RESEARCH ARTICLE

Effect of body mass index (BMI) on electrocardiographic P-wave dispersion among healthy adults

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ABSTRACT

Background: According to the results of an electrocardiogram (ECG), a higher body mass index (BMI) indicates body fat and is linked to a higher risk of cardiovascular illnesses. A noninvasive ECG signal called P-wave dispersion (Pd) can predict the likelihood of an atrial arrhythmia and reflects atrial remodeling. Pd is calculated by computing the difference between the maximum and minimum P-wave lengths captured from the 12 ECG lead recordings. To assess the early-stage risk of acquiring cardiovascular illnesses, we investigated the effect of BMI on P-wave dispersion in healthy, overweight, and obese adults. *Materials and Methods*: Depending on their body mass index (BMI), we divided 200 participants into three groups (normal weight, overweight, and obese) during a comparative cross-sectional study. We measured 12-lead surface ECG and P-wave dispersion in study participants. *Results*: Mean P-wave lengths were 33.23(± 6.08), 33.15(± 5.16), and 46.15 (± 5.37), respectively, for people who were normal weight, overweight, and obese. Between the normal-weight and obese groups and the overweight and obese groups, there was a statistically significant difference ($p < 0.001$), according to Tukey's post-hoc analysis. However, the normal-weight and overweight groups showed no statistically significant difference ($P = 0.997$). Pearson's correlation analysis shows that P-wave dispersion and BMI showed a significant positive connection ($r = 0.632$). *Conclusion*: A higher BMI is linked to a longer P-wave duration and dispersion, even in healthy adults. The likelihood of left atrial hypertrophy and atrial arrhythmia increases with a prolonged P-wave duration. This study emphasizes the importance of raising awareness about the need to adopt healthy lifestyles to avoid the harmful effects of obesity on the heart.

Keywords: BMI, Overweight, Obesity, ECG, P-wave, P-wave dispersion, Atrial fibrillation

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INTRODUCTION

BMI is frequently used to categorize people as underweight, normal weight, overweight, or obese. The Body Mass Index (BMI) is estimated by dividing a person's weight in kilograms by the square of their height in meters (kg/m^2). As per the World Health Organization (WHO), underweight person is defined as having a BMI below 18.5; normal weight BMI between 18.5 and 24.99; and overweight (pre-obesity) is defined as having a BMI between 25 and 29.99. Values greater than 30, on the other hand, indicated obesity, with subcategories for obesity class 1 (30–34.99), class 2 (35–39.99), and class 3 (>40). 39% of persons ages 18 and older were found to be overweight, and 13% were obese, as recorded by the WHO.[1] Obesity is a significant global socioeconomic and public health burden affecting developed and developing countries.[2] It is linked to several health issues, including metabolic syndrome, type 2 diabetes, hypertension, abnormal cholesterol levels, and coronary artery disease. Notably, sudden deaths have been documented in obese people with no known heart problems before.[3,4] Elevated BMI contributes to hemodynamic changes characterized by increased blood pressure, stroke volume, and pulmonary and left atrial pressure.[5] These modifications can potentially cause left ventricular heart failure and obesity-related left ventricular diastolic and systolic dysfunction by causing left atrial enlargement, remodeling, and hypertrophy.[6] Researchers have linked P-wave dispersion (Pd) to atrial fibrillation (AF), characterized by discontinuous and inhomogeneous sinus impulse propagation.[7] The evaluation of the electrical activity of the heart and the early diagnosis of cardiac abnormalities are possible with electrocardiography (ECG). Only a few studies have examined how BMI affects the length and dispersion of the P-wave. The idea of P-wave dispersion as a trustworthy indicator of cardiovascular risks has changed in recent years. Nevertheless, research has highlighted the methodological flaws in conventional P-wave dispersion estimations. For instance, low ECG paper speeds, the imprecision of single-point measurements, and a lack of sophisticated analytical methods have all been suggested as potential sources of inaccuracy.[8] However, in numerous clinical contexts, Pd is a sensitive and specific ECG predictor of AF. Physicians may use the prediction of AF recurrence to help them choose antiarrhythmic treatment plans. This

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study looked at P-wave dispersion to stratify the future risk of cardiovascular illness in a sample of healthy young adults.

