Novel Electrocardiographic Parameters of Altered Repolarization in Uncomplicated Overweight and Obesity

Annabella Braschi[1], Maurizio G. Abrignani[2], Vincenzo C. Francavilla[1] and Giuseppe Francavilla[1]

In addition to well-known electrocardiographic measurements, as QT, QT dispersion, and QT apex dispersion, new parameters such as Tpeak-Tend, Tpeak-Tend dispersion, and Tpeak-Tend/QT ratio have been recently introduced as indexes of increased arrhythmic risk. The aim of the present study was to test, in overweight and obese subjects not affected by conditions of comorbidity, the aforementioned markers of ventricular repolarization. We studied 60 athletic subjects with normal body weight (21 females and 39 males, BMI between 19 and 24, mean BMI 22.0 ± 2.0 kg/m², aged 14–64 years, mean age 32 ± 13.59) and 60 sedentary and overweight/obese subjects (34 overweight and 26 obese, 22 females, and 38 males, BMI between 26 and 55, mean BMI 30.7 ± 5.7 kg/m², aged 14–64, mean age 38 ± 14.49). Each subject underwent anthropometric measurements and a 12-lead electrocardiogram, from which the following different parameters were calculated: QT, corrected QT, QT dispersion, QT apex dispersion, Tpeak-Tend, Tpeak-Tend dispersion and Tpeak-Tend/QT ratio were calculated. The aforementioned repolarization markers resulted, respectively: 340.2 ± 25.1, 373.8 ± 25.9, 29 ± 16.2, 23.5 ± 14.6, 87.3 ± 12.8, 26.5 ± 16.8, and 0.22 ± 0.03 ms in control subjects and 362.5 ± 28.5, 397.4 ± 35.4, 34.5 ± 16.8, 30.7 ± 16.3, 90.5 ± 15.2, 27 ± 17.1, and 0.22 ± 0.04 ms in overweight/obese subjects. Neither uncomplicated obesity nor overweight were associated with a statistically significant difference in QT dispersion, QT apex dispersion, Tpeak-Tend, Tpeak-Tend dispersion, and Tpeak-Tend/QT ratio; QT and corrected QT were the only parameters that showed statistically significant variations between normal weight and overweight/obese subjects.

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INTRODUCTION

An excessive caloric intake and a low physical activity have as consequence the development of obesity, which, in turn, may lead to an increased risk of cardiovascular morbidity and mortality (1,2). In obese subjects, compared to normal weight subjects, it has been shown a lengthening of corrected QT (QTc) and QT dispersion (QTd) (3,4), parameters related to an increased incidence of ventricular tachyarrhythmias in various pathologies (5–10).

Interest has grown, recently, in new electrocardiographic parameters, as indirect indexes of ventricular repolarization; in particular the Tpeak-Tend (Tpe) interval, its dispersion (Tpe-d), and the relationship between Tpe interval and QT interval (Tpe/QT ratio) showed to be valid markers of arrhythmic vulnerability in coronary heart disease, in Brugada syndrome, in hypertrophic cardiomyopathy, and in long QT syndrome (11–15).

The aforementioned parameters have not yet been evaluated in overweight/obese subjects. The aim of this study was to verify their potential alteration in obesity without associated comorbid conditions.

METHODS AND PROCEDURES

A total of 120 subjects were studied, divided into two groups. The first group was composed of 60 healthy, athletic subjects with normal weight (BMI ≤24.9 kg/m²). They worked out at the gym three times a week for more than 60 min, engaging in aerobic and anerobic activities. The second group was composed of 60 sedentary, overweight (BMI between 25 and 29.9 kg/m²) and obese subjects (BMI ≥30 kg/m²). The first group comprised 21 women and 39 men with the following characteristics: BMI between 19 and 24 kg/m², mean BMI 22.0 ± 2.0 kg/m², aged 14–64 years, mean age 32 ± 13.59 years. The second group comprised 22 women and 38 men, 34 overweight and 26 obese subjects with the following characteristics: BMI between 26 and 55 kg/m², mean BMI 30.7 ± 5.7 kg/m², aged 14–64 years, mean age 38 ± 14.49 years. The subjects with a normal weight were recruited from the Division of Sports Medicine at Palermo University Hospital, the overweight and obese subjects came from a primary prevention ambulatory clinic. Nobody had a personal history of heart disease, hypertension, impaired glucose tolerance, diabetes, renal failure, hepatic or thyroid diseases. No one had a family history of long QT syndrome, electrolyte disorders

