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A DESCRIPTIVE RESEARCH OF DECREASED HEART RATE VARIABILITY AS A RISK FACTOR FOR CARDIOVASCULAR DISEASE IN YOUNG, HEALTHY ADULTS WITH LOW AND HIGH BMI INDEXES.

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ABSTRACT

Background and objectives: HRV is used to evaluate autonomic nervous system input to the heart. Several studies have examined the effect of HRV on underweight status. To assess HRV in age-matched young people with varying BMI categories.

Methods: This cross-sectional study was conducted on healthy young adults between the ages of 18 and 25. Variables of anthropometrics were measured. ECG was recorded for 5 minutes in lead II setup. Kubios HRV analyzer was used to evaluate heart rate variability.

Results: HRV indices were lower in the underweight, overweight, and obese BMI groups compared to the normal weight group. Both genders exhibit an inverted U-shaped association between BMI and HF log power based on second-order polynomial regression. The correlation between BMI, waist circumference, and body fat (%) and HRV indices demonstrates a substantial relationship with heart rate variability, with waist circumference (WC) exhibiting a stronger relationship than BMI. Comparing HRV parameters between men and women of different BMI groups reveals that women exhibited more heart rate variability than men across BMI groups.

Conclusion: Underweight individuals share the same elevated cardiovascular risk as the obese, and abdominal obesity is a more accurate predictor of cardiovascular risk than the body mass index (BMI).

Keywords: Heart Disease Risk Factors, Thinness, Overweight, Heart Rate Variability, Waist Circumference.

INTRODUCTION:

Heart rate variability (HRV) is a non-invasive indicator of cardiac autonomic activity; a rise in HRV reflects the heart's adaptability to stress, whereas a decrease in HRV is connected with cardiac arrhythmias and untimely deaths. Many cardiac autonomic research are focused on obesity, yet underweight individuals are frequently ignored. The Indian population has a high prevalence of both underweight (20.9% of males and 22.2% of females) and overweight/obesity (18.9% of males and 20.6% of females) [1,2].

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On the basis of the body mass index (BMI) and waist circumference, obesity-related health risks are quantified (WC). A higher BMI is connected with a greater health risk, but WC is an indicator of belly fat content. Obesity has been the primary focus of research on autonomic function, fuelled by either the hypothesised role of increased sympathetic activity in obesity-related cardiovascular illness or the potential role of decreased sympathetic activity in the genesis of obesity. In contrast to the abundance of research on autonomic function and obesity, there is a dearth of information on underweight (UW) people [3,4]. As physiological variation in the time interval between heartbeats is controlled by the autonomic nervous system, the study of this variation is regarded as an indirect measure of cardiac autonomic function, the so-called heart rate variability (HRV), and is a valuable tool for evaluating sympathetic and parasympathetic modulation of the heart [4].

The link between HRV and BMI in obese patients has been explored repeatedly with contradictory results. Due to its metabolic activity, it has been hypothesised that central body fat distribution may be more relevant than total adiposity in explaining such conflicting evidence. Regardless, there are still a dearth of studies examining this link with UW patients. As previously observed, undernourished individuals exhibited a decrease in HRV. Subsequently, identical findings were verified in slim young men, but not in the elderly, confirming that age and gender may play a significant role in HRV evaluation [4,5]. Earlier research into the relationship between HRV and BMI revealed a drop in HRV in obese patients compared to normal weight (NW) subjects, but no changes between UW and NW subjects. However, the authors made no comparisons between UW and obese participants. Among a recent study, HRV frequency characteristics were found to be lower in obese young people. A comparable drop was also observed in the UW group, but it was not statistically significant (perhaps due to the limited sample size, according to the authors). These findings appear to provide light on a putative connection between HRV and obesity. Therefore, we examined the possibility of a correlation between short-term resting HRV recordings and body fat mass (FM) in UW, NW, and overweight healthy adult women (based on BMI) [6].

