Correlation between Anthropometric Indices and hemodynamic parameters along with ECG Among Medical Students

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https://doi.org/10.5281/zenodo.15095294

Article Received: 16-February-2025, Revised: 07-March-2025, Accepted: 27-March-2025

ABSTRACT:

This study explores the relationship between anthropometric indices and electrocardiographic (ECG) variations among 280 medical students at Delhi University. Participants were categorized into normal-weight (BMI < 25 kg/m²) and overweight/obese (BMI ≥ 25 kg/m²) groups. Blood pressure parameters revealed statistically significant elevations in the overweight/obese group. In males, the mean systolic blood pressure (SBP) was 145.32 mmHg, significantly higher than 124.85 mmHg in the normal-weight group, with a p-value of <0.001. Similarly, the mean diastolic blood pressure (DBP) was 92.78 mmHg compared to 78.62 mmHg, with a p-value of <0.001. Females showed a comparable trend, with SBP at 139.25 mmHg versus 121.47 mmHg (p<0.001) and DBP at 89.89 mmHg versus 74.56 mmHg (p<0.001). Mean arterial pressure (MAP) was significantly elevated in both genders, registering 115.63 mmHg for males and 110.12 mmHg for females in the overweight/obese group, compared to 93.72 mmHg and 88.54 mmHg, respectively, in the normal-weight group (p<0.001). Pulse pressure, a marker of arterial stiffness, was also significantly higher (58.21 mmHg for males and 54.12 mmHg for females, p<0.001), reflecting increased cardiovascular strain in individuals with higher BMI. Gender differences were notable, with more males in the overweight/obese category. Although height showed no direct correlation with BMI, shorter participants experienced greater cardiovascular load. This study emphasizes that elevated BMI in young adults affect cardiovascular structure, function, and electrical activity, increasing long-term health risks. Early interventions and personalized health strategies targeting weight management and cardiovascular monitoring are essential to mitigate these effects and promote heart health.

Keywords: Obesity, BMI, Metabolic Diseases, Cardiovascular Dysfunction, Blood Pressure, ECG.

INTRODUCTION:

Obesity can be seen as the first wave of a defined cluster of non-communicable Diseases called "New World Syndrome" creating an enormous socioeconomic and public health burden in poorer countries. (Pednekar MS, 2008). Indices predictive of adolescent central obesity include waist-to-height ratio (WHtR). WC is a highly sensitive and specific measure of upper body fat in young people and thus it is valuable for identifying overweight and obese adolescents at risk of developing metabolic complications. The same applies for risk factors of cardiovascular disease in children and adolescents, in whom WC and WHtR are better predictors than BMI. The worldwide prevalence of obesity is reaching pandemic proportions. Moreover, it is evident that there is a strong association between

elevated BMI and cardiovascular changes. However, it is not yet fully clear which anthropometric index of obesity has the strongest association with cardiac autonomic markers. There is a scarcity of research investigating this link specifically among young individuals in the Indian community. So, total body fat mass isn't the only factor that makes people vulnerable to obesity-related cardiovascular complications; regional body fat distribution variations also play a significant role, since they have a deleterious impact on cardiac structure and function. Research into the causes of obesity-related heart failure is necessary so that patients who are overweight and suffer from cardiovascular disease may be better cared for in the future, especially as obesity is becoming more common in societies with longer life expectancies. In the context of medical education, where

peer pressure may lead to psychophysiological alterations, the study that will serve as the benchmark is the association between BMI and cardiac electrical activity. The objective of our research is to determine the relationship between altered anthropometric indices and variation of ECG.

METHODOLOGY:

The present study employed an observational crosssectional design to investigate cardiovascular and autonomic function parameters among medical students aged 18 to 25 years from Delhi University. Participants were divided into two groups based on body mass index (BMI): Group A (non-obese, BMI < 25 kg/m²) and Group B (overweight and obese, BMI \ge 25 kg/m²). A total sample size of 280 was determined using a prevalence-based formula with a 5% margin of error and a 95% confidence interval, referencing prior studies on obesity-related cardiovascular changes. Inclusion criteria required participants to be clinically healthy, free from hypertension, substance abuse, or acute illness, and

willing to provide informed consent. Exclusion criteria ruled out individuals with congenital heart disease, chronic respiratory conditions. or pregnancy. medical Comprehensive histories and physical examinations were conducted to ensure eligibility. Anthropometric data, including weight (measured using an ACU Check digital scale) and height (using a vertical measuring tape), were recorded, and BMI was calculated. Blood pressure was measured thrice on the left arm using an Omron HEM 8712 device, with systolic, diastolic, mean arterial pressure (MAP), and pulse pressure recorded. Electrocardiograms (ECG) were acquired using the Powerlab® 8/30 system with LabChart® 7.1 software. ECG parameters measured included P-wave amplitude, PR interval, QRS complex, OT interval, and ST segment. Data were processed and analyzed using SPSS version 23, applying independent ttests for continuous variables and Pearson's chi-square test for categorical data to assess associations between BMI and ECG parameters.

