e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2023; 15(8); 415-419

Original Research Article

Cross-Sectional Observational Research on Effect of Obesity on Electrocardiographic P-Wave Dispersion among Healthy Adults

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Received: 04-05-2023 Revised: 10-06-2023 / Accepted: 18-07-2023

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Conflict of interest: Nil

Abstract

Aim: The aim of the present study was to investigate the dispersion of P-wave in healthy young adults.

Methods: The study was conducted in the Department of Physiology for 10 months. Design of the study was cross-sectional observational and a total 100 young healthy adults both male and female aged between 18 to 40 years participated voluntarily in the study. Written informed consents were taken from all the participants after explaining the study protocol.

Results: There was no significant difference in age, sex and height between obese and non –obese group but a significant difference were found in weight and BMI between groups. Obese group had higher SBP, DBP, HR, maximum P-wave duration, minimum P-wave duration and P-wave dispersion compared to non-obese and statistically significant (p<0.001) was found.

Conclusion: It can be concluded that apparently healthy obese individuals may have higher anthropometric values and abnormal P—wave findings. Hence the present study gives an insight on the variations in P-wave in healthy adults who are obese and thereby helps in creating awareness so that; they can change their lifestyle in order to prevent the onset of the deleterious effects of obesity on their health.

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

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Introduction

Obesity is one of independent risk factors for development of cardiovascular diseases, including essential hypertension and myocardial ischemia, [1–3] and is also associated with sleep apnea syndrome [4] and insulin resistance. [5] It is well known that obesity is associated with left atrial enlargement and left ventricular filling abnormalities, [6] both known predictor for atrial fibrillation. Several studies suggested that obese subjects are associated with impaired heart rate variability. [7,8]

The world Health Organization (WHO) classified obesity as follows. BMI of 18 - 24.9 kg/m2 is considered normal weight, a BMI of 25.0-29.9 kg/m2 is considered overweight and a BMI of 30 kg/m2 or higher is considered obesity. [9] Obesity is often associated with many health consequences such as diabetes, hypertension, dyslipidemia, ischemic heart diseases, obstructive sleep apnoea, stroke, premature death, osteoporosis and a reduction of the overall quality of life. [10] Obesity causing changes in cardiac morphology such as LV hypertrophy and right ventricular hypertrophy are

well established. [11,12] However along with the changes in cardiac anatomy, obesity may also alter the electrocardiogram (ECG). According to Seyfeli et al., P-wave changes are highly specific in screening healthy obese individuals for the risk of cardiovascular diseases. 13 P-wave dispersion, which is the difference between maximum and minimum P-wave duration, has been recently defined as a new electrographic marker for the prediction of atrial fibrillation (AF). [13,14]

Although, in the Framingham Heart Study [15] body mass index (BMI) was not defined as an independent risk factor for AF, according to Wang et al. obesity has proved as an important, potential risk factor for AF. [16] The association of obesity with subsequent development of AF persists even after accounting for the influence of concominant conditions such as hypertension, diabetes mellitus myocardial infarction. and [17] electrocardiogram is a simple representation of the electrical activity of the heart muscle during the cardiac cycle. Recording of ECG is one of the easiest, cheap and reliable methods of assessing

cardiovascular function. Studies have shown that obesity induces changes in the normal ECG pattern, in healthy young women but the results have been inconsistent. [18,19]

The aim of the present study was to investigate the dispersion of P—wave in healthy young adults.

Materials and Methods

The study was conducted in the Department of Physiology, Patna Medical College, Patna, Bihar, India for 10 months. Design of the study was cross - sectional observational and a total 100 young healthy adults both male and female aged between 18 to 40 years participated voluntarily in the study. Written informed consents were taken from all the participants after explaining the study protocol.

Subjects were divided into two groups based on the BMI:

Group A – Normal/Non-obese (BMI: 18.5 - 24.99 kg/m2) = 50

Group B-Obese (BMI > 30 kg/m2) = 50

Subjects with history of cardiovascular disease, respiratory disease, thyroid disorder, diabetes, smoking, neuropsychiatric disorder, menstrual abnormality etc were excluded from study.

Methods of collection of data:

Measurement of BMI:

Body weight was measured on portable weighing machine without shoes and lightly clothed, and height was measured in barefoot using stadiometer. The subject stood against a standard meter scale, ears and the infra— orbital margins lay in one horizontal plane. Body weight was recorded in kilograms on an empty bladder and before lunch. BMI was calculated as body weight in kilogram divided by the square of the body height in meters.

[BMI = Weight (kg) / Height (m2)]

Measurement of Blood Pressure

Blood pressure (systolic blood pressure and diastolic blood pressure) was recorded in supine position in the right upper arm after the subject had rested for at least 5 minutes with standard mercury sphygmomanometer to the nearest 2 mmHg.

