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AN ELECTROCARDIOGRAPHIC ABNORMALITY IN OBESITY: A STUDY AT TERTIARY CARE HOSPITAL FROM CENTRAL INDIA

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ABSTRACT

Obesity is associated with a wide variety of electrocardiographic (ECG) abnormalities. Most of these reflect alterations in cardiac morphology. Some serve as markers of risk for sudden death. Key ECG abnormalities or alterations occurring with disproportionately high frequency in obese subjects include: leftward shifts of the P wave, QRS and T wave axes, various changes in P wave morphology, low QRS voltage, various markers of left ventricular hypertrophy (particularly the Cornell voltage and product), T wave flattening in the inferior and lateral leads, lengthening of the corrected QT interval and prolonged QT interval duration. Alterations in the signal-averaged ECG and in heart rate variability may be arrhythmogenic. Cardiac arrhythmias have been described in obese subjects but are often accompanied by left ventricular hypertrophy or the sleep apnea syndrome. Many of these ECG abnormalities are reversible with substantial weight loss. Thus, obesity is associated with a wide variety of ECG abnormalities, many of which are corrected by weight loss. The electrocardiogram of 515 obese subjects was correlated with the severity of obesity and with age, sex and blood pressure. The heart rate, PR interval, QRS duration, QTc interval and voltage (R + S or Q wave in leads I, II and III) increased and the QRS vector shifted to the left with increasing obesity. These changes were independent of age, sex and blood pressure. Bradycardia was present in 19% of the patients, but tachycardia in only 0.5%. ST and T wave abnormalities were present in 11%, correlating better with increasing age and bloodpressure than with severity of obesity. This study evaluates the electrocardiogram in 515 patients with various degrees of obesity. The electrocardiographic measurements were related to the degree of obesity and the data were controlled for age, sex and blood pressure Most obese subjects with no clinical heart disease have a normal electrocardiogram. The heart rate increases with increasing obesity, but tachycardia is infrequent. ST segment and T wave abnormalities were present in approximately 11% of our patients, correlating better with increasing age and blood pressure than with degree of obesity. The PR interval and QRS duration increased with increasing obesity, but conduction abnormalities were very infrequent. The QTc interval was often prolonged in obese subjects and increased with increasing obesity. Low voltage was present in only 3.9% of the patients and QTc prolongation was present in 28.3%. The heart rate and QRS voltage increase with increasing obesity. Conduction is slowed, and the QRS vector shifts toward the left as percent overweight increases. These changes must be considered when evaluating both baseline electrocardiographic studies in obese patients and the changes seen during weight reduction.

INTRODUCTION

It has been suspected for more than 50 years[1,2] that obesity is related to heart disease in general and to the electrocardiogram in particular but systematic studies ex- amining this relation have been incomplete and inconsistent. Considerable controversy regarding the nature and degree of this relation has persisted in textbooks on cardiology[3,5] and electrocardiography[6,7,8] and in published journal reports.[9,10,11,12,13] Some of this controversy may have oc- curred because previous investigators characterized patients as obese or nonobese without considering the degree or severity of obesity or controlling for potentially confounding factors such as the patient's age, sex and blood pressure. In addition, because previous studies[1 , 2 , 9 , 1 0] provided data on relatively few patients. Weak trends may not have been statisticallydetected. ECG abnormalities are associated with an increased risk of adverse cardiovascular outcomes, including high resting heart rate (HR), prolonged PR interval, QRS duration and QT interval and abnormal shift in electrocardiographic axes.[1,2,3] A prospective study has reported that resting HR has a significant positive association with cardiovascular and all-cause mortalities.[2] A high HR is a strong indicator of cardiovascular mortality while a

low HR is associated with an improved outcome.[5] Prolonged PR interval has been associated with an increased risk of heart failure (HF), incident atrial fibrillation (AF), pacemaker implantation and mortality in the Framingham Heart Study.[4,5] Prolonged PR interval is also correlated with endothelial dysfunction and activation of vascular repair, which may be a cause for adverse cardiovascular outcomes.[6] QRS duration prolongation is a potential marker of cardiac structural and functional abnormalities, including left ventricular systolic dysfunction, that may predispose individuals to an increased risk of HF5,8 A Framingham follow-up study demonstrated that healthy individuals with prolonged QRS duration were at a higher risk of future pacemaker implantation.[7,8] Prolongation of corrected QT (QTc) interval predicts the risk for development of diabetes mellitus independently from conventional risk factors.[11] It also predicts the risk of sudden death in patients without evidence of cardiac dysfunction.[11] Electrocardiographic axes are also important markers in cardiovascular prognosis. For example, an abnormal P-wave axis is predictive of future onset of AF[12] and its verticalization is an effective electrocardiographic diagnostic tool for emphysema in the general population.[13] Frontal QRS axis serves as a sign for left ventricular hypertrophy and bundle branch block. The T axis is a general marker of repolarization abnormality. A follow-up study on older patients revealed that T-wave axis deviation is a strong independent risk indicator of fatal and non-fatal cardiac events.[14] Therefore, it is necessary to investigate factors affecting or causing these electrocardiographic changes.