MATERIALS AND METHODS

Study Design

The Physiology Department of the Indira Gandhi Institute of Medical Sciences (IGIMS), Patna, Bihar, India, conducted this study. The formula n = (Z1 -)2pq/L2, where n denotes the sample size, p is the prevalence, q is 1-p, L is the error (0.05 or 95%), and Z1 - is the standard value (1.96), was used to estimate the sample size of 200 participants. We calculated the sample size to be 195 using a prevalence of 15% and the inputted values. Inclusion criteria: 200 healthy male and female adults aged 18–40. Of the participants, 120 were males, and 80 were females. After explaining the study protocol, we obtained informed written consent from all participants. We categorized all study participants into three groups based on their BMI (kg/m^2): normal weight (BMI 18–24.9) with 120 participants (80 males and 40 females), overweight (BMI 25–29.9) with 40 participants (20 males and 20 females), and obese (BMI > 30) with 40 participants (20 males and 20 females). Exclusion Criteria: individuals aged < 18 years and > 40 years; those with a history of cardiovascular disease, hypertension, or medication known to affect cardiac electrical activity (such as antiarrhythmic drugs); individuals with thyroid disorders; smokers; and individuals with sleep disorders, menstrual abnormalities, and neuropsychiatric disorders. Materials required for the study: weighing machine, stadiometer, sphygmomanometer, Vernier caliper, and a three-channel ECG machine (Medicaid, India).

This study was approved and conducted by the Institutional Ethical Committee of IGIMS Patna approval vide letter no. RC/IGIMS/Pat/1191/Academic. Following the National Ethical Guidelines for Biomedical and Health Research 'Involving Human Participants 2018' of the Indian Medical Research Council, all participants were included in the study after getting written consent, in accordance with the

Data Collection Methods

BMI: Body weight was measured using a portable weighing machine without shoes and with light clothing, whereas height was measured barefoot using a stadiometer. The participants stood against a standard meter scale, with their ears and infraorbital margins aligned horizontally. Body weight was recorded in kilograms before lunch with an empty bladder. We did not get a statistically significant difference when comparing the P-wave dispersion (Pd) between the overweight and normal-weight groups. We determined BMI as weight in kilograms divided by height in square meters (BMI = Weight (kg)/height (m2)).

Blood pressure measurements: The participants were supine, following five minutes of rest using a gold-standard mercury sphygmomanometer; we recorded the right arm's systolic blood pressure (SBP) and diastolic blood pressure (DBP).

Electrocardiographic recordings: The ECGs of the participants were recorded from 10:00 to 12:00 noon to minimize diurnal variations. The ECG paper speed was 50 mm/s, and the voltage was one mV/cm. We recorded resting ECGs of participants lying down after 10 minutes of rest in a quiet and comfortable room. We examined the participant's chest, forearms, and legs to avoid interference and removed electronic gadgets and metallic ornaments. To prevent interference, we kept participants away from the AC-operated machines. We clipped the participant's limb electrodes to the skin and placed chest leads at six locations. We used ECG gel for good electrical contact.

Measurement of P-wave dispersion (Pd): Using a handheld caliper, P-wave dispersion (Pd) was measured by Liu *et al*. and Magnani *et al*. We derived Pd by subtracting the minimum P-wave duration from the maximum period observed in any of the 12 ECG leads. The researchers defined P-wave onset as the initial deflection from the isoelectric baseline defined by the T-P segment. The researchers described the junction of the end of the P-wave and its return to the baseline as P-wave offset.[7-9]

Statistical Analysis

SPSS 20, a statistical software, was used to analyze the data. Means and standard deviations were estimated to express the distribution of parameters. To compare the mean values of the parameters among the average weight, overweight, and obese groups, one-way ANOVA was used. In contrast, the differences between the two groups were processed using Tukey's test. The relationships between the parameters were determined using Pearson's test. All statistical tests were conducted, accepting the significance at $p < 0.05$.

RESULTS

The chi-square test showed no significant difference (p=0.062) between males and females in the different BMI groups. The mean age (in years) of 200 subjects was 24.63 ± 5.14. The mean weight (kg) and height (m) were 64.87 ± 11.6 and 1.61 ± 0.08, respectively. The mean BMI of the study sample (kg/m^2) was 25.09 ± 4.64. The mean BMI of the normal-weight, overweight, and obese groups was 21.95 ± 1.86, 26.90 ± 1.25, and 32.70 ± 2.07, respectively.

