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The Impact of Body Mass Index and Body Composition on Cardiac Autonomic Function in Young Adult Saudi Females

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Abstract. This work was carried out to elucidate the impact of body mass index, body fat and its distribution, body water and body muscles on the cardiac autonomic control in healthy young adult female students at King Abdulaziz University during 2012. Heart rate variability parameters were correlated with anthropometric measurements and body composition (fat, muscles and water). Time domain and frequency domain parameters of heart rate variability were decreased as the body mass index and percentage of body fat increase. In addition, these anthropometric measurements revealed significant positive correlations with the ratio of low frequency to high frequency components, pointing to the adverse effect of body fat on sympathovagal balance. Waist circumference, an index of central obesity, was negatively correlated with the number of pairs of successive NNs that differ by more than 50 ms. The percentage of body water showed a significant negative correlation with low frequency to high frequency ratio. Also, body muscles percentage represented a negative correlation with this ratio, though insignificant. Therefore, this study concluded that high indices of overall and central obesity seem to impair cardiac autonomic balance in healthy adult females.

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Additionally, increased body water and lean body mass are associated with favorable effects on cardiac autonomic function.

Keywords: Heart rate variability, Body mass index, Body composition, Obesity.

Introduction

The autonomic nervous system (ANS) directs involuntary physiologic processes such as digestion, hormonal regulation, energy metabolism, blood pressure and heart rate, and is therefore, considered an important regulator of homeostasis[1,2].

Heart rate control is determined by the dynamic interaction of both the acceleratory sympathetic nervous system, and the decelerator parasympathetic nervous system activation resulting in rhythmical oscillations; heart rate variability (HRV). This variability in heart rate provides a non-invasive tool to assess cardiac autonomic function. Reduced variability in heart rate reflects autonomic imbalance. This imbalance has been shown to be a predictor of the morbidity and mortality in risk prone individuals[3].

The alteration in cardiac autonomic nervous system, measured in terms of heart rate variability, is found to be related to body mass[4]. There is evidence that sympathetic activity has been enhanced in obesity[5] and an enhanced vagal tone was demonstrated in chronically undernourished subjects[6]. In addition, studies have shown association between HRV indices and body fat parameters. The high body fat percentage was associated with low sympathetic modulation of the heart rate in healthy adolescent/young adults, implying that body fat percentage determines cardiac sympathovagal balance in healthy subjects[7]. Moreover, central fat is associated with less favorable indices of HRV, suggesting that distribution of fat might be an important measure to assess the cardiac autonomic functions[8]. However, in a recent study carried out by Krishna *et al.*[9] on young healthy adults, body fat parameters did not show any significant relation to heart rate variability. Likewise, little is known about the relationship between HRV and the individual components of body composition.

Therefore, the aim of the present study was to investigate cardiac autonomic activity in young adult females in relation to their body mass index and body composition, to clarify the impact of body fat and body fat distribution, body water and body muscles on the cardiac autonomic control.

Subject and Methods

Study Subjects

This study is a cross sectional one correlating HRV parameters with anthropometric measurements and body composition (fat, muscles and water) in healthy young Saudi adult females. It was carried out in the lab of Department of Physiology at Faculty of Medicine, King Abdulaziz University during 2012. Twenty-six female university students (19-20 years-old) were enrolled in this study. All participants were consented; they were non-diabetic, non-hypertensive, with no history of cardiovascular diseases and were currently using no medications that might affect HRV.

Prior to ECG recording and HRV assessment through the Power Lab system, anthropometric measurements were obtained from each subject followed by determination of body composition.

Methods

Anthropometric Measurements

Height and weight were measured, in standing position while subjects wearing light clothes and no shoes, to the nearest half centimeter and kilogram, respectively. Waist circumference (WC) (midway between the lower rib margin and the iliac crest), and hip circumference (HC) (the maximal circumference over the buttocks) were also measured while standing, in cm[10]. Additionally, body mass index (BMI), weight (kg)/square of height (m^2), and waist to hip ratio (W/H) were calculated.

Body Composition Monitoring

Body composition was determined, using Gima body weight scale (Gima S.p.A., Milano, Italy), through measuring the flow of electrical signals (total body electrical resistance or impedance) as they pass through fat, lean (muscle, bones, vital organs) or water.

