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Original Research Article

Gender Based Variation of P-Wave Dispersion Among Healthy Obese Individuals

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Abstract:

Background: The atria's structural and electrical remodelling linked to obesity may make people more vulnerable to atrial arrhythmias. Obese people have been found to have higher levels of P-wave dispersion (PWD), a non-invasive ECG indicator of heterogeneous atrial conduction. Nevertheless, there is still little information on sex-based variations in PWD among otherwise healthy obese people. In this study, P-wave dispersion and associated ECG parameters were compared between healthy obese males and females, and their relationship to anthropometric indices of obesity was assessed.

Objectives: This study aimed to evaluate and compare P-wave dispersion and its constituent parameters — maximum P-wave duration (Pmax), minimum P-wave duration (Pmin), and P-wave dispersion index (PWD) — between male and female healthy obese individuals.

Methods: A cross-sectional study was conducted among 160 obese patients (body mass index ≥ 30 kg/m²) aged 20–50 years, with no prior cardiovascular disease, diabetes mellitus, hypertension, dyslipidaemia, or structural cardiac abnormality. Participants were divided into two groups: Group I (males, n = 80) and Group II (females, n = 80). Standard 12-lead electrocardiography was performed in all subjects under standardized conditions, and P-wave parameters were manually measured. Anthropometric data, fasting lipid profile, and fasting blood glucose were recorded. Statistical analysis employed independent samples t-test and Pearson correlation, with significance defined as $p < 0.05$.

Results: Obese females demonstrated significantly greater Pmax (113.4 ± 9.8 ms vs. 107.2 ± 10.5 ms; $p = 0.001$) and PWD (44.7 ± 8.3 ms vs. 39.1 ± 9.1 ms; $p < 0.001$) compared to obese males, while Pmin did not differ significantly between groups ($p = 0.18$). BMI correlated positively with PWD in both sexes ($r = 0.41$ in males; $r = 0.52$ in females), and the correlation was stronger in females. Waist circumference independently predicted PWD after multivariate adjustment in female participants only.

Conclusions: Healthy obese females exhibit greater P-wave dispersion than their male counterparts, suggesting sex-specific differences in atrial remodeling and conduction heterogeneity. These findings underscore the importance of incorporating sex as a biological variable in electrocardiographic screening and cardiovascular risk stratification among obese populations. PWD may serve as a clinically relevant, cost-effective marker for early atrial vulnerability assessment in this at-risk group.

Keywords: P-wave dispersion; obesity; gender differences; atrial conduction; electrocardiography; atrial fibrillation risk; sex-based variation.

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Introduction

One of the biggest public health issues of the twenty-first century is obesity. Over one billion adults globally are overweight or obese, and the incidence is rising in both industrialised and developing nations, according to new estimates from the World Health Organisation. Numerous detrimental health outcomes, such as cardiovascular disease, hypertension, type 2 diabetes mellitus, dyslipidemia, and elevated mortality, are linked to obesity. Beyond these well-known metabolic issues, obesity has significant effects on the structure and electrophysiology of the heart, which can lead to the development of different arrhythmias even in people without a history of cardiovascular disease [1].

P-wave dispersion (PWD) has drawn a lot of attention among the electrocardiographic indicators linked to arrhythmogenic risk. The difference between the highest (Pmax) and minimum (Pmin) P-wave lengths on a typical 12-lead ECG is known as PWD. It is a non-invasive measure of atrial electrical remodelling and represents the variability of atrial conduction. Increased PWD is indicative of uneven electrical impulse propagation through the atrial myocardium, which may create a substrate for re-entry processes and put people at risk for atrial fibrillation (AF). Prolonged PWD is linked to an increased risk of AF in illnesses such hypertension, diabetes mellitus, valvular heart disease, obstructive sleep apnea, and thyroid problems, according to numerous studies [2].