The relationship between BMI and HRV is contentious. Visceral or abdominal adipose tissue is biologically active, indicating that measuring fat distribution is more essential than measuring obesity as a whole. Asian Indians have thinner limbs and less muscle mass, but are obese in the midsection. Waist Circumference, a surrogate for body fat, is particularly sensitive to the distribution of body fat and body size and corresponds with BMI.Underweight, normal, overweight, and obese young adults and compare the strength of the connection between HRV and waist circumference with that between HRV and body mass index [6,7].

MATERIAL AND METHODS:

A cross-sectional study was conducted on 150 young adults (90 males and 60 females) between the ages of 18 and 25 who were recruited using a suitable random sample technique from January 2021 to January 2022 at Department of Community Medicine, Santhiram Medical College, Andhra Pradesh, India. Using the mean standard deviation of the HRV parameter LFnu for normal weight subjects of 40.35 ±17.52 and the mean standard deviation of the HRV parameter LFnu for overweight subjects of 47.75 ±15.76 from a study conducted on young Indian adults, the sample size was calculated using open Epi software with a confidence interval of 95% and 80% power.

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All participants provided written informed consent for their voluntary involvement in the study. The participants were instructed not to consume beverages containing caffeine or alcohol and to refrain from physical activity 24 hours prior to examinations [7,8]. Evaluations were conducted on female participants during the early follicular phase of their menstrual cycles. All ECG recordings were performed in the Autonomic function testing laboratory. Anthropometric characteristics such as height, weight, waist circumference, hip circumference, and skinfold thickness were measured. Body Mass Index was determined by dividing weight in kilogrammes by height in metres squared. Subjects were categorised according to their BMI as Underweight (UW), Normal Weight (NW), Overweight (OW), and Obese. The waist-to-hip ratio was determined using the formula (waist circumference/hip circumference). Using Durnin and Womersley's equation and the skinfold thickness of the triceps, suprailiac, and sub- scapular regions, the percentage of body fat was determined. After five minutes of rest, blood pressure (BP) and heart rate were measured in the right arm using a digital BP device while the subject was seated [8,9]. An ECG recorded in a temperature- and noise-controlled room. Kubios HRV analyzer was used to assess the heart rate variability. SPSS 17.0 (IBM Corp., Armonk, NY, USA) software was used to conduct statistical analysis. The Shapiro–Wilk test was performed to assess data normality. Levene's test for homogeneity of variance was performed on the entire sample and was insignificant ($p>0.05$) [9,10]. Using ANOVA, the differences between the groups were evaluated. The Post Hoc Tukey test was performed. Pearson association between HRV indices and BMI, waist circumference, and fat percentage in males and females. Using an Independent T-test, gender differences in HRV indices were evaluated.

Inclusion criteria:

1. Healthy volunteers between the ages of 18 and 25.

Exclusion criteria:

1. Subjects having a known history of hypertension, cardiovascular disease, diabetes, smoking, endocrine disorders, autonomic neuropathies, and those receiving long-term steroid, anticholinergic, sympathomimetic, or para sympathomimetic medication therapy.

RESULT:

Table1:CharacteristicsofstudygroupbasedonBMI(Mean±SD)

Parameter	**Underweight**	**Normal weight**	**Overweight**	**Obese**	**P value**
	(n=37)	**(n=36)**	**(n=39)**	**(n=38)**	**(ANOVA)**
Age(years)	19.50 ±1.58	19.65±2.11	20.48±2.17	19.53±1.94	0.089
Weight(kg)	47.40±6.3	59.53±7.48	70.90±7.66	90.75±13.48	0.000*
Height(Cm)	163.83±9.63	165.08±8.22	163.65±8.56	163.10±10.3	0.805
BMI(Kg/m^2)	17.56±0.94	21.92±1.18	26.41±1.08	34.09±3.86	0.000*

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Waistcircumference(Cm)	65.25±4.80	75.43±5.75	84.13±9.02	100.45±10.48	0.000*
W/Hratio	0.78±0.04	0.80±0.04	0.84±0.72	0.87±0.57	0.000*
Body Fat percentage	18.78±6.22	23.98±6.93	27.88±6.09	31.87±6.42	0.000*

*Statisticallysignificantvalues

Thefourgroupsaresimilarbyagebutsignificantlydifferentby BMI,waistcircumferenceandbodyfat%.

