RESEARCH PAPER

Cardiac Autonomic Modulation across Body Mass Index Categories in Reproductive-Age Women: A Cross-Sectional Heart Rate Variability Study

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ABSTRACT

Objectives: To compare short-term heart rate variability (HRV) parameters among normal-weight, overweight and obese women aged 20–40 years.

Materials and Methods: This cross-sectional study included 90 women classified according to Asia-Pacific body mass index (BMI) criteria as normal weight (18.5–22.9 kg/m²), overweight (23.0–24.9 kg/m²) or obese (≥25.0 kg/m²), with 30 in each group. Five-minute supine lead-II electrocardiogram recordings were analysed for LF nu, HF nu, LF/HF ratio and mean heart rate. Groups were compared using one-way analysis of variance followed by Tukey's honestly significant difference post hoc test.

Results: Age was comparable among the groups (P = 0.932). LF nu increased progressively from 58.99 ± 8.10 in normal-weight women to 63.83 ± 3.52 in overweight women and 66.39 ± 5.99 in obese women. HF nu decreased from 41.90 ± 6.03 to 38.50 ± 3.79 and 34.66 ± 6.67, respectively. The LF/HF ratio increased from 1.45 ± 0.31 to 1.68 ± 0.26 and 2.00 ± 0.46, while mean heart rate increased from 80.10 ± 5.25 to 85.70 ± 6.64 and 91.60 ± 6.56 beats/min, respectively. Overall differences were significant for all four outcomes (P < 0.001).

Conclusion: Increasing BMI was associated with graded differences in frequency-domain HRV indices and resting heart rate among reproductive-age women. These differences were evident from the overweight category, although prospective studies are required to establish their predictive and clinical significance.

Keywords: Autonomic nervous system, cardiac autonomic modulation, heart rate variability, obesity, overweight

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INTRODUCTION

Overweight and obesity now affect more than a third of the world's adult population, and their prevalence continues to climb in low- and middle-income countries, including India, where nationally representative data indicate that abdominal obesity affects approximately 40 per cent of women.[1] Beyond established links to insulin resistance, hypertension, and dyslipidaemia, excess body weight is increasingly recognised as a contributor to autonomic nervous system dysfunction, characterised by altered sympathetic and parasympathetic modulation.[2]

Heart rate variability (HRV), the beat-to-beat variation in the interval between successive heartbeats, offers a non-invasive measure of cardiac autonomic modulation. Reduced HRV has been associated with a higher risk of cardiovascular events in population-based studies,[3] and short-term HRV assessment provides a practical method for evaluating autonomic modulation.[4] While several studies have described autonomic differences in obese cohorts, comparatively few have examined the trajectory of these differences across the overweight-to-obesity spectrum specifically in women, in whom sex hormones have been reported to influence autonomic tone.[2]

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This study was designed to characterise short-term frequency-domain HRV parameters across normal-weight, overweight, and obese women aged 20–40 years, to determine whether autonomic differences emerge gradually with increasing adiposity or are confined to obesity. Restricting the analysis to women is relevant because HRV is known to differ by sex and time of day, with large-scale reviews confirming meaningful sex-specific patterns in autonomic regulation that are frequently overlooked in mixed-sex cohorts.[5,6]

MATERIALS AND METHODS

This cross-sectional study was conducted over one year (September 2020–August 2021) in the HRV Research Laboratory, Department of Physiology, Madha Medical College and Hospital, Kovur, Chennai, after approval by the Institutional Ethics Committee, permission from the Dean and the Head of the Department of Physiology, and written informed consent from all participants. This study complies with the STROBE statement for cross-sectional studies.