Obesity is a strong independent risk factor for cardiovascular disease mortality and it predisposes patients to numerous cardiac complications, including hypertension, coronary heart disease, HF, stroke and sudden death.[15,16] The association between obesity and ECG has been investigated in previous studies. Obesity is closely associated with a wide variety of ECG abnormalities, including ischemic ECG observations, leftward shifts in electrocardiographic axes, markers of left ventricular hypertrophy and flattening of the T wave.[18] High resting HR and prolongation of PR interval and QRS duration are changes in electrocardiographic intervals induced by obesity.[19,20] A number of these ECG alterations may be reversed through weight loss, including rightward shift of the mean P-wave, QRS and T-wave axes.[22,26,28,31]

Numerous studies have reported a correlation between obesity and QTc interval prolongation[21,27,28,29] however, one study showed no correlation between body mass index (BMI) and QTc interval in a healthy population aged 22–25 years.[23,28,30,31,33] Similarly, the effect of obesity on QTc interval in children is also controversial.[24,25,28,29,30,31,32,33] Furthermore, ECG has been associated with age, gender, ethnicity and blood pressure.[26,27,29,30] However, the subjects in the majority of the previous studies were adults while the correlation between obesity and ECG in children and adolescents has not been studied in detail on a large-scale. An additional concern is that waist circumference (WC) and waist-to-height ratio (WHtR), measures of abdominal fat distribution, are more efficient risk factor predictors of cardiovascular disease than BMI in children. However, there have been no studies to date concerning the correlations between body fat distribution and ECG in children and adolescents.

Therefore, in this study, the differences in electrocardiographic intervals and axes associated with various degrees of obesity in normotensive and hypertensive children and adolescents were examined in a large-scale population. The final aims were to determine the effect of obesity on electrocardiographic variables besides the aforementioned possible risk factors and identify the possible association with body fat distribution in children and adolescents.

METHODS

515 patients with minimal to severe obesity were evaluated before they began a medically supervised weight reduction program. No patients were taking digoxin, antiarrhythmic drugs or calcium channel blockers. 03 patients were taking a beta-receptor blocking agent; this was discontinued 24 to 48 hours before the electrocar- diogram was recorded. 5 ***Weight.*** *Data were obtained in 515 patients: 437 (85%) women and 77 (15%) men with a mean age of 37 years (range 14 to 71). Most (93.3%) of the patients were between the ages of 20 and 59 years.Male patients were slightly older than the female patients.*

Weight was measured with the patient wearing indoor clothing and height was measured without shoes. Obesity was assessed using percent overweight, ponderal index, bodymass index, weight to height ratio and Benn's index. The percent overweight was determined by defining the midpoint weight for each height category[14] as normal for that particular height. The difference between the patient's weight and the normal weight divided by the normal weight and then mul-tiplied by 100 is defined as the patient's percent overweight. Using data from these patients. Colliver et al[15] demonstrated that the percent overweight, the ponderal index, the body mass index, the weight/height ratio and the Benn's index all measure the same thing therefore. only the percent overweight is referred to in our study.

Electrocardiogram. *Standard 12 lead electrocardiograms were recorded with the patient in the postprandial state and supine. Heart rate, PR interval, QRS interval, QT interval, QTc(QT/Yheart rate) interval and frontal plane QRS axis were measured using standard techniques.[16] Voltage was recorded as the maximal amplitude of the R and Q or S waves in leads I, II and III to identify low voltage patterns. ST segments and T waves were characterized as normal or*

abnormal using standard criteria. Blood pressure was taken three times at 1O minute intervals with the patient seated, using an obesity cuff when appropriate; the average of the three measurements was recorded.

Patients gave permission to have their medical records reviewed and to have the abstracted data included anonymously in our study.

