-frequency band absolute power (0.15-0.4 Hz).

With respect to frequency domain indices, low-frequency (LF) power, and LF normalised units (LFnus) were measures of cardiac sympathetic activity, while high-frequency (HF) power and HFnus were measures of cardiac parasympathetic activity. The LF-HF ratio was the measure of cardiac sympatho-vagal balance.

To explore the effect of central obesity on HRV indices, WC data was arranged in descending order and divided into 5 quintiles. Then HRV indices of the highest two quintiles were compared with the HRV indices of the lowest two quintiles. The data of the middle quintile was excluded from the comparison.

Data was analysed using GraphPad InStat software version 3.06. Data was expressed as mean and standard deviation (SD). To compare data between variables, an unpaired student's t-test was used. Kolmogorov-Smirnov test, Mann-Whitney U test were used, as apprpriate. The correlation between variables was calculated in Excel. P<0.05 was considered statistically significant.

Results

There were 53 women with mean age 37.4±10.5 years. Change in WC and LAP from the lowest two quintiles to

Table-1: Comparison between LFnu and HFnu at lowest and highest quintiles of waist circumference (WC) (n =48) and Lipid accumulation product (LAP) (n = 44)

	Waist circumference		P< 0.05
	Lowest two quintiles 65 - 88 cm	**Highest two quintiles 98 - 126 cm**	
LFnu	47.7 ± 18.7	59.2 ± 18.5	0.03
HFnu	52.1 ± 18.6	40.7 ± 18.5	0.03
	LAP		
	Lowest two quintiles 3-32	**Highest two quintiles 54-194**	**P< 0.05**
LFnu	44.4 ± 15.0	57.1 ± 20.7	0.02
HFnu	55.4 ± 14.9	42.8 ± 20.6	0.02

LFnu: Low-frequency normalised units, HFnu: High-frequency normalised units.

the highest two quintile was associated with a significant increase in LFnu and a significant decrease in HFnu (Table 1).

A significant and positive correlation was found for LFnu with WHR and WHtR, while these relationships were significantly and inversely correlated with HFnu (Table 2). WHR and LAP were positively and significantly correlated with LF-HF ratio (p<0.05).

Table-2: Correlation of LFnu, HFnu and LH-HF ratio with adiposity indices (n = 53).

Frequency band	Waist-Hip Ratio	Waist-Height Ratio	LAP
LFnu power band	r = 0.34, P<0.021	r = 0.29, P<0.05	NS
HFnu power band	r = -0.34, P<0.02	r = -0.29, P<0.05	NS
LH/HF Ratio	r = 0.29, P<0.05	NS	r = 0.29, P<0.05

LFnu: Low-frequency normalised units, HFnu: High-frequency normalised units, LAP: Lipid accumulation product, NS: Non-significant correlation.

Discussion

The current study was conducted on female subjects alone because very few of the male volunteers met the inclusion criteria. Consequently, the gender effect as a confounding factor on HRV indices can be excluded.

Abdominal obesity was determined with a cut-off point of ≥80cm for increased cardiovascular risk in women[6]. The application of such cut-off values to the current data would divide them into two groups: one with LAP <44, and the other with LAP ≥44. To ensure consistency in the way of comparison, a quintile-based division of the data was adopted.

The interaction between the autonomic nervous system (ANS) and body fat has been the subject of numerous theories. One focuses on the influence of glucose on ANS behaviour modulation[8]. Another is based on the thermogenic adaptation, which prevents certain people from gaining weight, and seems to be attributed to ANS involvement[9]. A third possibility is that adipocytes handle the secretion of adipokines, including leptin and adiponectin. Leptin by itself was shown to trigger the activation of neuronal signalling pathways, resulting in sympathetic activity[10].

There is a connection between abdominal fat and the activity ratio between sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) in HRV assessment.[11] The reduction in PNS activity as one's body size increases is a defensive mechanism against fat deposition. Increased SNS tone can mitigate the obesity-inducing effects of excessive energy consumption[12].

Several studies have reported that changes in HRV indices because of central adiposity were more accurate in the assessment of body fat content than was body mass index (BMI). This is most likely since the BMI does not distinguish between fat and muscle compartments[13]. Alternative measures of abdominal adiposities, such as WC and WHR, have been suggested as preferable to BMI in predicting CVD risk[14]. This is based on the idea that abdominal adipose tissue secretes pro-inflammatory biomarkers and cytokines[15]. Type 2 diabetes, hyperlipidaemia and CVD are all linked to these inflammatory mediators[16]. According to a study, rising free fatty acids in circulation necessitates more insulin release from the pancreas to regulate the metabolism of glucose, and the resulting hyperinsulinaemia desensitises insulin-sensitive tissues, putting people at risk for type 2 diabetes[17].

The current findings showed that subjects having the highest WC had lower PNS activity and higher SNS. They also had more cardiac autonomic dysfunction. These results are consistent with earlier findings[18]. The current results revealed that WC, WHR and WHtR had a direct relationship with SNS and an inverse relationship with PNS, which is in complete agreement with previous analysis[19]. When comparing overweight/obese people to average-weight people, a study[20] found a significant increase in LFnu and LF-HF ratio, as well as a decrease in HFnu, indicating a change in the autonomic balance towards sympathetic overactivity and reduced parasympathetic tone in overweight/obese people. The inverse correlation of WHR and WHtR with HFnu suggests early cardiovascular vagal tone changes that are an important determinant of cardiovascular health that affects HR, cardiac activity and BP. People with low vagal tone are more likely to develop heart problems, including myocardial infarction (MI) and HTN[21].

In contrast to the present findings, a study identified a general decrease in ANS activities in children with obesity when compared to non-obese children having similar physical activity levels[22]. The discrepancy between the results is not clear. However, the earlier results[22] were based on a sample of schoolchildren aged 6-12 years, while the current sample had adult female volunteers.

In a handful of studies looking at the change in ANS behaviour after weight-loss in obese patients, a spike in HRV was noted after weight-loss through gastroplasty[23], increased physical activity and calorie restriction[24], and anti-obesity care[25]. It was suggested that these changes in HRV were likely due to physical exercise, and these changes in HRV could not be produced by any of the body composition or nutritional factors[25].

The current study also showed that an increase in LAP was associated with autonomic dysfunction, represented by an increase in SNS and a decrease in PNS activity. To the best of our knowledge, the current study is the first to shed light on the effect of WC-TG interaction on HRV indices.

Limitation: The current study has limitations as the sample size was not calculated which could have affected the power of the study.

Conclusion

An increase in LAP was associated with autonomic dysfunction that was marked by an increase in SNS and a decrease in PNS activity.

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Conflict of Interest: None.

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