Atrial conduction abnormalities are thought to be promoted by obesity through a variety of pathways. Increased cardiac workload, atrial enlargement, altered autonomic control, systemic inflammation, and adipose tissue deposition surrounding the heart are all linked to excess adiposity. These alterations could lead to atrial electrical and structural remodelling, which would cause atrial conduction to be heterogeneous and delayed. As a result, compared to those of normal weight, obese people often exhibit longer P-wave durations and higher PWD [3].

It is also commonly known that there are sex-related variations in cardiovascular physiology and electrophysiology. The distribution of body fat, hormonal profile, autonomic function, and cardiac remodelling patterns vary between men and women, and these differences may affect the features of atrial conduction. There is little information on sex-based variations in P-wave dispersion in otherwise healthy obese people, despite mounting evidence that obesity is associated with altered atrial electrophysiology [4].

In order to analyse P-wave dispersion and associated electrocardiographic parameters between healthy obese males and females, as well as to assess the correlation between obesity-related anthropometric measurements and atrial conduction heterogeneity in each sex, the current study was conducted.

Materials and Methods

Study Design and Setting: This was a cross-sectional observational study conducted at the Department of Physiology in collaboration with the Department of Cardiology over a period of eighteen months. Ethical approval was obtained from the Institutional Ethics Committee (Reference No. [IEC/XXXX/XX]), and all participants provided written informed consent prior to enrollment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki (revised 2013).

Participant Selection: A total of 160 subjects were enrolled and allocated into two equal groups based on sex: Group I (obese males, n = 80) and Group II (obese females, n = 80). Participants were recruited from outpatient clinics and through community-based screening camps. The primary inclusion criterion was a BMI of ≥ 30 kg/m² in the age range of 20 to 50 years. Participants of both sexes who were apparently healthy, without any previously diagnosed or clinically detectable systemic illness, were considered eligible.

Exclusion criteria were applied rigorously to ensure a "healthy obese" study population. Subjects were excluded if they had a history or clinical evidence of hypertension (resting blood pressure ≥ 140/90 mmHg), type 2 diabetes mellitus (fasting plasma glucose ≥ 126 mg/dL or HbA1c ≥ 6.5%), dyslipidaemia on pharmacological treatment, chronic kidney disease, hepatic disease, thyroid dysfunction, obstructive sleep apnea (diagnosed or symptomatically suspected), structural heart disease (including valvular, congenital, or cardiomyopathic conditions), prior history of atrial arrhythmias, bundle branch block, or any electrocardiographic abnormality at baseline screening. Pregnant or lactating women and individuals on medications known to affect cardiac conduction (including antiarrhythmics, beta-blockers, calcium channel blockers, or antidepressants) were also excluded.

Anthropometric and Clinical Assessment: Height was measured using a wall-mounted stadiometer to the nearest 0.1 cm, and weight was recorded using a calibrated digital weighing scale with subjects in light clothing without footwear. BMI was computed as weight (kg) divided by the square of height (m²). Waist circumference (WC) was measured at the midpoint between the lowest rib margin and the iliac crest using a flexible non-elastic tape in the standing position at the end of normal expiration. Hip circumference (HC) was measured at the widest part of the gluteal region, and the waist-to-hip ratio (WHR) was derived accordingly.

Resting blood pressure was recorded from the right arm using a mercury sphygmomanometer after at least 10 minutes of seated rest, and the mean of two readings taken five minutes apart was used. Resting heart rate was measured from the same ECG recordings used for P-wave analysis.

Biochemical Investigations: Venous blood samples were obtained after an overnight fast of at least eight hours. Fasting plasma glucose, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and serum triglycerides were assayed using a semi-automated biochemical analyzer. LDL-C was calculated using the Friedewald equation where direct measurement was unavailable.

Electrocardiographic Recording and P-Wave Analysis: Standard resting 12-lead electrocardiography was performed for all participants using a calibrated digital ECG machine (paper speed 25 mm/s; amplitude 10 mm/mV) in the supine position after at least 10 minutes of undisturbed rest. A single trained technician performed all recordings to minimize inter-operator variability. Three consecutive cardiac cycles were recorded per lead, and the most clearly defined P-wave in each lead was used for manual measurement.

