The Electrocardiogram in Obesity: Statistical Analysis of 1,029 Patients

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The electrocardiogram in 1,029 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 blood pressure than with severity of obesity. Conduction abnormalities were infrequent. 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.

Methods

One thousand twenty-nine 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. Five patients were taking a beta-receptor blocking agent; this was discontinued 24 to 48 hours before the electrocardiogram was recorded.

Weight. Data were obtained on 1,029 patients: 874 (85%) women and 155 (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; the 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, body mass index, weight to height ratio and Benn’s index. The percent overweight was determined by defining the midpoint weight for each height category of the Metropolitan Life Insurance Company table (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 multiplied 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.

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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/√heart 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 10 minute intervals with the patient seated, using an obesity cuff when appropriate; the average of the three measurements was recorded.

This protocol was approved by the Springfield Committee on Research Involving Human Subjects. 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 and had 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 120 mm Hg was present in 10.1 and 3.0% of patients, respectively.

Rhythm on initial electrocardiogram. Sinus rhythm was present in 757 patients (73.5%), sinus bradycardia in 195 patients (19.0%), sinus tachycardia in only 5 patients (0.5%) and sinus arrhythmia in 50 patients (4.8%). Twenty-two patients (2.1%) had an abnormal rhythm; 13 (1.3%) had an atrioventricular junctional rhythm, 6 (0.6%) had an atrioventricular junctional bradycardia and three (0.3%) had atrial 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 105 (10.6%) of 995 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 overweight, 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 significant (r = 0.07, p = 0.0435). The regression equation for the PR interval related to percent overweight is: PR interval = 0.16 + 4.6 × 10⁻⁵ (percent overweight). A 10% increase in obesity is manifested in an increase in PR interval of 0.5 ms. This effect of increasing obesity on PR interval was found regardless of age, sex and blood pressure (17). The PR interval was abnormally short (<0.12 second) in 6 patients (0.6%) and abnormally prolonged (>0.20 second) in 33 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 × 10⁻⁵ (percent overweight). The increase in QRS duration with increasing weight was present regardless of age, sex and blood pressure (17). In 69 patients (6.7%) the QRS interval was prolonged (≥0.09 ≤ 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 intraventricular 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 + 1 × 10⁻⁴

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Limitations of previous studies. 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 (3) report that obese patients have no electrocardiographic evidence of ventricular hypertrophy or other important abnormalities, and that low voltage of all complexes is very common (4,8). It has been noted (4) that the frontal plane QRS axis tends to be more leftward than normal and that the failure of the electrocardiogram 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 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 overweight. Eisenstein et al. (10) evaluated the electrocardiographic changes before and after weight reduction and demonstrated a tendency toward “normalization,” but they did not assess the varying degrees of obesity and their effect on the electrocardiogram. Brohet et al. (13) evaluated electrocardiographic changes with weight reduction in 37 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 electrocardiogram has been noted (18,19), but these potentially confounding factors have been neglected in studies of patients with obesity.

Correlation with clinical data. 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) 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) found inferolateral T wave flattening in 49% of 144 patients with a "tendency to normalization 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 overweight, 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. Pipberger et al. (12) 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 interval 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 increasing 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 abnormalities or drug effect. It may be a reflection of the previously noted pattern of slowing or prolongation of conduction, manifested by sinus bradycardia, PR prolongation and an increase in QRS duration with increasing obesity. Whereas the bradycardia and the slight PR and QRS prolongation have no apparent clinical implications, the delay in repolarization manifested by QTc prolongation may have more profound clinical significance.

**Intractable ventricular arrhythmias,** polymorphic ventricular 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 (21,22). It is possible that the reported ventricular arrhythmias and sudden death were independent 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 of a tendency to left axis deviation that has been noted previously (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 in view of the large number of patients studied. The association 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 seven patients (0.7%) had a QRS vector less than -30°, and two 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) have commented on the low voltage seen in the electrocardiogram of obese patients, but there is no evidence in our data to substantiate this assertion. Ishikawa (11) 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 measurement 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 V1 plus R wave in lead V5 or V6) was highly significant (p = 0.0001) in a random sample of 100 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 electrocardiographic leads for measuring voltage, degree of associated 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.

**Clinical implications.** 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.

**References**

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