[1]Palermo University Hospital, Department of Internal Medicine, Cardiovascular Diseases and Nephro-urological Diseases, Palermo, Italy; [2]Operative Unit of Cardiology, S. Antonio Abate Hospital, Erice (Trapani), Italy. Correspondence: Annabella Braschi (annabellabraschi@alice.it)

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or was taking drugs known to affect electrocardiographic parameters. Exclusion criteria were: atrial fibrillation or flutter, pacemaker-rhythm, bundle branch block, atrioventricular block, ventricular pre-excitation, heart rate <50 bpm or above 100 bpm. All the 120 study participants were in sinus rhythm and none of them showed any previous criteria. Baseline characteristics are shown in **Table 1**.

Each subject underwent anthropometric measurements and a 12-lead electrocardiogramm, from which the following parameters were calculated: QT, QTc, QTd, QT apex dispersion (QTapex-d), Tpe, Tpe-d, and Tpe/QT ratio. Height measurements were done using a wall-mounted height scale, with 5-mm divisions, whereas weight was measured using a WUNDER-RE 300 balance. The BMI was calculated by the BMI formula: weight (kg)/height $(cm)^2$.

The obese and overweight subjects were weight-stable (<5% change from baseline body weight for 6 months before enrollment).

The electrocardiogramm was recorded with a standard digital recorder at a paper speed of 25 mm/s. The QT interval was measured from the beginning of the QRS to the end of the T wave (**Figure 1a**), defined as the intersection of the tangent to the downslope of the T wave and the isoelectric line (16), when U waves were present, the QT interval was measured to the nadir of the curve between the T and U waves (**Figure 1b**); QT interval length was assessed from lead D II, the QTc interval was obtained using Bazett's formula (17). A QTc interval longer than 440 ms for males and than 460 ms for females was considered prolonged. QT dispersion was determined as the difference between the maximum and minimum QT intervals (18), without using any formula to correct QTd for heart rate (19). The Tpe interval was measured in all precordial leads and obtained from the difference between QT interval and QT peak interval, measured from the beginning of the QRS to the apex of the T wave; in case of negative or biphasic T waves, QT peak was measured to the nadir of the T wave (**Figure 1c**). When a bifid T wave was present, the distance between the first and the second component was taken; if the time interval was ≤0.15 s the second component was interpreted as a part of the T wave (**Figure 1d**), otherwise the second component was considered U wave. Only two young subjects showed bifid T waves, seen in leads V2–V3. The presence of U waves was more common. To improve the reliability of T-wave offset determination, leads with low-amplitude T waves (<0.1 mV) were excluded from analysis. The leads in which the T wave was more frequently unreadable, because of a low amplitude, were the limb leads; however, all 120 subjects had at least 10 readable leads to assess the QTd and at least five readable precordial leads to assess the Tpe-d. The reported Tpe value is the maximum obtained in all precordial leads. The Tpe-d was defined as the difference between the maximum and the minimum Tpe interval in the precordial leads (12). The Tpe/QT ratio is the ratio of the interval from the peak to the end of the T wave divided by the interval from the onset of the Q wave to the end of the T wave and it was measured using V5 (14). The measurements were performed manually by an experienced observer who had no knowledge of the clinical data.

Statistical analysis

Statistical analysis was performed using R software (R Development Core Team 2009) (20).

The two groups were compared using a Student's *t*-test for nonpaired data. The predetermined α level was 0.01. Data are presented as mean ± s.d. To highlight the differences in the behavior of the studied parameters, the second group was further subdivided into two subgroups, considering separately overweight and obese subjects. An ANOVA was therefore performed, studying how the mean of every single variable changed in the three groups (normal weight, overweight, and obese). A Tukey's *post hoc* test was later done to evaluate from which difference the significance originated.