Age, height of the subject as well as being athlete or not were first entered before stepping on the scale bare footed for 10 seconds; immediately, body weight, fat, muscle and water percentages become calculated and presented on the screen.

Heart Rate Variability Assessment

ECG recording was performed using a ML 865 Power Lab system 4/25T (4 channels) with a built-in Bio Amp (through channels 3 and 4) to analyze HRV. The software version of the Power Lab was 5.5 and the 1st version of HRV analysis software was used.

Each subject was asked to lie in supine position, while all electromagnetic devices were kept away, and was connected to the Power Lab for about 1-3 minutes to be relaxed before recording. Recording was done for three and a half minutes while the subject was asked not to move. Two and half artifact-free consecutive minutes were used in the analysis. Files were imported into the software program for computation of standard time and frequency domains.

- Time Domain

Time domain parameters are associated mostly with the overall variability of the R-R intervals over the time of recording. They include: Standard deviation of all NN intervals (SDNN), square root of the mean of the sum of the squares of differences between adjacent NN intervals (rMSSD), the number of pairs of successive NNs that differ by more than 50 ms (NN50), as well as the heart rate (HR).

- Frequency Domain

The HRV signal is a slowly varying signal with a band width of 0-0.5 Hz. A high frequency component (HF; > 0.15Hz) reflects parasympathetic nervous activities and a low frequency component (LF; 0.04–0.15 Hz), mainly reflecting both sympathetic and parasympathetic nervous activities. In addition to analysis of these 2 frequencies, the ratio of low frequency to high frequency components (LF/HF) was estimated as a relative marker of sympathetic nervous activities or sympathovagal balance[11].

Statistical Analysis

All variables are presented as mean ± Standard Error of Mean (SEM). The one-sample Kolmogorov-Smirnov test was used to test for normality of variables. Those variables that were not normally distributed were transformed (normalized) and expressed in form of natural logarithms. The independent variables: Body weight, height, body mass index, waist circumference, hip circumference, waist/hip ratio, body fat, body water and body muscles were all assessed in Pearson's correlation analysis versus the frequency domain and time domain HRV parameters. P values below 0.05 were considered as statistically significant. SPSS windows version 20 (SPSS Inc., Chicago, IL USA) was used in the analysis.

Results

Table 1 shows the mean values (± SEM) of anthropometric measures namely body weight, height, body mass index, waist circumference, hip circumference, waist/hip ratio as well as the percentages of body fat, body water and body muscles. Table 2 presents the mean values (± SEM) of time and frequency domain parameters of heart rate variability. Table 3 shows the correlation coefficients between heart rate variability parameters and the different anthropometric measures and body composition.

As shown in Table 3, body weight exhibited a significantly negative correlation with SDNN, rMSSD and NN50, as well as with HF. Upon correlating LF/HF ratio to body weight, a significant

positive correlation was observed. Body height showed the same correlations observed with body weight, though only SDNN and rMSSD were statistically significant. Body mass index (BMI) showed a positive relationship with average heart rate, though not significant. Also, negative correlations were established between BMI and the time domain parameters SDNN, rMSSD and NN50, though only the correlation with the later one was statistically significant ($r = - 0.405$, $p < 0.04$). In addition, a significant positive correlation was demonstrated between BMI and the frequency domain parameter LF/HF ($r = 0.427$, $p < 0.03$) Table 3 and Fig. 1.

Table 1. Anthropometric characteristics and percentages of body fat, body water and body muscles of the study subjects.

	Mean ± SEM N = 26
Body Weight (Kg)	54.9 ± 2.12
Body Height (m)	1.56 ± 0.01
BMI (Kg/m^{-2})	22.6 ± 0.71
Waist Circumference (cm)	67.42± 1.74
Hip Circumference (cm)	94.46± 1.65
Waist-to-Hip Ratio	0.71 ± 0.01
Body Fat (%)	26.00 ± 1.35
Body Water (%)	52.01 ± 1.21
Body Muscles (%)	34.34 ± 0.47

Waist circumference (WC) was negatively correlated with SDNN, rMSSD and NN50, being significant only with the later ($r = -0.435$, $p < 0.03$), Table 3 and Fig. 1. This was accompanied with a non-significantly positive correlation with LF/HF. Similarly, hip circumference was negatively correlated with SDNN, rMSSD and NN50, and positively correlated with LF/HF, though they were all statistically non-significant. Likewise, the W/H represented the same non significant relationships, except for SDNN Table 3.