Electrocardiographic recording

The electrocardiographic recording was done by using 3- channel ECG machine by Medicaid India. To avoid from diurnal variations, we took ECG recordings of all subjects at the same time interval (10:00 am - 12 noon). [20] The speed of ECG paper was 25 mm/sec and the voltage was 1mv/cm. A resting ECG was recorded in lying posture after duly assuring them the non- invasive nature of the procedure and after resting of 10 min in a well ventilated quiet room. The subject's chest, forearms and legs were uncovered. Objects such as electronic gadgets, metallic ornaments etc. were removed to avoid interference. Location for placing electrodes on arms and legs was selected by choosing a place where there was minimum movement. Sufficient quantity of ECG gel was applied approximately 2cm on the skin at the chosen location to ensure good electrical contacts. Limb electrodes were clipped to subject's skin to give proper contact. Chest leads were placed over six different locations. Care was taken so that gel does not smear between the chest electrode sites.

e-ISSN: 0976-822X, p-ISSN: 2961-6042

Measurement of P-wave dispersion (Pd)

All ECG papers were scanned and digital files were created. Then after doing 200% magnification P-wave duration was measured using Adobe Photoshop-7 software. Pd was derived by subtracting the minimum P-wave duration from the maximum in any of the 12 ECG leads. P-wave onset was determined as the initial deflection from the isoelectric baseline defined by the T-P segment and the P-wave offset was defined as the junction of the end of the P wave and its return to baseline. [21]

Statistical Analysis:

The data was compiled in Microsoft excel and analysed using SPSS (Statistical Package for Social Sciences) version 20. The variables were expressed as mean and standard deviation, and P value <0.05 was considered statistically significant. Independent sample t-test was used to compare the results of obese to non- obese control group subjects.

Results

Table 1: Comparison of baseline anthropometric data between non-obese and obese group

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	Group A	Group B(Obese)(N=40)	
Parameters	(Non-obese) (N=50)		p– value
Age (Years)	24.16±4.76	28.82±3.37	0.999
Weight (Kg)	58.52±7.83	82.28±7.63	< 0.001
Height (meter)	1.62±0.08	1.58±0.04	0.859
BMI (Kg/m ²)	22.18±1.86	32.78±2.06	< 0.001

There was no significant difference in age, sex and height between obese and non –obese group but a significant difference were found in weight and BMI between groups.

Variables Group A(Non-obese) **Group B(Obese)** p-value SBP (mm of Hg) 112.28 ± 7.83 126.14±6.04 < 0.001 DBP (mm of Hg) 76.64±5.08 82.28±3.67 < 0.001 HR(b/m)79.51±4.16 88.52 ± 8.92 < 0.001 P max(ms) 87.63 ± 14.76 114.16±6.34 < 0.001 $70.12 \pm \overline{7.43}$ P min(ms) 55.75 ± 10.52 < 0.001 35.15±5.92 47.13±5.45 < 0.001 P d(ms)

Table 2: Baseline assessment of cardiovascular parameters (Blood pressure, Heart rate and P-wave duration and dispersion)

Obese group had higher SBP, DBP, HR, maximum P-wave duration, minimum P-wave duration and P-wave dispersion compared to non-obese and statistical significant (p<0.001) was found.

Discussion

Obesity is defined as a disease process in which excess body fat has accumulated to an extent that health may be adversely affected. According to WHO classification of body mass index (BMI) a person whose BMI is more than or equal to 30 Kg/m2 is obese and when BMI is between 18.5 to 24.99 then the person is considered normal. [22] Obesity is the first wave of a defined cluster of non-communicable diseases called 'New World Syndrome's creating an enormous socioeconomic and public health burden. [23] It has a strong impact on cardiovascular changes which is manifested in electrocardiogram (ECG). [24] Currently it is a serious public health problem with established cardiovascular co-morbidities and a major cause of sudden death in developed as well as developing countries.25 According to the National Family Health Survey-4 (NFHS-4) in 2015- 16 conducted by Ministry of Health and Family Welfare (MOHFW) in India, the percentage of men and women aged 15-49 years who are obese are 19% and 21% respectively. [25] In a large prospective study 'Framingham Heart study' there is evidence for inclusion of obesity as a major modifiable cardiovascular risk factor by American Heart Association and also sudden cardiac death has been reported 40 times higher in obese men and women. [26]