RESULTS

Mean values of QT, QTc, QTd, QTapex-d, Tpe, Tpe-d, and Tpe/QT ratio in the normal weight group and in the overweight/obese group are shown in **Table 2**. As regards QT and QTc, the difference was statistically significant, with a *P* equal to $9.67 \cdot 10^{-6}$ for the QT (340.2 ± 25.1 ms vs. 362.5 ± 28.5 ms) and equal to $4.54 \cdot 10^{-5}$ for the QTc (373.8 ± 25.9 vs. 397.4 ± 35.4). QTc behavior appeared to be closely correlated to BMI, as QTc increased with rising BMI; obese subjects showed a greater QTc value both compared to normal weight subjects and to overweight subjects (**Figure 2**). In the overweight/obese group

Table 1 Baseline characteristics of study participants

Variables	Normoweight	Overweight/obese	
Number of subjects	60	60	
Males	39	38	
Females	21	22	
Mean BMI (kg/m^2)	22.0 ± 2.0	30.7 ± 5.7	
Mean age (years)	32 ± 13.59	38 ± 14.49	$P > 0.01$
Mean systolic blood pressure (mm Hg)	115 ± 10.22	122 ± 8.87	$P < 0.01$
Mean diastolic blood pressure (mm Hg)	72 ± 6.97	74 ± 11.59	$P > 0.01$
Mean heart rate (bpm)	72.67 ± 10.35	73.05 ± 12.33	$P > 0.01$
Mean serum K^+ level (mEq/l)	4.23 ± 0.37	4.16 ± 0.35	$P > 0.01$
Mean serum Mg^{++} level (mg/dl)	2.01 ± 0.22	2.11 ± 0.19	$P > 0.01$
Mean serum Ca^{++} level (mg/dl)	9.01 ± 0.42	8.97 ± 0.28	$P > 0.01$

Data are shown as means ± s.d. *P* values were calculated by a *t*-test.

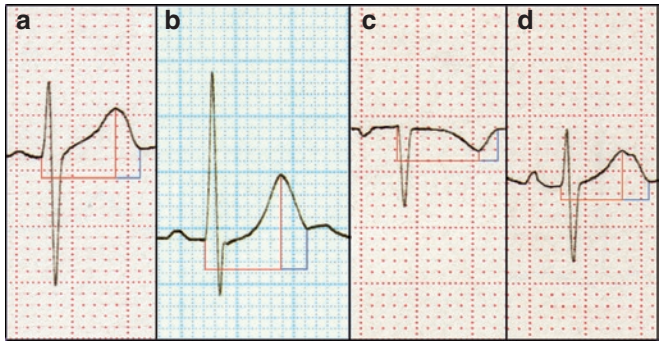

Figure 1 (**a**) In presence of positive T wave, the T-wave peak was identified with the apex of the parabola fitted to the T wave, whereas the QT end was measured at the intersection point between the tangent to the downslope of the T wave and the isoelectric line. The QT peak interval is shown in red, the Tpe in azure. (**b**) When U waves were present, the end of the T wave was measured to the nadir of the curve between the T and U waves. The QT peak interval is shown in red, the Tpe in azure. (**c**) When negative T wave was present, the apex of the T wave was measured to the nadir of the T wave. The QT peak interval is shown in red, the Tpe in azure. (**d**) Bifid T wave with distance between the first component (the highest peak, T1) and the second component (the lower peak, T2) ≤0.15, T peak is the former peak, the latter peak is a notch so QT peak was calculated from QRS onset to the peak of T1, T end was measured at the point of intersection of the downslope of T2 with the isoelectric baseline. The QT peak interval is shown in red, the Tpe in azure.

Table 2 Mean values of repolarization markers in the normoweight subjects and in the overweight/obese subjects

Groups	QT	QTc	QTd	QTapex-d	Tpe	Tpe-d	Tpe/QT
BMI ≤24.9	340.2 ± 25.1	373.8 ± 25.9	29 ± 16.2	23.5 ± 14.6	87.3 ± 12.8	26.5 ± 16.8	0.22 ± 0.03
BMI ≥25	362.5 ± 28.5	397.4 ± 35.4	34.5 ± 16.8	30.7 ± 16.3	90.5 ± 15.2	27 ± 17.1	0.22 ± 0.04
P	<0.01	<0.01	>0.01	=0.01	>0.01	>0.01	>0.01

QTc, corrected QT; QTd, QT dispersion; QTapex-d, QT apex dispersion; Tpe, Tpeak-Tend; Tpe-d, Tpeak-Tend dispersion; Tpe/QT, Tpeak-Tend/QT ratio.
Data are shown as means ± s.d. *P* values were calculated by a *t*-test.