Table 2. Time domain and frequency domain parameters of heart rate variability in the study subjects.

	Mean ± SEM N = 26
Time Domain Parameters	
HR (beat min^{-1})	78.63 ± 1.15
In SDNN (ms)	4.29 ± 0.13
In rMSSD (ms)	4.33 ± 0.16
In NN50	3.48 ± 0.19
Frequency Domain Parameters	
In LF (ms^2)	7.12 ± 0.25
In HF (ms^2)	7.33 ± 0.29
In LF/HF	-0.21 ± 0.11

HR, heart rate: In natural logarithm; SDNN, standard deviation of all normal RR intervals; rMSSD, root mean square of successive differences; NN50, the number of adjacent cycles which are > 50 ms apart; LF, low frequency power; HF, high frequency power and LF / HF, ratio of the low / high frequency power.

Table 3. Correlation coefficients between heart rate variability parameters and body weight (BW), body mass index (BMI), height, waist circumference (WC), hip circumference (HC), waist/hip ratio (W/H) as well as the percentages of body fat, body water and body muscles.

	BW	**Body Height**	**BMI**	**WC**	**HC**	**W/H Ratio**	**Body Fat**	**Body Water**	**Body Muscle**
Time Domain Parameters									
HR (beat/min^{-1})	0.169	-0.012	0.195	0.131	0.064	0.151	0.211	-0.214	-0.143
In SDNN (ms)	-0.454*	-0.445*	-0.298	-0.225	-0.326	0.004	-0.271	0.262	0.219
In rMSSD (ms)	-0.471*	-0.469*	-0.303	-0.238	-0.299	-0.041	-0.280	0.274	0.202
In NN50	-0.395*	-0.132	-0.405*	-0.435*	-0.337	-0.343	-0.344	0.328	0.352
Frequency Domain Parameters									
In LF (ms^2)	-0.314	-0.335	-0.188	-0.154	-0.210	-0.012	-0.150	0.141	0.141
In HF (ms^2)	-0.436*	-0.358	-0.326	-0.255	-0.316	-0.059	-0.284	0.274	0.254
In LF/HF	0.434*	0.186	0.427*	0.137	0.351	0.127	0.402*	-0.396*	-0.343

**Significant correlation, $p < 0.05$*

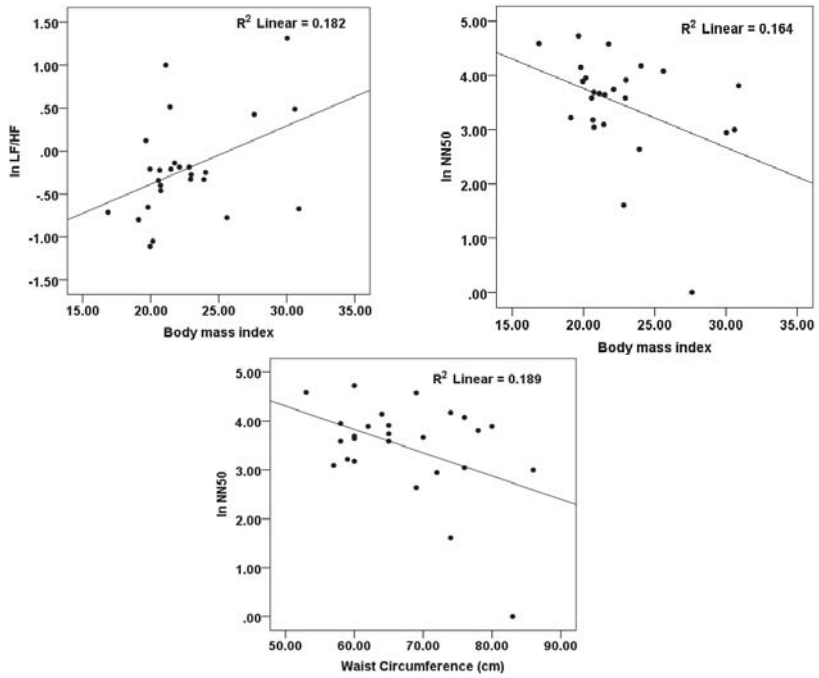

Fig. 1. Correlations between body mass index and waist circumference with the different HRV parameters.