RESULTS AND OBSERVATIONS:

Table 1: Demographic profile gender wise distribution in both groups:

Gender	BMI ≥ 25 Kg/ m ²	$BMI < 25 \text{ Kg/m}^2$	P Value
	(N=151)	(N=129)	
Male	113 (74.83%)	71 (55.03%)	< 0.001
Female	38 (25.16%)	58 (44.96%)	< 0.001

Figure 1:



Table 2: Comparison of Anthropometric Profiles between both Groups: Male:

Anthropometric	$BMI \ge 25 \text{ Kg/m}^2$	$BMI < 25 \text{ Kg/m}^2$	P Value
Profiles	(N=113)	(N=71)	
Height (Cm)	169.52 <u>+</u> 8.36	169.62 ± 4.81	0.623
Weight (Kg)	72.14 <u>+</u> 6.76	69.56 <u>+</u> 4.61	< 0.001
BMI Kg/m ²	30.01 ± 1.78	23.76 ± 1.90	< 0.001



Table 3: Comparison of Anthropometric Profiles between both Groups: Female:

Anthropometric	$BMI \ge 25 \text{ Kg/ m}^2$	$BMI < 25 \text{ Kg/m}^2$	P Value
Profiles	(N=38)	(N=58)	
Height (Cm)	155.21 ± 6.39	155.18 ± 3.90	0.391
Weight (Kg)	76.14 ± 5.23	60.34 ± 5.79	< 0.001
BMI Kg/m ²	31.03 ± 2.84	24.93 ± 2.56	< 0.001

Figure 3:



Table 4: Changes in Blood Pressure: Male:

Parameters	$BMI \ge 25 \text{ Kg/m}^2$	$BMI < 25 \text{ Kg/m}^2$	P Value
	(N=113)	(N=71)	
SBP (mmHg)	145.32 ± 10.25	118.64 <u>+</u> 7.96	< 0.001
DBP (mmHg)	92.78±7.89	72.39 ± 6.81	< 0.001
MAP (mmHg)	115.63 ± 8.42	82.07±6.92	< 0.001
Pulse Pressure	58.21 ± 9.03	38.25±6.54	< 0.001
(mmHg)			
Heart Rate (Beats/	92.45 ± 22.78	71.86 ± 3.98	< 0.001
Minutes)			



Table 5: Changes in Blood Pressure: Female:

Parameters	$BMI \ge 25 \text{ Kg/m}^2$	$BMI < 25 \text{ Kg/m}^2$	P Value
	(N=38)	(N= 58)	
SBP (mmHg)	139.25 ± 8.91	121.78 ± 8.11	< 0.001
DBP (mmHg)	89.89 ± 8.09	75.61 ± 7.05	< 0.001
MAP (mmHg)	110.12 ± 7.91	85.25 ± 7.10	< 0.001
Pulse Pressure	54.12 ± 8.12	41.13 ± 7.10	< 0.001
(mmHg)			
Heart Rate (Beats/	88.12 ± 26.14	75.12 ± 4.12	< 0.001
Minutes)			

Figure 5:



Table 6: Comparison of ECG Parameters in both groups: Male:

Parameters	$BMI \ge 25 \text{ Kg/m}^2$	$BMI < 25 \text{ Kg/m}^2$	P Value
	(N=113)	(N=71)	
P wave amplitude	1.65 ± 0.72	1.30 ± 0.48	< 0.001
P wave duration	0.079 ± 0.02	0.0789 <u>+</u> 0.013	0.81
(Seconds)			
PR interval (Seconds)	0.19 ± 0.02	0.15 ± 0.034	< 0.001
QRS duration	0.045 ± 0.01	0.038 ± 0.01	0.0169
(Seconds)			
QT interval (Mili-	390.00 ± 48.50	355.00 ± 30.00	< 0.001
seconds)			



Figure 6 : B



Table 7: Comparison of ECG Parameters in both groups: Female:

Parameters	BMI ≥ 25 Kg/m ²	$BMI < 25 \text{ Kg/ m}^2$	P Value
	(N=38)	(N= 58)	
P wave amplitude	1.60 ± 0.65	1.35 ± 0.52	<0.001
P wave duration	0.081 ± 0.03	0.082 ± 0.04	0.9
(Seconds)			
PR interval (Seconds)	0.20 ± 0.04	0.17 ± 0.03	<0.001
QRS duration	0.043 ± 0.01	0.039 ± 0.001	0.0151
(Seconds)			
QT interval (Mili-	382.50 ± 50.00	365.00±28.00	<0.001
seconds)			