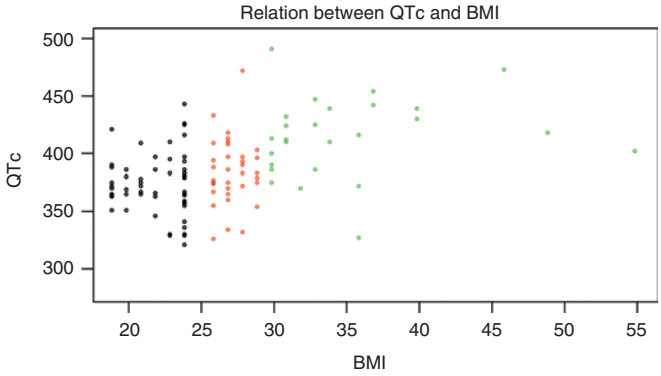

Figure 2 Normal weight subjects are shown in black, overweight subjects are shown in red, obese subjects are shown in green. Some patients in the overweight/obese group had a QTc interval higher than 440 ms, whereas none in the normal weight group showed an abnormal QTc.

a QTc ≥440 ms was evident in four men and a QTc ≥460 ms in two women for a total of six subjects (10% of the sample), only one was overweight, all the others had a BMI ≥30 kg/m^2. None of the normal weight subjects exceeded the cut-off values.

QTd (29 ± 16.2 ms vs. 34.5 ± 16.8 ms) and QTapex-d (23.5 ± 14.6 ms vs. 30.7 ± 16.3 ms) showed smaller differences, which were not statistically significant.

Regarding QTapex-d it was not possible to determine if the differences between the means were due to chance or not, in as much as the result obtained from test t was borderline ($P = 0.01$).

In the QTd distribution (**Figure 3a**), the middle 50% was between 20 and 40 ms in the normal weight group and between 20 and 45 ms in the overweight/obese group. The minimum value and the first quartile of the distributions were the same, whereas the values of the other quartiles were higher in the second group. The average was 29 ms (99% confidence interval (CI), 23.5–34.5) in the first group and 34.5 ms (99% CI, 28.8–40.2) in the second group.

In the QTapex-d distribution (**Figure 3b**) of the first group, excluding three outliers, the range was between 10 and 40 ms with a mean of 23.5 ms (99% CI, 18.6–28.4); in the second group the range was wider (0–60 ms) with a mean of 30.7 (99% CI, 25.1–36.2).

Tpe, Tpe-d, and Tpe/QT ratio values did not show statistically significant differences (respectively 87.3 ± 12.8 vs. 90.5 ± 15.2 ms, 26.5 ± 16.8 vs. 27 ± 17.1 ms and 0.22 ± 0.03 vs. 0.22 ± 0.04).

Tpe values (**Figure 3c**) were distributed in a rather wide range, the middle 50% was between 80 and 100 ms in both groups with the last quartile higher in the second group. The mean was 87.3 ms (99% CI, 83.0–91.7) in the first group and 90.5 ms (99% CI, 85.3–95.6) in the second group.

Regarding Tpe/QT (**Figure 3d**) there were no meaningful difference between the two distributions (mean of 0.22 with a 99% CI of 0.20–0.23 in both groups).

Comparing men and women, there was a statistically significant difference in both groups either for the QTc and/or for the Tpe_{max}. QTc was higher in women than in men with a $P < 0.01$ in both groups (394.1 ± 22.6 ms in women vs. 363.0 ± 19.1 ms in men in the first group and 418.6 ± 29.8 ms in women vs. 385.2 ± 31.7 ms in men in the second group).

On the contrary, Tpe_{max} was higher in men if compared with women with a $P < 0.01$ in subjects with a normal weight and a $P < 0.05$ in overweight/obese subjects (81.4 ± 10.6 ms in women vs. 90.5 ± 12.5 ms in men in the first group and 85.4 ± 12.6 ms in women vs. 93.4 ± 15.6 ms in men in the second group).