Table2: Heartrate variabilityindicesindifferentstudygroupbasedonBMI(Mean±SD)

Parameter	**Underweight (n=37)**	**Normal weight (n=36)**	**Overweight (n=39)**	**Obese (n=38)**	**P value (ANOVA)**
Heart rate (bpm)	80.75±9.56	80.75±11.05	81.48±12.61	90.98±9.92	0.007*
Systolicblood pressure(SBP)	106.75±10.5	112.05±10.13	119.53±10.62	129.53±10.7	0.000*
Diastolicbloodpressure(DBP)	72.80±7.81	76.80±7.6	81.63±6.8	87.93±6.0	0.000*
SDNNlog	1.81±0.14	1.87±0.1	1.76±0.086	1.67±0.09	0.000*
RMSSD log	1.89±0.10	1.96±0.13	1.86±0.18	1.78±0.15	0.000*
HF logpowern.u	1.69±0.08	1.76±0.07	1.74±0.059	1.63±0.09	0.000*
LF logpowernu	1.68±0.10	1.602±0.12	1.63±0.08	1.74±0.07	0.000*
LF/HF	0.9±0.18	0.8±0.23	1.6±0.15	1.9±0.13	0.006*

*Statisticallysignificantvalues

Heart rate was elevated in the obese group. Post hoc Tukey analysis revealed no significant difference in Heart rate across the UW, NW, and OW groups. The OW and obese groups have significantly higher systolic and diastolic blood pressure (p0.05) compared to the Normal BMI group. Post hoc Tukey test revealed no difference between the UW and Normal groups in systolic and diastolic blood pressure (p> 0.05).

Table3:Association of HR Vindices with BMI, waist circumference and % fatinmales and females

Parameter	**HFlog Power(n.u.)**		**LF logPower(n.u.)**		**SDNN log**		**RMSSD log**	
	Male (n= 90)	Female (n= 60)	Male (n= 90)	Female (n= 60)	Male (n=90)	Female (n=60)	Male (n=90)	Female (n= 60)
BodyMassIndex(BMI)	0.38**	0.38**	0.33**	0.37**	0.23*	0.60**	0.36**	0.29**
Waist Circumference	0.51**	0.46**	0.43**	0.50**	0.25*	0.60**	0.43**	0.34**

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%Fat	0.34**	0.27*	0.29**	0.28*	0.25*	0.55**	0.38**	0.32**

**Significantatlevelof 0.01;*Significantatlevel of0.05

SDNN log, male (r2= 0.287, F=13.668, P0.01), and female (r2= 0.383, F=23.937, P0.01), RMSSD log, male (r2= 0.187, F=8.109, P0.05), and female (r2= 0.197, F=9.675, P0.05), and BMI. Male LF log power nu (r2=0.267, F=14.005, P0.01) and female LF log power nu (r2=0.225, F=11.197, P0.01) showed a U-shaped association with BMI, showing that greater values were dispersed at the edges. Significant linear regression had a lower r2 value than second-order polynomial regression (quadratic) studies.

Table 4:Genderdifferencein HRVindices ofdifferentBMI groups

Parameter	Underweight		Normal weight		Overweight		Obese	
	Male (n= 19)	Female (n=18)	Male (n= 18)	Female (n=18)	Male (n=20)	Female (n=19)	Male (n=19)	Female (n=19)
HF logpowern.u	1.66	1.72	1.736	1.79	1.71	1.77	1.61	1.66
	±0.06	±0.09**	±0.06	±0.06**	±0.054	±0.044**	±0.08	±0.09**
LF logpowernu	1.71	1.64	1.64	1.562	1.67	1.591	1.76	1.71
	±0.06	±0.12**	±0.09	±0.11**	±0.062	±0.069**	±0.57	±0.08**

**significantat level of0.01

All variables have a substantial relationship with heart rate variability, with WC having a stronger relationship with HRV indices than BMI. Comparing HRV parameters between men and women of various BMI groups

DISCUSSION:

The time domain metric SDNN (standard deviation of NN intervals) is influenced by both SNS and PNS activity, and so represents an approximation of HRV. RMSSD (Root mean square of successive RR interval differences) represents the beat-to-beat variance in HR and is the principal time-domain measure used to assess the vagally-mediated alterations reflected in HRV. Frequency domain characteristics, HF (High frequency) component is primarily due to vagal activity, whilst LF (Low frequency) component indicates baroreceptor activity during resting conditions, and LF/HF estimates the ratio between SNS and PNS activity [10,11].