RESULTS

Weight. *The mean initial weight for all patients was 87.5 kg (range 50 to 196.8). The range for women was 50 to 185.1 kg (mean 83.3) and the range for men was 62.4 to 196.8 kg (mean 107.4). The mean percent overweight for all patients was 51.5%. A few patients with apparently normal weight were included in the study. Some of these subjects had a distorted preoccupation with their weight andhad self-perceived obesity and some were participating in a weight maintenance program.*

Blood pressure. *The mean initial blood pressure was 124.7/80.6 mm Hg. A systolic blood pressure greater than 150 or 170 mm Hg was present in 4.2 and 1.7% of patients, respectively. A diastolic blood pressure greater than 90 or 100 mm Hg was present in IO. I and 3.0% of patients, respectively.*

Rhythm on initial electrocardiogram. *Sinus rhythm was present in 379 patients (73.5%), sinus bradycardia in 98 patients (19.0%). Sinus tachycardia in only 3patients (0.5%) and sinus arrhythmia in 25 patients (4.8%). 11 patients (2.1%) had an abnormal rhythm; 7 (1.3%) had an atrioventricular junctional rhythm, 3 (0.6%) had an atrioventricular junctional bradycardia and (0.3%) hadatrial fibrillation.*

Heart rate (Table 1). *Heart rate increased with increasing percent overweight. The regression equation relating heart rate to increasing obesity is: heart rate = 63.62 + 0.076 (percent overweight). Thus, each 10% increase in obesity is associated with an average increase in heart rate of 0. 76 beats/min. This relation is linear and is independent of sex, age and blood pressure.*[17]

ST and T wave abnormalities. *Repolarization abnormalities were present in approximately 11% of patients. ST segment abnormalities were seen in 28 (10.6%) of patients and T wave changes were present in 116 (11.7%) of 995. The frequency of these abnormalities was similar for men and women and was independent of percent over- weight, but it increased with age and with increasing systolic and diastolic blood pressure.*

PR interval (Table 1). *The relation between the PR interval and percent overweight was small, but in view of the large number of patients studied, was statistically sig- nificant (r = 0.07, p = 0.0435). The regression equation for the PR interval related to percent overweight is: PR interval = 0.16 + 4.6 x 10-^{5} (percent overweight). AI 0% increase in obesity is manifested in an increase in PR interval of 0.5 ms. This effect of increasing obesity on PR*[17] *interval was found regardless of age, sex and blood pressure. The PR interval was abnormally short (<0. I 2 second) in 3 patients (0.6%) and abnormally prolonged (>0.20 sec-ond) in 16 patients (3.2%).*

QRS duration (Table 1). *There was a weak but statistically significant correlation between QRS duration and percent overweight (r = 0.07, p = 0.02). The regression equation for the entire sample is: QRS duration = 0.067 + 3.1 x 10-^{5} (percent overweight). The increase in QRS duration with increasing weight was present regardless of age, sex and blood pressure.*[16,17,18] *In 69 patients (6.7%) the QRS interval was prolonged (2:::0.09 ::S 0.12 second) in the range characterized as an intraventricular conduction delay. Only one patient (0.1%) had a prolonged QRS interval greater than 0.12 second consistent with a nonspecific intraventric- ular conduction block.*

QTc interval (Tables 1 and 2). *The QTc interval tended to increase slightly as percent overweight increased. The regression equation is: QTc interval = 0.40 + I x 10-^{4}*

Table I: Regression Equations Relating Electrocardiographic Variable to Percent Overweight
Correlation Coefficient

		I	*p Value*
Heart rate	*63.62 + 0.076 (percent overweight)*	*0.22*	*0.0001*
PR mterval	*0.16 + 4.6 x 10-^{5} (percent overweight)*	*0.07*	*0.0435*
QRS duration	*0.067 + 3.1 X 10-^{5} (percent overweight)*	*0 07*	*0 0208*
QTc interval	*0.40 + I x 10-^{4} (percent overweight)*	*O.IO*	*0.0008*
QRS vector	*47.4 – 0.18 (percent overweight)*	*-0.22*	*0 0001*
QRS voltage	*23.2 + 0.03 (percent overweight)*	*0.15*	*0.0001*

QTc-Interval
(percent overweight) (r = 0.10 and p = 0.0008). A 10% increase in percent overweight can account for an increase in QTc interval of 1.0 ms. This change was independent of age, sex and blood pressure.[17,18,19] *The QTc interval was abnormally short (<0.35) in 7 patients (1.35%), but was prolonged in 145 patients (28.3%).*