The cardiovascular parameters were compared by ANOVA and found to be statistically significant between the groups. The average heart rate is 80.70 ± 7.03 beats per minute. The average heart rates were relatively higher in the overweight and obese groups (Table 1). Statistical analysis using ANOVA revealed significant differences between the groups ($p<0.001$). Post-hoc analysis further indicated a significant difference in heart rate between the overweight and normal-weight groups ($p = 0.001$). The obese, overweight, and normal weight groups significantly differed in their heart rate ($p< 0.001$). Furthermore, the Pearson correlation test demonstrated a strong correlation between heart rate and BMI ($r = 0.629$).

Table 1: Comparison of cardiovascular parameters among groups

Parameters	*Study Groups*			*r value*
	Normal Weight (n=120)	*Overweight (n=40)*	*Obese (n=40)*	
Heart rate (beats/min)	78.03 ± 4.80	81.90 ± 5.51 [a]	87.50 ± 8.98 [ab]	0.629 [c]
Systolic BP (mmHg)	114.07 ± 8.95	124.05 ± 5.67 [a]	128.15 ± 6.03 [a]	0.670 [c]
Diastolic BP (mmHg)	75.87 ± 5.05	82.00 ± 3.81 [a]	84.20 ± 3.65 [a]	0.666 [c]
Pmax (ms)	88.93 ± 12.74	105.15± 3.0 [a]	116.10 ± 6.29 [ab]	0.894 [c]
Pmin (ms)	55.70 ± 10.63	72.00 ± 6.52 [a]	69.95 ± 7.14 [a]	0.731 [c]
Pd (ms)	33.23 ± 6.08	33.15 ± 5.16	46.15 ± 5.37 [ab]	0.632 [c]

Pmax: Maximum P-wave duration, Pmin: Minimum P-wave duration, Pd: P-wave dispersion duration. All the values are mean ± standard deviation. [a] indicates a significant difference with the Normal Weight group, and [b] indicates a significant difference with the Overweight group (Tukey post-hoc analysis). [c] indicates a significant correlation for the Pearson correlation coefficients (r values).

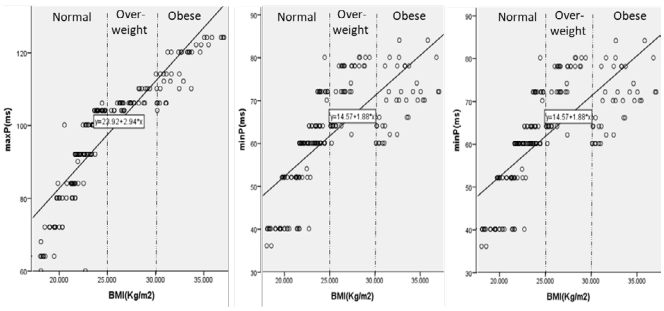

Figure 1: Distribution of Pmax, Pmin, and Pd against the BMI grouped into normal, overweight, and obese

The average systolic blood pressure (SBP) of all the participants was found to be 118.88 ± 9.89 mmHg. The mean SBP values for the normal-weight, overweight, and obese groups were 114.07 ± 8.95, 124.05 ± 5.67, and 128.15 ± 6.03, respectively. Statistical analysis using ANOVA indicated significant differences between the groups (p< 0.001). Tukey's test showed that the normal-weight group had a significantly different mean value from the overweight group (p< 0.001). However, the mean value of the overweight group was not significantly different from that of the obese group (p = 0.054). Pearson's test showed a strong positive relationship between BMI and SBP (r = 0.670).

The mean diastolic blood pressure in (study) was 78.76 ± 5.81 mmHg. Among the different groups, the mean DBP values were 75.87 ± 5.05 for the normal-weight group, 82.00 ± 3.81 for the overweight group, and 84.20 ± 3.65 for the obese group. The ANOVA result values indicated statistically significant differences between the groups (p< 0.001). Post-hoc analysis revealed significant differences between the normal-weight and overweight groups (p< 0.001), as well as between normal-weight and obese groups (p< 0.001). The overweight and obese groups showed no statistically significant difference (P = 0.907). Pearson's correlation test also showed a strong correlation between DBP and BMI (r = 0.666).