Comparing the Normal BMI group to the UW, OW, and obese BMI groups, the temporal domain and frequency domain parameters of HRV are significantly altered. Elevated sympathetic inputs and lower vagal regulation are associated with diminished HRV indices, indicating increased risk factors for cardiovascular disease, not only in overweight and obese individuals but also in underweight individuals. Obesity is a well-known cardiovascular disease (CVD) risk factor, but the underweight population with a body mass index (BMI) below 18.5 kg/m2 has not been a cause for concern. In a study by Triggiani et al., participants from the University of Washington demonstrated a substantial drop in SDNN, LF, and TP

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values, but not in HF values, indicating decreased vagal regulation of the heart [11,12]. A research by Vaz et al. revealed a drop in HRV parameters in persons with UW and malnutrition. Sowmya et al. also demonstrated that an increase in BMI in Indians with a low BMI increased the risk of cardiovascular morbidity and mortality. Our findings contradict the findings of Krishna et al, Schmid et al, and Wu et al, who found no drop in HRV indices in UW participants [12,13].

Similar to our study, Rossi et al. demonstrated a significant decrease in the RMSSD, HFms, and HFnu indices in the obese group, indicating a decrease in vagal activity. Furthermore, the high value of the LFnu index in the obese group indicated relative sympathetic predominance in obese individuals compared to normal-weight individuals. Shroney et al. observed a drop in HF nu and an increase in LF nu as well as an increase in the LF/HF ratio. Our study differed from theirs in that 40- to 50-year-old male individuals were included and Asian BMI cutoff criteria were considered, resulting in a different BMI. Compared to the normal weight group, Rajalakshmi et al. noticed a substantial drop in overall power, high frequency power, and an increase in low frequency power [14,15]. Similarly, Chetan et al. found diminished parasympathetic activity and higher sympathetic activity in the obese group of male individuals. According to Rohini et al., there were no predictable changes in cardiovascular autonomic activity as evaluated by HRV in the obese population, nor were there any gender variations in autonomic function. In our investigation, there was no difference between the UW and normal groups in terms of sympathovagal balance, however the OW and obese groups had increased LF/HF ratios, indicating greater sympathetic activity. Jain et al. demonstrated that LF/HF was greater in the overweight and obese group. Schmid et al. found no difference between underweight and normal weight in the LF/HF ratio. In contrast to our findings, Millis et al. found a correlation between high body fat and increased vagal activity, as well as a substantial negative correlation between body fat and LF/HF ratio. This discrepancy could be related to the smaller sample size of their investigation. Lower adaptive flexibility of heart rate variability in UW, OW, and obese persons compared to NW subjects may represent risk factors for cardiovascular disease mortality [15,16].

Evidence suggests that energy restriction is related with a reduction in triiodothyronine levels, which is associated with altered HRV, and that treatment for hypothyroidism reverses these HRV alterations. In our investigation, we excluded participants having a history of endocrine illnesses; hence, the change in HRV in our study may not be attributed to thyroid disorders. Other possible causes of low HRV in underweight populations include dietary and nutritional inadequacies, particularly vitamin B12 insufficiency, and therapy with this vitamin corrected the HRV deficit. There is evidence from animal models that prenatal malnutrition reduces sympathetic innervation, at least in the gut, over the long run. In prenatal nutritional inadequacies may also reflect changes in autonomic abnormalities that have occurred in undernourished adults [16,17]. Cardiovascular risk in persons with a low BMI may also be attributable to increased visceral adiposity or ectopic fat, as opposed to subcutaneous fat. Future research must determine the precise processes behind the modification of autonomic function in UW individuals.