There was no significant difference in age, sex and height between obese and non –obese group but a significant difference were found in weight and BMI between groups. Obese group had higher SBP, DBP, HR, maximum P-wave duration, minimum P-wave duration and P-wave dispersion compared to non-obese and statistical significant (p<0.001) was found. Wang et al [27] previously have shown that obesity is a risk factor for AF, and they observed that obesity was associated with a 50% increase in the risk of AF. Furthermore, Frost et al. have suggested that AF and flutter should be added to the list of diseases caused by overweight and obesity. [28] In our study, obese women had

higher blood pressure,max. P-wave duration, and P-wave dispersion compared with non-obese women. While the high blood pressure in obesity is well established, the left atrial enlargement, which is an important precursor of AF, may contribute to the increase in P-wave duration and P-wave dispersion associated with obesity. [29] Some studies have shown that BMI is one of the most powerful determinants of left atrial size. [30,31]

Left atrial enlargement leads to atrial fibrillation which contributes to increase in the P wave duration. In obese individuals. left atrial enlargement and electrical instability may be caused by elevated plasma volume, ventricular diastolic dysfunction and enhanced neurohormonal activity. In addition, the autonomic control of the heart is abnormal in obese subjects due to prevalence of sympathetic over parasympathetic limb of the autonomic balance. This affects intraatrial and interatrial conduction times and leave them prone to develop atrial arrhythmias, such as atrial fibrillation. Duru and his colleagues noted that P wave duration and dispersion significantly decreased after substantial (10%) weight loss and the decrease in the level of P wave dispersion clearly correlated with the percentage of weight loss. [32]

Conclusion

Obesity leads to significant increase in systolic blood pressure, diastolic blood pressure and heart rate, thus increasing the risk of coronary heart disease and hypertension in these subjects. Prolongation of the P-wave duration increases the possibility of left atrial enlargement and atrial fibrillation. Thus, it can be concluded that apparently healthy obese individuals may have higher anthropometric values and abnormal P-wave findings. Hence the present study gives an insight on the variations in P-wave in healthy adults who are obese and thereby helps in creating awareness so that; they can change their lifestyle in order to prevent the onset of the deleterious effects of obesity on their health.

References

1. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk

- factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation. 1983 May;67(5):968-77
- 2. Kannel WB, Plehn JF, Cupples LA. Cardiac failure and sudden death in the Framingham Study. American heart journal. 1988 Apr 1;115(4):869-75.
- 3. de Divitiis OR, Fazio SE, Petitto M, Maddalena G, Contaldo F, Mancini M. Obesity and cardiac function. Circulation. 1981 Sep;64(3):477-82.
- Rajala R, Partinen M, Sane T, Pelkonen R, Huikuri K, Seppäläinen AM. Obstructive sleep apnoea syndrome in morbidly obese patients. Journal of internal medicine. 1991 Aug; 230(2):125-9.
- Galinier M, Fourcade J, Ley N, Boveda S, Solera S, Solera ML, Massabuau P, Elhabaj S, Fauvel JM, Valdiguie P, Bounhoure JP. Hyperinsulinism, heart rate variability and circadian variation of arterial pressure in obese hypertensive patients. Archives des maladies du coeur et des vaisseaux. 1999 Aug 1; 92(8):1105-9.
- 6. Alpert MA, Terry BE, Kelly DL. Effect of weight loss on cardiac chamber size, wall thickness and left ventricular function in morbid obesity. The American journal of cardiology. 1985 Mar 1;55(6):783-6.
- 7. Mureddu GF, de Simone G, Greco R, Rosato GF, Contaldo F. Left ventricular filling pattern in uncomplicated obesity. The American journal of cardiology. 1996 Mar 1;77(7):509-14
- 8. Rabbia F, Silke B, Conterno A, Grosso T, De Vito B, Rabbone I, Chiandussi L, Veglio F. Assessment of cardiac autonomic modulation during adolescent obesity. Obesity research. 2003 Apr;11(4):541-8.
- 9. Harik-Khan Raida I, Wise Robert A, Fleg Jerome L: The effect of gender on the relationship between bodyfat distribution and lung function. J Clin Epidemiol 2001; 54(4):399-06.
- 10. Yusuf S, Hawken S, Ounpuu S, et al; INTERHEART Study Investigators. Obesity and the risk of myocardialinfarction in 27,000 participants from 52 countries: A case-control study.Lancet 2005;366:1640-49.
- National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults – The evidence report. National Institutes of Health. Obes Res 1998; 6:551-59.
- 12. Iacobellis G, Ribaudo MC, Leto G, Zappaterreno A, Vecci E, Di Mario U, Leonetti F. Influence of excess fat on cardiac morphology and function: study in