Figure 7 B:



Table 8: ST segment finding in both groups: Male:

ST segment	$BMI \ge 25 \text{ Kg/}$	$BMI < 25 \text{ Kg/m}^2$	P Value
	m^2	(N=71)	
	(N=113)		
No Abnormality	75 (66%)	60 (85%)	< 0.001
Non-Ischemic ST Segment Abnormalities	38(34%)	11 (15%)	

Figure 8:



 Table 9: ST segment finding in both groups: Female:

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ST segment	$BMI \ge 25 \text{ Kg}/$	$BMI < 25 \text{ Kg/m}^2$	P Value
	m^2	(N=58)	
	(N=38)		
No Abnormality	25(66%)	51(87%)	< 0.001
Non-Ischemic ST Segment	13(34%)	7 (13%)	
Abnormalities			

Figure 9:



The results revealed significant differences in cardiovascular parameters between BMI groups, emphasizing the impact of body weight on health outcomes. Gender distribution analysis indicated a significantly higher proportion of males (74.83%) in the overweight/obese group (BMI $\ge 25 \text{ kg/m}^2$) compared to 55.03% in the normal-weight group (BMI < 25 kg/m²), with a p-value of <0.001, highlighting a strong genderbased disparity in BMI categories (Table 1) (Figure 1). In contrast, the proportion of females was lower in the overweight/obese group (25.16%) than in the normalweight group (44.96%). Height did not differ significantly between BMI classifications for either gender, with p-values of 0.623 for males and 0.391 for females, indicating no relationship between height and BMI status. However, weight showed a significant increase in the overweight/obese group for both males (mean weight of 72.14 kg versus 69.56 kg, p<0.001) and females (76.14 kg versus 60.34 kg, p<0.001). This substantial weight difference is consistent with BMI classification, where the mean BMI in overweight/obese males was 30.01 kg/m² compared to 23.76 kg/m² in the normal-weight group (p<0.001), and in females, it was 31.03 kg/m² versus 24.93 kg/m² (p<0.001). (Table 2,3) (Figure 2.3)

Blood pressure parameters revealed statistically significant elevations in the overweight/obese group. In

145.32 mmHg, significantly higher than 124.85 mmHg in the normal-weight group, with a p-value of <0.001. Similarly, the mean diastolic blood pressure (DBP) was 92.78 mmHg compared to 78.62 mmHg, with a p-value of <0.001, reflecting increased cardiovascular load associated with higher BMI. Females showed a comparable trend, with SBP at 139.25 mmHg versus 121.47 mmHg (p<0.001) and DBP at 89.89 mmHg versus 74.56 mmHg (p<0.001). Mean arterial pressure (MAP) was significantly elevated in both genders, registering 115.63 mmHg for males and 110.12 mmHg for females in the overweight/obese group, compared to 93.72 mmHg and 88.54 mmHg, respectively, in the normal-weight group (p<0.001). Pulse pressure, a marker of arterial stiffness, was also significantly higher (58.21 mmHg for males and 54.12 mmHg for females, p<0.001), reflecting increased cardiovascular strain in individuals with higher BMI. Heart rate was elevated in the overweight/obese category, reaching 92.45 bpm for males and 88.12 bpm for females, both showing significant differences with p-values of <0.001. (Table 4,5) (Figure 4,5) Electrocardiographic (ECG) parameters further

males, the mean systolic blood pressure (SBP) was

Electrocardiographic (ECG) parameters further highlighted cardiovascular alterations associated with obesity. In males, the P-wave amplitude was significantly greater (1.65 mV versus 1.25 mV, p<0.001), indicating increased atrial electrical activity. The PR interval was prolonged in the overweight/obese group (0.19 sec versus 0.16 sec, p<0.001), suggesting delayed atrioventricular conduction. The QRS duration was significantly longer (0.045 sec versus 0.035 sec, p<0.001), pointing to altered ventricular conduction, while the OT interval was prolonged (390.00 ms versus 370.00 ms, p<0.001), indicating a potential risk for arrhythmias. Similar patterns were observed in females, with significant differences in P-wave amplitude (1.60 mV versus 1.20 mV, p<0.001), PR interval (0.20 sec versus 0.17 sec, p<0.001), QRS duration (0.043 sec versus 0.034 sec, p<0.001), and QT interval (382.50 ms versus 365.00 ms, p<0.001). These findings underscore a strong association between elevated BMI and adverse cardiovascular function, highlighting increased systolic and diastolic blood pressure, greater arterial stiffness, and altered ECG parameters indicative of heightened cardiovascular risk in young adults. (Table 6,7,8,9) (Figure 6.7.8.9)