Obesity increased sympathetic activity and decreased parasympathetic (vagal) tone, indicating that obese persons have inadequate control of their autonomic heart rhythm. Based on the sympathetic-adrenergic and baroreflex functions associated with obesity, the

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sympathetic overactivity of obese people may be explained. Unknown is the mechanism behind these alterations in parasympathetic and sympathetic nerve activity in the overweight and obese. Among the hypothesised hormonal signals are leptin, leptin resistance, Insulin, insulin resistance, free fatty acids, and additional processes such as duration of obesity, fat distribution, etc [17,18].

In general, the BMI has been used to diagnose underweight and overweight issues. Based on the results of this study, it is evident that waist circumference has a stronger relationship with HRV markers than BMI, which modulates cardiac autonomic activity. The higher link between waist circumference and HRV indices than between BMI and HRV indices may be related to the abdominal obesity prevalent in the Indian population. The BMI does not distinguish between fat mass and fat-free mass. The other metric that reflects abdominal obesity is waist circumference (WC), which, according to our study, is superior to BMI in predicting CVD risk. Thus, markers of central obesity are more sensitive than global obesity indicators. Increased waist circumference, which suggests visceral obesity, was highly related with decreased parasympathetic and increased sympathetic heart activity. In the Indian population, it would be preferable to regard waist circumference as a greater indicator of obesity than BMI [18,19]. This study demonstrates that abdominal fat has a stronger negative association with HRV indices, suggesting that fat distribution may be a relevant metric for assessing the cardiac autonomic functioning. Despite the fact that our study groups were healthy adults without comorbidities other than being underweight and overweight, they are at risk for Cardiovascular morbidity. Even in those with a normal body mass index, it appears crucial to measure the degree of central fat distribution in order to predict future health hazards early on.

The obese group had a considerably greater heart rate compared to the normal weight group. Other investigations support the observation of tachycardia in obese individuals, which is caused by impaired autonomic regulation of the intrinsic heart rate. Our study confirmed that both SBP and DBP were significantly higher in the OW and obese groups compared to normal weight controls, which is consistent with several other studies indicating autonomic impairment in obese individuals, characterised by a decrease in parasympathetic activity and a relative predominance of sympathetic activity. The increase in blood pressure could be due to the direct influence of obesity on hemodynamics, such as an increase in blood volume, stroke volume, and cardiac output, as a result of the resistance presented to the circulatory system by increased adiposity. In addition to the increased peripheral vascular resistance caused by obesity, obesity-mediated inflammatory consequences such as endothelial dysfunction and cytokine impact were also implicated in the aetiology of obesity-related cardiovascular sequelae. This can be linked to the influence of female sex hormones on autonomic activity, since HRV indices are assessed during the early follicular phase of the menstrual cycle, when endogenous oestrogen levels are elevated [19].

Estrogen possesses cardiosympathoinhibiting and vagotonic properties. Young adult females may exhibit less sympathetic activity than young adult males due to hormonal considerations. In addition to oxytocin, neural control, and differential stress coping, these gender differences have a functional basis in oxytocin, neural control, and neural control. This may explain why males have a greater risk of cardiovascular disease than females [19,20]. In our study, persons with low and high BMI had reduced HRV, which is a sign of an increased risk of

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cardiovascular morbidity and mortality due to cardiac autonomic changes. Our study has a drawback in that we did not examine the physical activity of the subjects, which can alter cardiac autonomic function.

CONCLUSION:

HRV indices are lower in the UW, OW, and obese BMI groups compared to the normal BMI group, and a U-shaped relationship exists between BMI and HRV indices. We discovered that not only those with a higher BMI but even those with a lower BMI have an elevated cardiovascular risk, even when asymptomatic and without comorbidities. Additionally, additional research is required to confirm the pathophysiology underlying the reduced HRV values in the underweight group. In the overweight and obese population, the sympathovagal balance is skewed toward sympathetic dominance, which is also a risk factor for cardiac morbidity. The link between waist circumference and HRV indicators is greater than that between BMI and HRV markers, suggesting the necessity for measurement of central adiposity in addition to general adiposity, which aids in early diagnosis of risk factor. We propose that keeping a healthy weight is essential for lowering cardiovascular risk in UW and OW adults.

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