As demonstrated in Table 3 and Fig. 2-A, the percentage of body fat showed a significant positive correlation with LF/HF ($r = 0.402$, $p < 0.04$) , that was associated with insignificantly negative correlations with SDNN, rMSSD, NN50 as well as HF, and insignificantly positive correlation with average heart rate.

In contrast to body fat percentage, the percentage of body water revealed a significantly negative correlation with LF/HF ($r = -0.396$, $p < 0.05$) (Table 3 and Fig. 2-B), and positively correlated with SDNN, rMSSD, NN50 as well as HF though not significant. The percentage of body muscles represented a negative correlation with LF/HF, and a positive one with NN50 that did not reach the level of significance ($r = -0.343$, $p < 0.08$ and $r = 0.352$, $p < 0.07$, respectively).

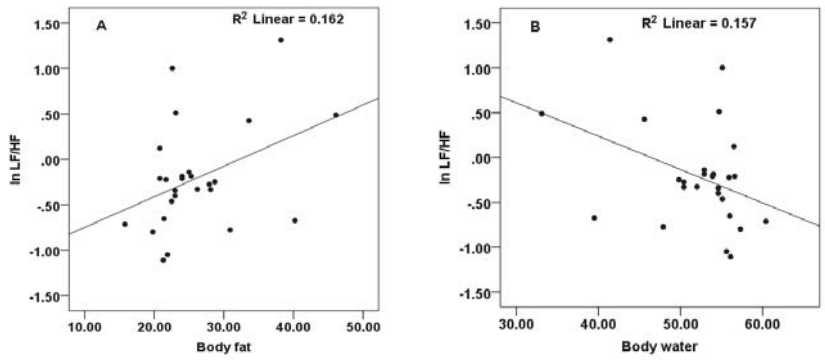

Fig. 2. Correlations between In LF/HF and each of the body fat (A) and body water (B).

Discussion

The present study was conducted to evaluate the relationship between HRV and body weight, body mass index, indices of body fat distribution as well as body composition to elucidate the impact of body fat and body fat distribution, body water and body muscles on the cardiac autonomic control in healthy young adult females.

The current results revealed that both time domain (SDNN, rMSSD and NN50) and frequency domain (HF) parameters of HRV were decreased as the BMI and its components (body weight and height) as well as the percentage of body fat increase, reflecting parasympathetic withdrawal and sympathetic predominance in association with increased adiposity. In addition, the significant positive correlation demonstrated in the present study between each of BMI and body fat percentage with LF/HF would point to the adverse effect of body fat on sympathovagal balance.

Such observed autonomic imbalance, with sympathetic overdrive and parasympathetic withdrawal is supported by the clear tendency of increasing heart rate in concomitance with increasing BMI and percentage of body fat. Hence, suggesting that increased body fat shifts the autonomic balance more towards the sympathetic

component. Therefore, it could be concluded that under resting conditions, individuals with high body fat tend to be more prone to have increased work load on the heart, due to sympathetic overdrive, with subsequent increment of heart rate.

The demonstrated significant negative correlations observed between body height and the time domain parameters SDNN and rMSSD are similar to those obtained with body weight, and are mostly due to the significant relation between body weight and height together with the dependence of autonomic nervous system modulation on body weight[12].

Consistent with our findings are many studies that reported parasympathetic withdrawal associated with sympathetic predominance in case of increased adiposity. Chen *et al.*[12] demonstrated that both body weight and body height were negatively correlated with HF and positively correlated with LF/HF. Furthermore, Rabbia *et al.*[5] detected a decrease in SDNN and rMSSD as the body weight increases in adolescent obese subjects. Such autonomic dysfunction was also reported in obese children[5,13,14]. A study carried out by Shetty *et al.*[15] demonstrated that women with increased BMI had significant changes in autonomic nerve function that included reduced parasympathetic control and elevated sympathovagal balance.

The diminished vagal modulation and the increased sympathetic activity observed in the present study with higher BW, BMI and the percentage of body fat could be explained by the role of adipose tissue-derived hormones, such as leptin and adiponectin that influence the ANS[16]. Additionally, von Känel *et al.*[17] reported that adiposity is associated with over-secretion of many adipokines and/or inflammatory markers, whose effects remain to be investigated. Leptin carries out its primary task of regulating food intake and energy homeostasis *via* increasing sympathetic nervous system outflow by binding to specific leptin receptors in the hypothalamus[18,19]. Also, excess leptin increases noradrenaline activity[20]. Moreover, Tanida *et al.*[21] suggested that adiponectin acts in the brain and suppresses renal sympathetic nerve activity.