Ninety women aged 20–40 years with regular menstrual cycles were recruited using convenience sampling from female attenders accompanying patients to the General Medicine outpatient department. Participants were categorised according to the Asia-Pacific BMI criteria as normal weight (18.5–22.9 kg/m²), overweight (23.0–24.9 kg/m²) or obese (≥25.0 kg/m²), with 30 participants in each group. A fixed sample of 90 participants, comprising 30 participants in each BMI category, was included. A formal a priori sample-size calculation was not documented. The study should therefore be regarded as exploratory, and the precision of the estimates is presented using 95% confidence intervals.

Women below 20 or above 40 years of age, and those with a BMI below 18.5 kg/m², were excluded, along with women with diabetes mellitus, hypertension, coronary artery disease, dyslipidemia, thyroid or other endocrine disorders, renal failure, abnormal hepatic function, pregnancy, current smoking or alcohol use, a history of intake of drugs known to affect autonomic function or prolong the QT interval (such as antiarrhythmics, quinidine, diuretics, digoxin, sotalol, or cisapride), oral contraceptive use, or menstrual irregularity (Figure 1).

After explaining the purpose of the study to each participant in her native language and obtaining written informed consent, demographic and socioeconomic details were recorded, along with a brief history of lifestyle, physical activity, dietary habits, and any chronic illness. General physical examination, including vital signs, and a systemic examination were performed for every participant. Height was measured barefoot using a stadiometer and weight using a standardised weighing machine; BMI was calculated using Quetelet's index (weight in kilograms divided by height in metres squared). Waist and hip circumference were measured in centimetres using a non-stretchable measuring tape, and the waist-hip ratio was calculated.

Five-minute supine lead-II electrocardiogram recordings were obtained between 9 a.m. and 12 p.m. using a Polyrite version 2.2D data-acquisition system with software version 2.5.2, in accordance with the Task Force guidelines of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.[4] Recordings were performed in a quiet room maintained at 25–28°C, 2–4 hours after breakfast. Participants were asked to empty their urinary bladder, switch off mobile phones and other nearby electrical devices, and rest comfortably in the supine position. Electrode sites were made oil-free using an alcohol prep pad, conductive gel and clip-type electrodes were used to maintain adequate skin contact. Participants remained awake with their eyes closed and avoided movement during recording. A five-minute segment showing normal sinus rhythm without artefacts was retained for Fast Fourier Transform analysis. LF nu was calculated as LF/(total power − very-low-frequency power) × 100 and HF nu as HF/(total power − very-low-frequency power) × 100. LF nu was interpreted as a frequency-domain measure influenced predominantly by sympathetic modulation, whereas HF nu was interpreted as predominantly reflecting cardiac vagal modulation. The LF/HF ratio was reported as a conventional index of relative autonomic modulation, while recognising that it is not a direct measure of sympathovagal balance.[4,7]

Data were analysed using SPSS version 20 (IBM Corp., Armonk, NY, USA) and expressed as mean ± standard deviation (SD). Differences among the three BMI groups were assessed using one-way analysis of variance (ANOVA), followed by Tukey's honestly significant difference post hoc test for pairwise comparisons. A two-sided P value <0.05 was considered statistically significant.