QRS vector (Table 1). *The mean frontal plane QRS axis (QRS vector) tended to shift to the left as percent overweight increased (r = -0.22, p = 0.0001). The regression equation for the entire sample of patients relating the mean QRS vector and percent overweight is: QRS vector = 47.4 - 0.18 (percent overweight). A 10% increase in obesity is manifested by a shift of 1.8° in the QRS vector leftward (superiorly). Despite the tendency toward left axis shift with increasing obesity, only 3 patients (0. 7%) had a frontal plane vector that could be identified as left axis deviation (less than - 30°). Two patients (0.2%) had a frontal plane vector consistent with right axis deviation (> +90°). The relation of the QRS vector to age and blood pressure was not independent, but no consistent pattern could be discerned.*

QRS voltage (Table 1). *The QRS voltage. as measured by the sum of the S (or Q) and R waves in leads I, II and III, increased with increasing percent overweight (r = 0. 15, p = 0.0001). The regression equation is QRS voltage = 23.2 + 0.03 (percent overweight). Each 10% increase in percent overweight was associated with an increase in volt- age of0.3 mV. Voltage was not abnormally high (>60 mV) in any patient and was abnormally low (< 15 mV) in 18 patients (3.9%). In a random sample of 100 of the 1,029 patients. voltage was also measured using the sum of the S wave in lead V_1 and the R wave in lead V_5 or V_0. There was a highly significant (p = 0.0001) correlation between the two methods. Voltage was independent of age, sex and blood pressure.*[17,21,22,23]

The relation between heart rate, PR interval, QTc interval and QRS voltage with percent overweight was linear. The relation of QRS duration and mean frontal plane axis with percent overweight deviated slightly from a straight line. Although this deviation was statistically significant, it prob- ably is not clinically important.

DISCUSSION

The early studies on cardiologic abnormalities in obese persons have persisted unchallenged or only slightly modified until recently. Master et al[1] *reported that 86% of 97 obese patients had left axis deviation. Proger et al*[2] *noted that 71% of 55 patients with uncomplicated obesity had a left QRS axis. In these early studies, the definition of both obesity and left axis deviation was arbitrary and imprecise.*

Standard textbooks of cardiology report that obese patients have no electrocardiographic evidence of ventric- ular hypertrophy or other important abnormalities, and that low voltage of all complexes is very common.[4,8,9,10] *It has been noted*[4] *that the frontal plane QRS axis tends to be more leftward than normal and that the failure of the elec- trocardiogram to reflect myocardial hypertrophy in the grossly obese subject is probably related to the effect of the anatomy of the thorax on the transmission of electrical impulses.*

Previous studies have examined the electrocardiogram in[3,4,5,6] *patients with "severe" or "marked" or extreme obesity, without further defining obesity. In this study, we examined patients with obesity ranging from mild to extreme and correlated electrocardiographic findings with percent over- weight. Eisenstein et a*[6,7,10] *evaluated the electrocardio- graphic changes before and after weight reduction and dem- onstrated a tendency toward "normalization," but they did not assess the varying degrees of obesity and their effect on the electrocardiogram. Brohet et al*[13,14,15] *evaluated e!ectrocardiographic changes with weight reduction in 18 patients. They noted a decrease in QRS amplitude, and concluded that the changes were too slight to be clinically apparent. The effect of age and blood pressure on the elec- trocardiogram has been noted*[18.19.24.25.28]*, but these potentially confounding factors have been neglected in studies of pa- tients with obesity.*

Our study was designed to define the electrocardiographic changes in obesity and to evaluate these abnormalities at varying degrees of obesity. It was not intended to compare the electrocardiogram in obese persons with that of a normal population. There is, therefore, no "normal" or control group for comparison. Electrocardiographic values for normal persons have been clearly established in an abundance of observations over a wide variety of populations.