Morbidly obese subjects were found to have high plasma levels of leptin, which stimulate the sympathetic nervous system[18] and low level of adiponectin that protects the cardiovascular system by modulating the cardiac vagal activity[22]. Moreover, Chen *et al.*[12] reported that in obese subjects the power spectrum of RR interval is shifted from HF to LF, mostly due to vagal withdrawal and high renin-angiotensin-aldosterone modulation.

In fact, the obesity associated loss of vagal protective reflex and diminished HRV indices represent a risk factor for *diabetes mellitus*, ventricular tachyarrhythmia and cardiovascular diseases. However, fortunately, the changes in HRV are reversible; weight reduction with decreased body fat has been linked to improved ANS function[4,23] and weight loss after increased physical activity, calorie restriction[24,25], gastroplasty[4], as well as anti-obesity medication[26] increases the HRV indices.

In contrast to the results obtained in the current study, some investigators reported different patterns of autonomic dysfunction. Peterson *et al.*[27] demonstrated HRV indices that reflect a lower sympathetic activity and higher parasympathetic activity in obese patients in comparison to normal weight controls. Also, Gao *et al.*[28] recorded higher sympathetic and parasympathetic drives in obese women, especially when there was a combination of upper body obesity and visceral obesity. Further, obesity was reported to be associated with decreased sympathetic responsiveness[27,29] and lower parasympathetic activity[23,30]. In addition, high BMI and body fat mass were found to be associated with a global reduction in cardiac autonomic nervous activity[31,32].

This controversy might be explained on basis of the discrepancy in age, sex or body fat distribution in subjects involved in these studies from those in the present one. Zhang *et al.*[33] demonstrated that age had a greater impact on HRV than sex. The older age group had consistently lower HRV than younger people. Windham *et al.*[34] reported that abdominal adiposity may contribute to poorer sympathetic and parasympathetic function as well. They added that abdominal adiposity, as opposed to overall adiposity tissue, could

adversely affect ANS function at younger ages with potentially unfavorable effects in later life.

The negative correlations demonstrated in the present study between the vagal modulation indices (SDNN, rMSSD, NN50 and HF) and the WC, HC as well as the W/H ratio support the hypothesis that the abdominal obesity adversely affects cardiac autonomic nervous system. This is in accordance with Chen *et al.*[12] who reported that abdominal obesity is pathophysiologically related to the vagal withdrawal and sympathetic overactivity, and consequently represents a risk factor for cardiovascular disease[35,36].

Many recent studies claimed that WC and W/H ratio, indicators of central obesity, are better than BMI, an index of overall obesity, in reflecting the extent of the morbidity and mortality of obesity[37,38]. Furthermore, it has been suggested that the W/H ratio is a much stronger predictor of myocardial infarction, angina pectoris, stroke[36] and death[39] as compared to BMI. Windham *et al.*[34] added that central adiposity induces premature aging of the ANS in young adults. Moreover, central fat affect HRV independently, irrespective of the difference in age, sex and cardiovascular risk specially in overweight subjects[40].

Limited data is available with regard to the mechanism by which central fat affects the cardiac autonomic activity. Hyperinsulinism observed in central obesity might be the cause of sympathetic overactivity and vagal withdrawal as reported in children with higher trunk fat[41,42]. Similarly, it was claimed that insulin resistance is perhaps the most important factor that impairs sympathovagal balance in favor of sympathetic over activity in obese children[43]. The adipose tissues are innervated by ANS and positively correlated to catecholamine production by ANS[44,45]. In addition, adipokines secreted by visceral fat may contribute to the autonomic dysfunction associated with central adiposity[46,47].

The current study revealed that, in contrast to the effects of fat percentage on HRV, body water percentage exhibited a significant negative correlation with LF/HF together with positive associations with SDNN, rMSSD and NN50. This could be explained by the

reciprocal relationship between fat and water; the more fat mass accumulation, the less water mass is present. Oshakbayev *et al.*[48] reported that as fat increases in the cells, it replaces molecular water; such reduction in intracellular water leads to disorder of nerve impulse conduction. Therefore, the detrimental effect of the decrease in body water on HRV might be mediated by the negative effect of the associated increase in body fat percentage on the cardiac autonomic function.