The mean maximum P-wave duration (Pmax) was 97.61 ± 15.23 ms. Among the different groups, the normal-weight group had a mean Pmax of 88.93 ± 12.74 ms, while the overweight and obese groups had mean values of 105.15 ± 3.0 ms and 116.10 ± 6.29 ms, respectively. The one-way ANOVA test expressed a statistically significant difference in the mean Pmax values between the groups (p< 0.001). Post-hoc analysis showed significant differences between the normal-weight and overweight groups (p< 0.001), as well as between normal-weight and obese groups (p< 0.001). The overweight and obese groups showed a statistically significant difference (p< 0.001). Pearson's correlation test also demonstrated a strong correlation between Pmax and BMI (r = 0.894), indicating a strong positive linear relationship (Figure 1).

The mean minimum P-wave duration (Pmin) was 61.81 ± 11.94 ms. Among the different groups, the normal-weight group had a mean Pmin of 55.70 ± 10.63 ms, while the overweight and obese groups had mean values of 72.00 ± 6.52 ms and 69.95 ± 7.14 ms, respectively. The one-way ANOVA test expressed a statistically significant difference in the mean Pmin values between the groups (p< 0.001). Post-hoc analysis showed significant differences between the normal-weight and overweight groups (p< 0.001), as well as between normal-weight and obese groups (p< 0.001). The overweight and obese groups showed no statistically significant difference (p = 0.587). Pearson's correlation test also demonstrated a strong correlation between Pmin and BMI (r = 0.731), with a correlation coefficient close to 1, indicating a strong positive linear relationship.

The study population's mean P-wave dispersion (Pd) duration was 35.80 ± 7.74 ms. Among the different groups, the normal-weight group exhibited a mean Pd duration of 33.23 ± 6.08ms, the overweight group had a mean of 33.15 ± 5.16 ms, and the obese group had the highest mean value of 46.15 ± 5.37 ms. A one-way ANOVA expressed a statistically significant difference in the mean P-wave dispersion values between the groups (p< 0.001). Tukey's post-hoc analysis revealed significant differences between the normal-weight and obese groups (p< 0.001), as well as between the overweight and obese groups (p< 0.001). The normal weight and overweight groups expressed no statistically significant difference (p = 0.997). Furthermore, the Pearson's correlation test demonstrated a strong correlation between Pd and BMI (r = 0.632).

DISCUSSION

Medical professionals recognize resting electrocardiography (ECG) as a valuable diagnostic tool for detecting asymptomatic or silent heart diseases.[10] Our analysis revealed an association between electrocardiographic P-wave parameters and BMI in healthy, obese young adults, demonstrating that higher BMI is independently associated with increased P-wave duration and P-wave dispersion. Therefore, we selected P-wave dispersion as a pivotal parameter because it is a sensitive and specific indicator for predicting atrial fibrillation (AF), a common cardiac arrhythmia linked with significant morbidity and mortality. P-wave dispersion reflects sinus impulses' heterogeneous and discontinuous propagation across the atria, indicating an increased susceptibility to atrial arrhythmias. The well-documented association between obesity and cardiovascular complications is the rationale for investigating the impact of BMI on P-wave dispersion. The increasing prevalence of obesity and its implications for cardiovascular health underscores the significance of exploring the relationship between BMI and P-wave dispersion. Investigating this connection can offer insights into the underlying mechanisms through which obesity contributes to atrial electrical abnormalities, aiding early detection and targeted intervention. By understanding how excess body weight might influence P-wave dispersion, this study aimed to shed light on the pathways that link obesity to atrial arrhythmias, thereby providing valuable guidance for clinical management and preventive strategies.

Although specific studies have questioned the clinical significance of P-wave dispersion due to the vectorial nature of atrial conduction and its negligible values, it can reach precise measurements.[11,12] These conflicting perspectives may explain the discrepancies in the literature regarding the relationship between BMI and P-wave dispersion. Our study employed a traditional approach to measure P-wave dispersion, which could contribute to the observed differences in results compared with studies utilizing more advanced vector analysis techniques.

Furthermore, our study found a statistically significant increase in heart rate among overweight and obese individuals compared with normal-weight individuals. The mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) were found to be significantly higher in overweight and obese participants than in normal-weight participants. Although the mean SBP and DBP were higher in obese than overweight individuals, the difference was insignificant.