- uncomplicated obesity. Obesity research. 2002 Aug;10(8):767-73.
- 13. Seyfeli E, Duru M, Kuvandık G, Kaya H, Yalcin F. Effect of obesity on P-wave dispersion and QT dispersion in women. International journal of obesity. 2006 Jun;30(6):957-61.
- 14. Dilaveris PE, Gialafos EJ, Sideris SK, Theopistou AM, Andrikopoulos GK, Kyriakidis M, Gialafos JE, Toutouzas PK. Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. American heart journal. 1998 May 1;135(5):733-8.
- 15. Benjamin EJ, Levy D, Vaziri SM, D'Agostino RB, Belanger AJ, Wolf PA. Independent risk factors for atrial fibrillation in a population-based cohort: the Framingham Heart Study. Jama. 1994 Mar 16;271(11):840-4.
- Wang TJ, Parise H, Levy D, D'Agostino RB, Wolf PA, Vasan RS, Benjamin EJ. Obesity and the risk of new-onset atrial fibrillation. Jama. 2004 Nov 24;292(20):2471-7.
- 17. Warnes CA, Roberts WC. The heart in massive (more than 300 pounds or 136 kilograms) obesity: analysis of 12 patients studied at necropsy. The American journal of cardiology. 1984 Nov 1;54(8):1087-91.
- 18. Moss AJ. Measurement of the QT interval and the risk associated with QTc interval prolongation: a review. The American journal of cardiology. 1993 Aug 26;72(6):B23-5.
- 19. Frank S, Colliver JA, Frank A. The electrocardiogram in obesity: statistical analysis of 1,029 patients. Journal of the American College of Cardiology. 1986 Feb 1:7(2):295-9.
- Yusuf M. Suraj, Muhammad A, Mabrouk, JosephO. Ayo. Comparative Study of Diurnal Variations In Electrocardiographic Intervals of Non-Athletes And Athletes In Zaria, Nigeria. International Journal of Scientific and Technology Research. 2013 Jun; 2:2277-8616.
- Andres Ricardo Perez-Riera, Luiz Carlos de Abreu, Raimundo Barbosa-Barros, Jose Grindler, Acacio Fernandes –Cardoso, Adrian Baranchuk.P- Wave Dispersion: An Update. Indian Pacing and Electrophysiology Journal. 2016; 16:126–133.
- 22. World Health organization: Obesity: Preventing and Managing the Global Epidemic Geneva: WHO;2004.
- 23. Pednekar MS, Hakama M, Hebert JR, Gupta PC. Association of body mass index with all-cause and cause-specific mortality: findings from a prospective cohort study in Mumbai (Bombay), India. International journal of epidemiology. 2008 Jun 1;37(3):524-35.
- 24. Wormser D, Kaptoge S, Di Angelantonio E, WoodAM, Pennells L, Thompson A, et al.

- Separate and combined associations of bodymass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. Lancet. 2011; 377:1085–1095.
- 25. Prentice AM. "The emerging epidemic of obesity in developing countries." Int J Epidemiol. 2006Feb;35(1):93-99.
- 26. Fourth National Family Health Survey. Mumbai: International Institute for Population Sciences. 2015-16. Eckel RH, Krauss RM. For the American Heart Association Nutrition Committee. Obesity as a major risk factor for coronary heart disease. Circulation 1998; 97:2099-2100.
- 27. Wang TJ, Parise H, Levy D, D'Agostino RB, Wolf PA, Vasan RS, Benjamin EJ. Obesity and the risk of new-onset atrial fibrillation. Jama. 2004 Nov 24;292(20):2471-7.
- 28. Frost L, Hune LJ, Vestergaard P. Overweight and obesity as risk factors for atrial fibrillation

- or flutter: the Danish Diet, Cancer, and Health Study. The American journal of medicine. 2005 May 1:118(5):489-95.
- 29. Vaziri SM, Larson MG, Lauer MS, Benjamin EJ, Levy D. Influence of blood pressure on left atrial size: the Framingham Heart Study. Hypertension. 1995 Jun;25(6):1155-60.
- 30. Pritchett AM, Jacobsen SJ, Mahoney DW, Rodeheffer RJ, Bailey KR, Redfield MM. Left atrial volume as an index ofleft atrial size: a population-based study. Journal of the American College of Cardiology. 2003 Mar 19:41(6):1036-43.
- 31. Vaziri SM, Larson MG, Benjamin EJ, Levy D. Echocardiographic predictors of nonrheumatic atrial fibrillation. The Framingham Heart Study. Circulation. 1994 Feb;89(2):724-30.
- 32. Duru M, Seyfeli E, Kuvandik G, Kaya H, Yalcin F. Effect of weight loss on P wave dispersion in obese subjects. Obesity. 2006 Aug;14(8):1378-82.