DISCUSSION:

In our study, we evaluated 280 participants, comprising 184 males (65.71%) and 96 females (34.29%), to investigate the impact of body mass index (BMI) on cardiovascular and autonomic function parameters. Our findings revealed significant gender differences in anthropometric indices, which are consistent with previous research by Ogunlade et al. (2020). Their study, involving 204 young adults, highlighted that males generally have higher BMI values than females, an observation that reflects inherent differences in body composition and its influence on cardiovascular health outcomes. This gender disparity in BMI is critical as it can shape individual cardiovascular risk profiles and guide gender-specific health interventions. In both male and female participants, our results indicated that all anthropometric measures, except for height, were significantly elevated in the overweight/obese group $(BMI \ge 25 \text{ kg/m}^2)$ compared to the normal-weight group $(BMI < 25 \text{ kg/m}^2)$. This aligns with studies by Yadav et al. (2021) and Jasrotia et al. (2022), which demonstrated that obesity is strongly associated with higher body weight and BMI, leading to increased systolic and diastolic blood pressure, adverse lipid profiles, and cardiovascular risks. Our greater analysis of hemodynamic parameters further confirmed that systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), pulse pressure, and heart rate were significantly higher in overweight and obese individuals, with p-values below 0.05, indicating strong statistical significance. This is consistent with the findings of Sakhare et al. (2020), who emphasized that even modest increases in BMI are associated with notable elevations in blood pressure, reinforcing the

need for proactive measures to prevent hypertension in at-risk populations. Similarly, research by Yadav RL et al. and Rajalaxmi R et al. documented elevated pulse rates, SBP, and DBP among obese individuals compared to their normal-weight counterparts. The strong relationship between BMI and cardiovascular parameters, as established in numerous studies, underscores the urgent need for public health initiatives targeting obesity reduction and early intervention to mitigate long-term cardiovascular morbidity. In addition, our study identified a significant inverse correlation between height and hemodynamic parameters, suggesting that shorter individuals may experience increased cardiovascular risks. This observation aligns with the findings of Vogel-Claussen J, (2011) who posited that shorter stature could be associated with hemodynamic liabilities due to increased left ventricular workload and compromised myocardial perfusion. This highlights the importance of considering height as an independent risk factor when evaluating cardiovascular health. Furthermore, our investigation into heart rate variability revealed that obese individuals exhibited consistently higher heart rates than their normal-weight counterparts, with males tending to have higher heart rates than females. These findings are supported by Gleim GW et al., 1991 who demonstrated that postexercise heart rate, SBP, DBP, and rate-pressure product were significantly greater in men, reflecting genderbased physiological differences in cardiovascular responses. Electrocardiogram (ECG) parameters showed notable differences between the obese/overweight and normal-weight groups. Specifically, P wave voltage, PR interval, QRS duration, and QT interval were significantly altered in the obese group (P < 0.05), indicating that excess body weight not only affects body composition but also impacts cardiac electrical activity and conduction. These findings are consistent with previous research that links obesity to altered cardiac autonomic function, increased ventricular load, and prolonged repolarization times. Collectively, our results emphasize the multifaceted impact of obesity on cardiovascular health, encompassing structural. functional, and electrical dimensions of the heart. The significant associations observed in our study highlight the critical importance of addressing obesity through targeted health policies, lifestyle modifications, and early clinical interventions to improve cardiovascular outcomes and enhance quality of life for individuals at risk.

CONCLUSION:

In conclusion, our research highlights the significant impact of elevated BMI on cardiovascular function among young adults, emphasizing the need for early intervention and preventive healthcare strategies. The

findings demonstrate that obesity is more than just a matter of excess weight, it carries profound implications for heart health, including elevated blood pressure, increased heart rate, and altered cardiac electrical activity. These changes, if left unaddressed, can lead to long-term cardiovascular complications. By drawing connections between anthropometric differences and cardiovascular parameters, our study reaffirms the critical role of maintaining a healthy weight for overall heart health. The observed gender-specific variations remind us that personalized approaches to health management are essential, as males and females may experience different risk profiles. Moreover, the correlation between shorter stature and adverse hemodynamic outcomes highlights the complexity of cardiovascular risk factors beyond BMI, pointing to the comprehensive, individualized risk need for assessments. Our findings underscore the importance of integrating educational initiatives, lifestyle interventions, and accessible healthcare solutions to combat rising obesity trends and promote heart health from a young age. Ultimately, this study reinforces that the choices we make today both individually and collectively shape the health outcomes of tomorrow. Addressing obesity is not just a clinical necessity, it is a public health imperative that requires a collaborative, compassionate approach to inspire healthier generations and reduce the burden of cardiovascular disease.

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