We identified a positive correlation among BMI, heart rate, and blood pressure, aligning with the earlier findings. According to a study by La Rovere *et al*., sympathetic nervous system activation occurs early in obesity and is crucial for regulating the cardiovascular system and energy expenditure.[17] Similarly, the study by Shigetoh *et al*. showed that the heart rate increases with increased body fat percentage. Additionally, a 10% increase in body weight was associated with a decline in the parasympathetic tone and an increase in the heart rate. Conversely, heart rate decreased during weight reduction.[18] Julius *et al*., in their study, reported a positive relationship between being overweight and the development of hypertension in adolescents.[19]

Regarding P-wave parameters, our study revealed that overweight individuals had a significantly higher maximum P-wave duration ($p< 0.001$) and minimum P-wave duration ($p< 0.001$) than normal-weight individuals. Our study observed no statistically significant difference between the overweight and normal-weight groups' P-wave dispersion (Pd). In contrast, the obese group showed a higher maximum P-wave duration ($p< 0.001$), minimum P-wave duration ($p< 0.001$), and Pd ($p< 0.001$) than the normal weight group. Further, Pearson correlation analysis demonstrated a positive and significant correlation between Pmax, Pmin, and Pd, with exceptionally high correlations observed for Pmax and Pmin and a moderate correlation for Pd. These findings suggest that obese individuals have an extended P-wave duration. Our study aligns with previous research that found higher P-wave duration in subjects with increased BMI without complications than age-matched subjects with normal-weight 17-19 BMI.[20-22] Duru *et al*. noted that P-wave duration and Pd significantly decreased after substantial (10%) weight loss, and the decrease in Pd correlated with the weight loss percentage.[23] However, our study found no statistically significant difference in Pd between the overweight and normal weight groups. Our findings suggest that being overweight in healthy adults is not associated with left atrial enlargement and does not play a role in electrical instability, unlike obesity. Pritchett *et al*. and Akyuz *et al*. showed similar results.[24,25] The pathophysiology behind left atrial enlargement in obese participants is adiposity, which is associated with an increase in total blood volume, typically resulting in increased cardiac output. When the left ventricular filling pressures increase, diastolic dysfunction is observed. Over time, persistently raised filling pressures and left ventricular hypertrophy may lead to systolic impairment.[6] The increased accumulation of epicardial and pericardial fat, commonly observed in obesity, is likely to contribute to the progression of these findings.[23,24] Cardiac fat deposition has metabolic and inflammatory functions that can contribute to fibrotic remodeling of atrial tissue.[25] Studies conducted by others also showed similar results.[26-28] A study conducted by Esposito *et al*. explained that autonomic dysfunction might lead to nocturnal BP falls in obese normotensive patients, further leading to increased cardiovascular risk.[26] Alexander *et al*. observed that autonomic imbalance in obese subjects might affect intra-arterial and inter-atrial conduction times and alter the P-wave measurements. Left atrial enlargement and electrical instability in obese patients may also be influenced by high plasma volume, ventricular diastolic dysfunction, and enhanced neurohormonal activity. Changes in left atrial dimension and pressure may impact P-wave duration.[27] In addition, increased BMI is

associated with left atrial enlargement and increased left atrial pressure and volumes.[28] Earlier studies have laid the groundwork for understanding the link between P-wave dispersion and obesity.; our study offers novel insights by focusing on healthy young adults and understanding the early cardiovascular consequences of change in BMI.

Nevertheless, the limitation of manual calculation of the P-wave measurements using a hand caliper is mentioned worthy. Additionally, the current study included only a few patients in the selected population, highlighting the need for further multicenter studies to validate these findings. Our study did not classify whether obesity in the participants was central or visceral. Our study did not include neurohumoral parameters or echocardiography to measure left atrial size. The current findings must be interpreted in light of the evolving understanding of P-wave dispersion.

CONCLUSION

Elevated BMI levels were linked to increased blood pressure, higher heart rate, and prolonged P-wave duration. Longer P-waves indicate electrocardiogram dispersion, left atrial enlargement, and a higher risk of arrhythmia in young obese individuals, which may lead to severe complications like stroke, heart failure, dementia, and death. P-wave duration and Pd are the most important noninvasive ECG markers to assess atrial arrhythmia. Atrial arrhythmia can be predicted using P-wave dispersion (Pd). Our study further emphasizes the significance of monitoring P-waves and adopting healthier lifestyles to prevent cardiovascular deaths in overweight and obese young adults.

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PEER-REVIEWED CERTIFICATION

During the review of this manuscript, a double-blind peer-review policy has been followed. The author(s) of this manuscript received review comments from a minimum of two peer-reviewers. Author(s) submitted revised manuscript as per the comments of the assigned reviewers. On the basis of revision(s) done by the author(s) and compliance to the Reviewers' comments on the manuscript, Editor(s) has approved the revised manuscript for final publication.