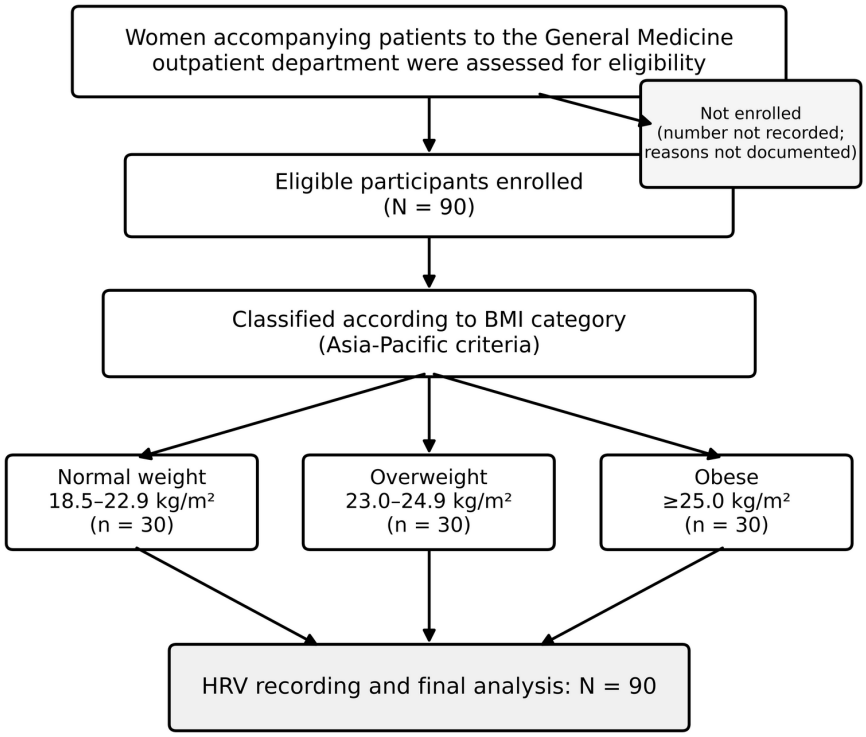


Figure 1. Study flow diagram. Women accompanying patients to the General Medicine outpatient department were assessed for eligibility. The total number screened and reasons for non-enrolment were not prospectively documented. Ninety eligible women were classified as normal weight, overweight or obese according to Asia-Pacific BMI criteria (n = 30 per group); all underwent HRV recording and were included in the final analysis.

RESULTS

Weight, height, BMI, waist circumference, hip circumference and waist–hip ratio differed significantly across groups (Table 1).

Table 1. Anthropometric characteristics according to BMI category (mean ± SD)

Parameter	Normal weight (n=30)	Overweight (n=30)	Obese (n=30)	F(2,87)	P value
Age (years)	26.87 ± 4.85	27.33 ± 5.58	27.30 ± 5.65	0.071	0.932
Weight (kg)	58.00 ± 6.97	70.47 ± 5.14	81.13 ± 10.62	64.189	7.5×10^{-18}
Height (m)	1.66 ± 0.09	1.72 ± 0.06	1.68 ± 0.09	4.24	0.0172
BMI (kg/m²)	20.92 ± 0.99	23.84 ± 0.54	28.56 ± 2.57	169.678	9.42×10^{-31}
Waist (cm)	77.90 ± 5.18	85.10 ± 4.25	100.83 ± 9.81	87.787	1.35×10^{-21}
Hip (cm)	89.83 ± 7.48	97.07 ± 4.14	106.20 ± 8.50	41.675	2.02×10^{-13}
WHR	0.83 ± 0.05	0.88 ± 0.05	0.94 ± 0.09	21.096	3.39×10^{-8}

Values are mean ± SD. Overall comparisons were performed using one-way ANOVA. BMI, body mass index; SD, standard deviation; WHR, waist–hip ratio.

HRV parameters differed progressively with increasing BMI category (Tables 2 and 3, Figure 2). LF nu and heart rate increased, whereas HF nu decreased, from the normal weight to the overweight and obese groups. The LF/HF ratio was approximately 38 per cent higher in the obese group than in the normal-weight group. Overall one-way ANOVA results are shown in Table 2, and Tukey-adjusted pairwise comparisons with 95% confidence intervals are shown in Table 3.

Table 2. Heart rate variability parameters according to BMI category (mean ± SD)

Parameter	Normal weight (n=30)	Overweight (n=30)	Obese (n=30)	F(2,87)	P value
LF nu	58.99 ± 8.10	63.83 ± 3.52	66.39 ± 5.99	11.173	4.8×10^{-5}
HF nu	41.90 ± 6.03	38.50 ± 3.79	34.66 ± 6.67	12.407	1.82×10^{-5}

LF/HF ratio	1.45 ± 0.31	1.68 ± 0.26	2.00 ± 0.46	17.835	3.23×10^{-7}
Heart rate (beats/min)	80.10 ± 5.25	85.70 ± 6.64	91.60 ± 6.56	25.965	1.44×10^{-9}

Values are mean ± SD. Overall comparisons were performed using one-way ANOVA. LF, low frequency; HF, high frequency; nu, normalised units; SD, standard deviation.