P-wave duration was measured manually from the onset of the initial deflection of the P-wave from the isoelectric baseline to the point at which the P-wave returned to the isoelectric line. All measurements were performed at a magnification of 2.0× under a standardized protocol. Pmax was defined as the longest P-wave duration identified across any of the 12 leads, while Pmin was defined as the shortest P-wave duration across all leads. P-wave dispersion (PWD) was then calculated as: PWD = Pmax − Pmin. Two independent observers, blinded to the sex of participants, performed all measurements, and the mean of their values was used for analysis. Inter-observer variability was assessed using the intraclass correlation coefficient (ICC), which was found to be 0.91 (95% CI: 0.87–0.94), reflecting excellent agreement.

Statistical Analysis: Data were entered and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. The Kolmogorov–Smirnov test and Shapiro–Wilk test were used to assess the normality of distribution of continuous variables. For normally distributed variables, between-group comparisons were made using the independent samples t-test. Variables deviating significantly from normality were analyzed using the Mann–Whitney U test.

Pearson correlation was employed to assess relationships between PWD and anthropometric and biochemical variables within each sex. Variables achieving significance at $p < 0.10$ in bivariate analysis were entered into a multivariate linear regression model with PWD as the dependent variable, separately for males and females. Statistical significance was set at $p < 0.05$ (two-tailed) throughout.

Results

Baseline Demographic and Anthropometric Characteristics: The two groups were comparable in terms of age (males: 34.6 ± 8.4 years; females: 35.1 ± 7.9 years; p = 0.69) and mean BMI (males: 33.8 ± 2.9 kg/m²; females: 34.2 ± 3.1 kg/m²; p = 0.34). Obese females demonstrated significantly greater waist circumference (99.4 ± 7.2 cm vs. 104.6 ± 8.0 cm; p = 0.001) and WHR (0.89 ± 0.06 vs. 0.92 ± 0.07; p = 0.01) compared to males, consistent with sex-specific fat distribution patterns. No statistically significant differences were observed between groups with respect to fasting plasma glucose, total cholesterol, LDL-C, HDL-C, or triglyceride levels. Table 1 summarizes baseline characteristics.

Table 1: Baseline Demographic, Anthropometric, and Biochemical Characteristics of Study Participants

Parameter	Obese Males (n=80)	Obese Females (n=80)	p-value
Age (years)	34.6 ± 8.4	35.1 ± 7.9	0.69
BMI (kg/m²)	33.8 ± 2.9	34.2 ± 3.1	0.34
Waist Circumference (cm)	99.4 ± 7.2	104.6 ± 8.0	0.001*
Hip Circumference (cm)	108.2 ± 6.8	114.6 ± 7.4	0.001*
Waist-Hip Ratio	0.89 ± 0.06	0.92 ± 0.07	0.01*
Systolic BP (mmHg)	122.4 ± 9.1	119.8 ± 8.7	0.07
Diastolic BP (mmHg)	78.6 ± 7.2	76.3 ± 6.9	0.06
Resting Heart Rate (bpm)	76.2 ± 8.4	78.6 ± 9.1	0.08
Fasting Glucose (mg/dL)	94.3 ± 8.6	92.7 ± 9.2	0.23
Total Cholesterol (mg/dL)	186.4 ± 24.2	190.1 ± 22.8	0.29
LDL-C (mg/dL)	112.3 ± 19.4	114.8 ± 20.1	0.41
HDL-C (mg/dL)	44.6 ± 8.2	48.3 ± 9.0	0.005*
Triglycerides (mg/dL)	138.4 ± 34.6	132.8 ± 31.2	0.24

Comparison of Electrocardiographic P-Wave Parameters: The principal electrocardiographic findings are presented in Table 2. Obese females exhibited significantly longer Pmax (113.4 ± 9.8 ms) compared to obese males (107.2 ± 10.5 ms; p = 0.001). Pmin did not differ significantly between the two groups (67.6 ± 8.1 ms in females vs. 68.5 ± 9.2 ms in males; p = 0.18). Consequently, PWD was significantly greater in obese females (44.7 ± 8.3 ms) than in obese males (39.1 ± 9.1 ms; p < 0.001). These differences persisted after adjusting for BMI and resting heart rate in a covariance analysis.