As regards the muscle percentage, the negative correlation demonstrated in the current study with LF/HF, as well as the positive associations observed with SDNN, rMSSD and NN50 represent a clue for the beneficial effect of muscle mass, on the expense of fat mass, on HRV. In accordance with this is a recent study carried out by Andrew *et al.*[49] who demonstrated that lower levels of markers for central adiposity and higher lean mass in the extremities predict higher levels of HRV in a population of police officers.

Limitations

Several potential limitations to our study should be considered. This study was carried only on young adult females, so that no conclusion could be generalized to involve the whole population. Also, phases of menstrual cycle were not considered in our results; however, a recent study[50] reported no significant relationship between the HRV parameters and the normal cyclic variation in sex hormones during menstrual cycle.

Furthermore, this study included a relatively small sample in a cross sectional analysis and therefore, the causal relationship could not be well established.

Although our findings relate the body fat and body fat distribution to HRV in healthy subjects, thus providing the opportunity for early detection of autonomic dysfunction in apparently healthy individuals and early intervention, our observations could not be generalized to persons in poorer health.

Conclusion

High indices of overall obesity particularly body fat percentage and body mass index, together with high indices of central obesity, WC, HC and W/H ratio, all seem to impair cardiac autonomic balance in healthy adult females. Our study depicted such cardiac autonomic dysfunction as parasympathetic withdrawal and sympathetic predominance that carries the risk of cardiovascular morbidity and mortality in later life. Additionally, increased body water and lean body mass are associated with favorable effects on cardiac autonomic function.

Recommendations

Our data suggest the importance of controlling obesity, especially the central type, at an early age to reduce the incidence of sympathovagal imbalance and consequently, the future occurrence of cardiovascular diseases.

Future Works

Future longitudinal studies are recommended to analyze the influence of changes in WC on autonomic nervous system activity at young ages and if they contribute to cardiovascular risk in late life. Moreover, the exact role of adipokines in mediating the effects of increased body fat on ANS function should be investigated.

Conflict of Interest

None declared.

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تأثير معامل كتلة الجسم و مختلف مكونات الجسم على المتغيرية فى نبضات القلب في السعوديات البالغات

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المستخلص. بالرغم من أن كتلة الجسم ومحتوى الجسم من الدهون وتوزيعها تؤثر على التوازن بين طرفي الجهاز العصبي اللاإرادي المغذي للقلب، فإن هناك الكثير من التضارب في نتائج الأبحاث في هذا المجال. تحرى هذا البحث تأثير معامل كتلة الجسم ومختلف مكونات الجسم من دهون وماء وعضلات، على المتغيرية في نبضات القلب في مجموعة من البالغات. وقد تم إجراء هذه الدراسة في قسم علم وظائف الأعضاء بكلية الطب جامعة الملك عبد العزيز في عام ٢٠١٢ بالاستعانة بطالبات جامعيات أصحاء. وقد تم دراسة ارتباط كلٍ من القياسات الجسمانية ومختلف مكونات الجسم مع مؤشرات المتغيرية في نبضات القلب. أظهرت النتائج وجود ارتباط عكسي بين كلٍ من مؤشر كتلة الجسم ونسبة الدهون في الجسم ومؤشرات المتغيرية في نبضات القلب سواء الخاصة بالوقت أو التردد، وكذا بالنسبة لمحيط الخصر مع مؤشرات المتغيرية في نبضات القلب الخاصة بالوقت. أما نسبة الماء والعضلات بالجسم فقد أوضحت الدراسة وجود ارتباط عكسي مع مؤشرات المتغيرية في نبضات القلب

الخاصة بالتردد (LF/HF ratio). وعلى ذلك فإنه يمكن استنتاج أن زيادة مؤشرات السمنة – سواء الكلية أو المركزية– تؤدي إلى تدهور في توازن الجهاز العصبي اللاإرادي على وظيفة القلب في الإناث البالغات. بالإضافة لذلك فإن هذا التوازن يتحسن بزيادة كلٍ من نسبة الماء والعضلات بالجسم.