HF nu was lower in the obese group than in the normal-weight and overweight groups; the difference between normal-weight and overweight women was not statistically significant after Tukey adjustment (Table 3).

The LF/HF ratio and mean heart rate differed significantly across all three Tukey-adjusted pairwise comparisons (Table 3).

Table 3. Tukey HSD pairwise comparisons of heart rate variability parameters

Parameter	Comparison	Mean difference*	95% CI	Adjusted P value
LF nu	Normal weight vs overweight	4.84	1.05 to 8.64	0.00859
LF nu	Normal weight vs obese	7.41	3.61 to 11.20	3.42×10^{-5}
LF nu	Overweight vs obese	2.56	−1.23 to 6.36	0.247
HF nu	Normal weight vs overweight	−3.40	−6.87 to 0.07	0.0562
HF nu	Normal weight vs obese	−7.24	−10.71 to −3.77	9.55×10^{-6}
HF nu	Overweight vs obese	−3.84	−7.31 to −0.38	0.0261
LF/HF ratio	Normal weight vs overweight	0.23	0.01 to 0.45	0.0353
LF/HF ratio	Normal weight vs obese	0.55	0.33 to 0.77	1.64×10^{-7}
LF/HF ratio	Overweight vs obese	0.32	0.10 to 0.53	0.00269
Heart rate (beats/min)	Normal weight vs overweight	5.60	1.79 to 9.41	0.00205
Heart rate (beats/min)	Normal weight vs obese	11.50	7.69 to 15.31	5.92×10^{-10}
Heart rate (beats/min)	Overweight vs obese	5.90	2.09 to 9.71	0.0011

**Mean difference was calculated as the second group minus the first group. P values were adjusted using Tukey's honestly significant difference test. CI, confidence interval; HF, high frequency; LF, low frequency; nu, normalised units.*

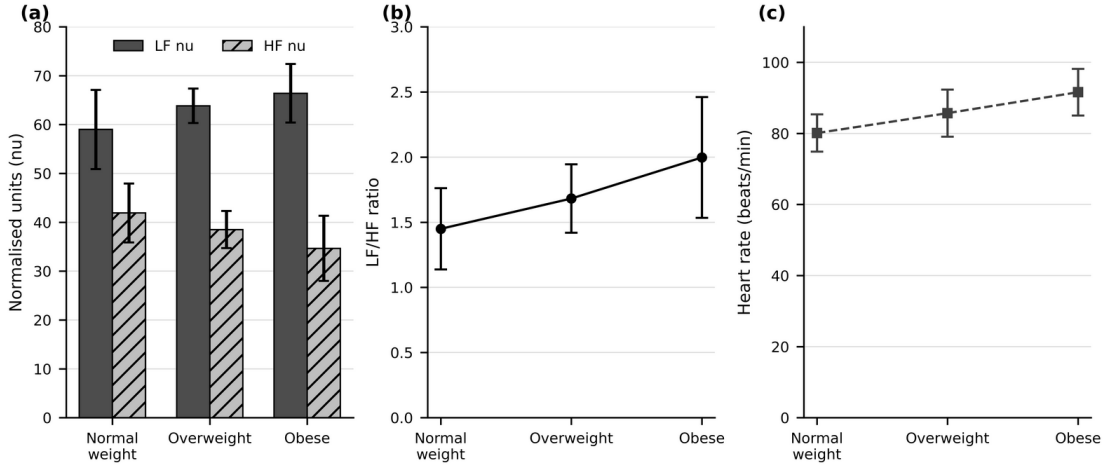


Figure 2. Frequency-domain heart rate variability indices and heart rate across body mass index categories. (a) Mean LF nu and HF nu values. (b) Mean LF/HF ratio. (c) Mean heart rate. Error bars represent standard deviations; n = 30 per group. Overall comparisons were performed using one-way ANOVA followed by Tukey's HSD test; exact pairwise results are reported in Table 3. LF, low frequency; HF, high frequency; nu, normalised units.