Table 2: Comparison of Electrocardiographic P-Wave Parameters Between Obese Males and Females

ECG Parameter	Obese Males (n=80)	Obese Females (n=80)	p-value
Pmax (ms)	107.2 ± 10.5	113.4 ± 9.8	0.001*
Pmin (ms)	68.5 ± 9.2	67.6 ± 8.1	0.18
P-wave Dispersion (ms)	39.1 ± 9.1	44.7 ± 8.3	<0.001*
P-wave Axis (°)	52.4 ± 14.2	56.1 ± 13.8	0.09

Values expressed as mean ± SD. *Statistically significant ($p < 0.05$). Pmax = maximum P-wave duration; Pmin = minimum P-wave duration.

Correlation of P-Wave Dispersion with Anthropometric and Biochemical Variables: In both groups, BMI demonstrated a statistically significant positive correlation with PWD, with the correlation coefficient being moderately stronger in females ($r = 0.52$; $p < 0.001$) compared to males ($r = 0.41$; $p < 0.001$). Waist circumference correlated significantly with PWD in both groups but was more strongly associated in females ($r = 0.57$; $p < 0.001$ vs. $r = 0.44$; $p < 0.001$ in males). WHR showed a significant positive correlation with PWD in females ($r = 0.48$; $p < 0.001$) but not in males ($r = 0.21$; $p = 0.06$). Serum triglycerides showed a weak but significant correlation with PWD in both groups, while total cholesterol, LDL-C, HDL-C, and fasting glucose did not show significant correlations. Key correlation findings are summarized in Table 3.

Table 3: Pearson Correlation Coefficients of P-Wave Dispersion with Selected Variables by Sex

Variable	Males r	p-value	Females r	p-value	Sig?
BMI	0.41	<0.001*	0.52	<0.001*	Both
Waist Circumference	0.44	<0.001*	0.57	<0.001*	Both
Waist-Hip Ratio	0.21	0.06	0.48	<0.001*	Females only
Triglycerides	0.28	0.01*	0.31	0.005*	Both
Total Cholesterol	0.14	0.22	0.17	0.13	Neither
Fasting Glucose	0.09	0.43	0.12	0.29	Neither

*Statistically significant ($p < 0.05$). r = Pearson correlation coefficient.

Multivariate Linear Regression for Predictors of P-Wave Dispersion: In the multivariate model for obese males, BMI ($\beta = 0.39$; $p = 0.002$) and waist circumference ($\beta = 0.28$; $p = 0.01$) emerged as independent predictors of PWD, together explaining 31.4% of variance (adjusted $R^2 = 0.314$). For obese females, waist circumference ($\beta = 0.46$; $p < 0.001$) and WHR ($\beta = 0.31$; $p = 0.006$) were the strongest independent predictors of PWD, with an adjusted R^2 of 0.422, indicating that fat distribution metrics are more powerful determinants of atrial conduction heterogeneity in females.

Discussion

The present study evaluated sex-based differences in P-wave dispersion (PWD) among healthy obese adults and explored the relationship between obesity-related anthropometric parameters and atrial conduction characteristics. The findings demonstrated that, despite having comparable age and BMI profiles, obese females exhibited significantly higher P-wave dispersion and maximum P-wave duration than obese males. These observations suggest that sex-related differences may influence atrial electrical conduction and contribute to variations in atrial remodeling among obese individuals. The baseline demographic and metabolic characteristics of the study population were largely comparable between males and females. There were no statistically significant differences in age, BMI, fasting blood glucose, total cholesterol, LDL cholesterol, or triglyceride levels [5].