Rate and rhythm. The incidence of arrhythmias was not different from that detected in other studies[20.22.26.27] *examining nonobese patients free of clinically significant heart disease. Heart rate at rest did increase with increasing obesity but remained within the normal range.*

ST and T wave changes. Previous investigators have noted the frequency of ST segment and T wave abnormalities in obesity. Eisenstein et al.[10.30.31.32] *found inferolateral T wave flattening in 49% of 72 patients with a ·'tendency to nor- malization of the T wave" with weight loss. ST segment and T wave abnormalities were seen in 10.6 and 11.7% of patients, respectively, in this study. The frequency of these abnormalities increased with age and with increasing blood pressure, but was independent of increasing percent over- weight, suggesting that these nonspecific repolarization changes were not related to obesity.*

PR and QRS intervals. Although significant conduction abnormalities were infrequent in our study,

there was a slight but statistically significant tendency toward increasing PR interval and QRS duration with increasing obesity. Pipber- ger et al[10,33] noted slight increases in the PR and QRS intervals with increasing weight. The increases were small but statistically significant. Our study demonstrates that the increased PR interval and QRS duration were independent of age, sex and blood pressure.

Prolonged QTc interval. Prolongation of the QTc inter-val may have significant implications not only for defining a normal value in obese patients, but also for treating obesity and the associated complications. A small but statistically significant increase in QTc interval was recorded with in- creasing percent overweight. This was also independent of age, sex and blood pressure. A prolonged QTc interval (>0.42 second) was present in the initial electrocardiogram at rest in 28.3% of patients and a markedly prolonged QTc interval (>0.45 second) was noted in 4.0% of patients. This prolongation could not be attributed to electrolyte abnor- malities or drug effect. It may be a reflection of the pre- viously noted pattern of slowing or prolongation of con- duction, manifested by sinus bradycardia, PR prolongation and an increase in QRS duration with increasing obesity. Whereas the bradycardia and the slight PR and QRS pro- longation have no apparent clinical implications, the delay in repolarization manifested by QTc prolongation may have more profound clinical significance.

Intractable ventricular arrhythmias, polymorphic ven-tricular tachycardia (torsade de pointes), QT prolongation and sudden death have been reported in obese patients treated with collagen (liquid protein) or other very low calorie- modified fasting procedures.[21,25,29] It is possible that the reported ventricular arrhythmias and sudden death were in-dependent of the diet, the liquid protein or hypokalemia because, as our study demonstrates, QTc prolongation is so common in healthy obese subjects.

Left axis deviation. This study confirms the presence ofa tendency to left axis deviation that has been noted pre- viously.[1,2,4,7,9,12] The mean frontal QRS vector shifted slightly toward the left (superiorly) with increasing obesity. The trend is clinically very small but statistically significant. The asso- ciation was present in women only and was not entirely independent of age or blood pressure, but no consistent pattern was apparent.

The mean QRS frontal plane vector was normal for most patients. Only 3 patients (0.7%) had a QRS vector less than -30°, and 2 patients (0.2%) had a QRS vector of greater than +90°. The explanation for the progressive shift of the mean QRS vector toward the left may be related to increasing left ventricular hypertrophy or a change in the anatomic position of the heart in the thorax, or both.

Voltage. Previous investigators[3,4,8,12] [3,4,8,12] have commented on the low voltage seen in the electrocardiogram of obese patients, but there is no evidence in our data to sub- stantiate this assertion. Ishikawa[11,32,33] reported increasing voltage with increased weight in a nonobese patient group. The sum of the S (or Q) and R waves in leads I, II and III was chosen to measure the voltage because this measure- ment is standard and more sensitive for identifying low voltage than other indicators and has been used by other investigators.[10,21] The correlation with the precordial voltage (S wave in lead V_I plus R wave in lead V_5 or V_6) was highly significant ($p = 0.0001$) in a random sample of 50 patients. The electrocardiographic voltage is attenuated by its passage through a thickened and fat-laden chest wall and is related to many factors, including anatomic position of the heart in the thorax, degree of fatty infiltration of the heart, fatty infiltration of the myofibrils, selection of electro- cardiographic leads for measuring voltage, degree of as- sociated chronic lung disease and left ventricular muscle mass. In contrast to previous reports, our study documents an increasing amplitude of QRS voltage with increasing percent overweight.

CONCLUSION

Most obese subjects with no clinical heart disease have a normal electrocardiogram. The heart rate increases with increasing obesity, but tachycardia is infrequent. ST segment and T wave abnormalities were present in approximately 11% of our patients, correlating better with increasing age and blood pressure than with degree of obesity. The PR interval and QRS duration increased with increasing obesity, but conduction abnormalities were very infrequent. The QTc interval was often prolonged in obese subjects and increased with increasing obesity. The mean QRS vector shifted toward the left with increasing obesity, but intraventricular conduction abnormalities were infrequent. The voltage increased with increasing obesity.

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