DISCUSSION

The present study demonstrated significant differences in cardiac autonomic modulation and resting heart rate across BMI categories among women aged 20–40 years. At the group level, LF nu, LF/HF ratio and heart rate increased, whereas HF nu decreased, from normal weight through overweight to obesity. Pairwise analysis showed that LF nu, LF/HF ratio and heart rate differed significantly between normal-weight and overweight women. However, HF nu did not differ significantly between these groups

after Tukey adjustment. These findings suggest that differences in selected autonomic indices may be detectable from the overweight category, although a uniform stepwise difference across all HRV parameters was not demonstrated.

Higher LF nu accompanied by lower HF nu is compatible with reduced cardiac vagal modulation and relatively greater sympathetic influence. Nevertheless, LF nu is influenced by both sympathetic and parasympathetic activity, and the LF/HF ratio cannot be interpreted as a direct quantitative measure of sympathovagal balance.[4,7] The findings should therefore be described as BMI-related differences in cardiac autonomic modulation rather than definitive evidence of sympathetic overactivity or autonomic dysfunction.

The observed pattern is broadly consistent with previous studies linking higher BMI and adiposity indices with lower vagally mediated HRV.[10,11] Surana Gandhi et al. reported associations between anthropometric measures and HRV among overweight individuals, supporting the possibility that autonomic differences may appear before more severe obesity develops.[11] Tomar et al. similarly reported altered HRV indices in obesity together with changes in ventricular repolarisation parameters.[10] The present study extends these observations by comparing normal-weight, overweight and obese women within a restricted reproductive-age range and by reporting Tukey-adjusted pairwise differences with 95% confidence intervals.

The increase in resting heart rate across BMI categories supports the HRV findings. Resting heart rate was significantly higher in overweight women than in normal-weight women and was further increased in the obese group. A higher resting heart rate may reflect reduced vagal restraint, increased sympathetic influence or differences in cardiovascular and metabolic demand. However, heart rate itself can affect several HRV indices; consequently, the observed HRV differences cannot be assumed to be entirely independent of the accompanying differences in mean heart rate.

Age and sex are important determinants of HRV.[5,6] Alyahya et al. demonstrated independent influences of age and sex on resting HRV and cardiometabolic function in healthy adults.[5] A systematic review of 24-hour ECG recordings also identified sex-related differences in HRV across daytime and night-time periods.[6] Restricting the sample to women aged 20–40 years reduced heterogeneity related to age and sex. However, because men were not included, the findings cannot be generalised to male populations or interpreted as evidence of sex-specific effects.

Ovarian hormonal variation is an additional consideration when HRV is assessed in reproductive-age women. Previous studies have demonstrated variation in HRV across menstrual-cycle phases and associations with circulating oestradiol and progesterone concentrations.[12,13] Although all participants had regular menstrual cycles, recordings were not standardised to a specific menstrual phase. Hormonal variation may therefore have contributed to within-group variability and remains a potential source of residual confounding.

Several biological pathways may explain the association between excess adiposity and altered cardiac autonomic modulation, although these mechanisms were not directly investigated. Adipose-tissue expansion is associated with altered adipokine secretion and chronic low-grade inflammation. Mediators such as leptin, interleukin-6 and tumour necrosis factor-alpha may influence central autonomic regulation and sympathetic outflow.[14] Obesity-related insulin resistance and endothelial dysfunction may also impair cardiovascular reflex regulation and reduce vagal modulation.[14,15] These pathways provide biological plausibility but should not be interpreted as mechanisms demonstrated in the present participants.