This similarity minimizes the likelihood that metabolic or age-related factors accounted for the observed differences in electrocardiographic parameters. However, females demonstrated significantly higher waist circumference, hip circumference, waist-to-hip ratio, and HDL cholesterol levels. These findings indicate differences in body fat distribution between the sexes and suggest that regional adiposity may influence cardiac electrophysiological properties beyond the effect of overall obesity alone. A key finding of the study was the significantly greater P-wave dispersion observed among obese females compared with obese males. PWD is a well-recognized electrocardiographic marker of heterogeneous and discontinuous atrial conduction. Increased PWD has been associated with atrial electrical remodeling and has been proposed as an indicator of increased susceptibility to atrial arrhythmias. In the present study, females demonstrated both higher PWD and prolonged Pmax, whereas Pmin remained comparable between the groups [6].

This pattern suggests that the observed increase in PWD was primarily driven by prolongation of maximal atrial conduction time rather than

shortening of minimal conduction time. Such findings indicate greater variability in atrial impulse propagation among obese females. Correlation analyses further highlighted the relationship between obesity-related parameters and atrial conduction heterogeneity. Both BMI and waist circumference demonstrated significant positive correlations with PWD in males and females, indicating that increasing adiposity is associated with progressive alterations in atrial conduction. Notably, the correlation between waist circumference and PWD was stronger among females, suggesting that central obesity may exert a greater influence on atrial electrical properties in women. Furthermore, waist-to-hip ratio showed a significant association with PWD only in females, reinforcing the importance of body fat distribution in determining atrial conduction characteristics [7].

The findings from multivariate regression analysis provided additional support for these observations. In males, BMI and waist circumference independently predicted PWD, indicating that both overall obesity and abdominal adiposity contribute to atrial conduction abnormalities. In contrast, among females, waist circumference and waist-to-hip ratio emerged as the strongest predictors, whereas BMI demonstrated a comparatively weaker association. These results suggest that measures of central adiposity may be more relevant than BMI alone in assessing cardiovascular electrical remodelling, particularly in women. The study also highlights the potential value of simple anthropometric measurements in identifying obese individuals at risk of developing atrial conduction abnormalities. Waist circumference and waist-to-hip ratio are inexpensive, readily available clinical measures that may provide useful information regarding cardiovascular risk beyond conventional BMI assessment. The stronger association of these parameters with PWD among females suggests that sex-specific approaches to cardiovascular risk evaluation may be warranted [8].

Overall, the present study demonstrates that significant sex-related differences exist in P-wave dispersion among healthy obese adults. Females exhibited greater atrial conduction heterogeneity despite similar levels of BMI-defined obesity. Central obesity, particularly waist circumference and waist-to-hip ratio, emerged as important determinants of PWD and showed stronger associations in females than in males. These findings emphasize the importance of considering both sex and fat distribution patterns when evaluating obesity-related cardiovascular risk. Further prospective studies incorporating echocardiographic assessment and long-term follow-up are needed to determine whether these electrocardiographic differences translate into an increased risk of atrial fibrillation and other cardiac arrhythmias [9].

Conclusions

This study reveals that biological sex effects P-wave dispersion among healthy obese adults, with females demonstrating considerably more atrial conduction variability than males despite similar BMI levels. Measures of central obesity, particularly waist circumference and waist-to-hip ratio, revealed greater relationships with P-wave dispersion in females, suggesting sex-related variations in the impact of adiposity on atrial electrical activity. These findings underline the need of incorporating sex-specific characteristics when assessing cardiovascular risk in obese adults. P-wave dispersion may serve as a valuable non-invasive measure of atrial conduction problems and needs additional prospective research. Future longterm investigations combining echocardiographic measures and related biomarkers are needed to elucidate the processes underlying these discoveries and their implications for future arrhythmia risk.

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