Reduced HRV has been associated with an increased risk of cardiovascular events among populations without recognised cardiovascular disease.[3] Obesity has also been associated with structural and electrical cardiac remodelling and increased susceptibility to atrial fibrillation and other arrhythmias.[8,9] Associations between adiposity and ventricular repolarisation abnormalities, including QT and QTc prolongation, have also been reported.[10,16,17] However, ventricular repolarisation was not evaluated in the present analysis, and the current findings cannot establish a relationship between HRV differences and arrhythmic outcomes.

The findings may be relevant to India, where abdominal obesity affects a substantial proportion of women.[1] Differences in selected autonomic indices among overweight women support further investigation of whether HRV provides clinically meaningful information beyond established cardiovascular risk measures. Nevertheless, this study did not evaluate diagnostic accuracy, prospective cardiovascular events or the incremental predictive value of HRV. Short-term HRV should therefore not be presented as an established screening or diagnostic test based on these data alone. Evidence from intervention studies suggests that structured weight-reduction programmes may improve cardiac autonomic modulation in overweight and obese individuals.[18] The present study did not include an intervention or follow-up assessment and cannot determine whether the observed differences were reversible.

The study has several strengths. Anthropometric and HRV measurements were obtained using a standardised protocol, within a defined time window and in accordance with international Task Force recommendations.[4] Restricting the sample to women within a relatively narrow age range reduced variation attributable to age and sex.[5,6] Reporting overall ANOVA results together with Tukey-adjusted pairwise comparisons and 95% confidence intervals provided a clearer representation of the between-group differences.

Several limitations should be acknowledged. The cross-sectional design prevents determination of temporality or causality. The single-centre setting, fixed sample of 30 participants per category and absence of a documented a priori sample-size calculation limit precision and generalisability. Selection bias cannot be excluded because participants were recruited from women accompanying patients attending a General Medicine outpatient department, and the total number screened and reasons for non-enrolment were not prospectively documented. HRV was measured only once, menstrual-cycle phase was not standardised, and residual confounding by physical activity, dietary habits, sleep, psychological stress and other unmeasured factors remains possible. Although waist circumference and waist–hip ratio were measured, their independent associations with HRV were not evaluated. The groups also differed significantly in height and other anthropometric characteristics, and adjusted multivariable analyses were not reported.

Larger multicentre prospective studies should include an a priori sample-size calculation, standardisation or adjustment for menstrual-cycle phase, repeated HRV measurements and assessment of relevant metabolic, behavioural and hormonal covariates. Analyses examining BMI and central-adiposity measures as continuous exposures may provide additional information beyond categorical comparisons. Prospective follow-up is required to determine whether BMI-related HRV differences predict clinically relevant cardiovascular outcomes or improve risk assessment beyond established clinical measures.

CONCLUSION

Increasing BMI was associated with graded differences in cardiac autonomic modulation, with selected indices differing even between normal-weight and overweight women. These cross-sectional findings describe BMI-related differences in autonomic modulation but do not establish autonomic dysfunction or the screening utility of HRV. Larger prospective studies are needed to determine their predictive and clinical significance.

CONTRIBUTION DETAILS

SV: concept, design, clinical studies, data acquisition, data analysis, statistical analysis, manuscript preparation. AR: literature search, data acquisition, manuscript editing. RS: concept, design, manuscript review. VD: clinical studies, manuscript review. SK: design, manuscript review, guarantor support. GN: data acquisition, manuscript review. Dr. V. Santhosini is the guarantor for the integrity of the work as a whole.

FUNDING

The study received no external funding.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY

De-identified data are available from the corresponding author on reasonable request, subject to institutional and ethical requirements.

DISCLOSURE OF AI USE

Generative artificial intelligence was used only for language editing and manuscript-formatting assistance. The authors independently verified all scientific content, statistical results, citations and references and accept full responsibility for the final manuscript. Artificial intelligence was not used to generate or modify the study data or results.

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