Thus, the subanalysis by gender showed longer QTc in women than in men, in contrast men had longer Tpe than women. No influence of gender on the other parameters was found.

To assess the influence of BMI on repolarization variables the study subjects were further divided, using the BMI as stratification variable, in three groups (normoweight, overweight, and obese) and a one way ANOVA was thus performed. Mean values and standard deviations of the studied parameters in the three groups are shown separately in **Table 3**. It was evident a great influence of BMI on QTc values, with a mean QTc which was significantly different among the three groups ($P = 2.15 \times 10^{-7}$). All the quartiles showed a growing positive trend (**Figure 4**). Confidence intervals for each mean were calculated using the Tukey's test. Normoweight subjects had a mean of 373.8 ms (99% CI, 355.74–392.08), overweight subjects had a mean of 384.2 ms (99% CI, 366.03–402.37), obese subjects had a mean of 414.7 ms (99% CI, 392.67–436.78).

At the *post hoc* Tukey's test, the difference among the averages was statistically significant between normal weight and obese subjects, between overweight and obese subjects, but not between normal weight and overweight subjects. It was noted, in fact, that mean and median values of QTc in overweight group approached more closely to those shown by normal weight subjects than obese subjects (**Figure 4**).

Regarding QTd and QTapex-d, the *P*-value of the ANOVA *F*-test appeared to be borderline, it was, respectively 0.0158 for the first parameter and 0.0160 for the latter. In the Tukey's *post hoc* tests no statistically significant difference was shown among mean values of the three groups, except for a borderline value obtained for both parameters considering normal weight

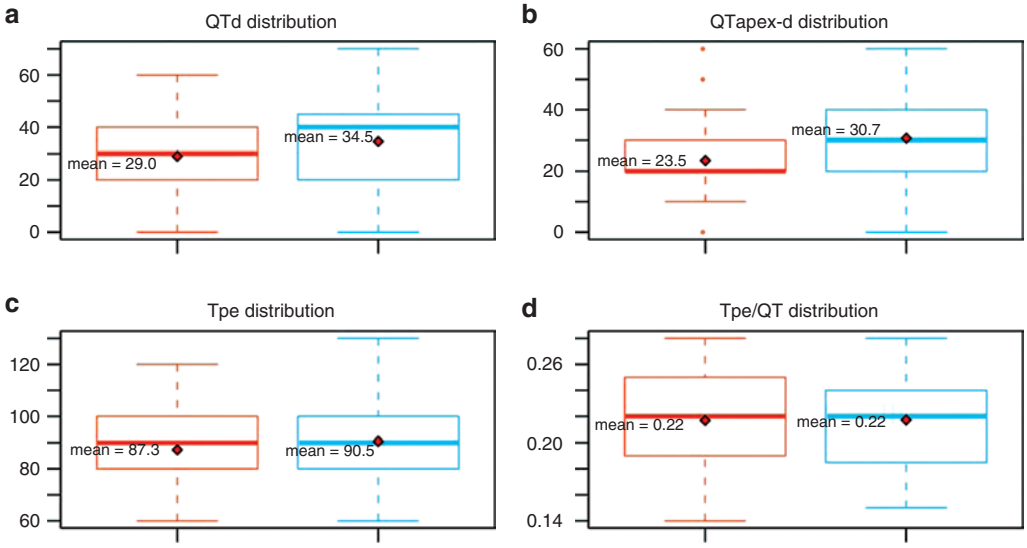

Figure 3 (**a**) Normal weight subjects are shown in red, overweight/obese subjects in azure. Values of QT dispersion in normoweight subjects (median 30 ms and mean 29 ms) and in overweigt/obese subjects (median 40 ms and mean 34.5 ms). (**b**) Normal weight subjects are shown in red, overweight/obese subjects in azure. Values of QT apex dispersion in normoweight subjects (median 20 ms and mean 23.5 ms) and in overweigt/obese subjects (median 30 ms and mean 30.7 ms). (**c**) Normal weight subjects are shown in red, overweight/obese subjects in azure. Tpeak-Tend values in normoweight subjects (median 90 ms and mean 87.3 ms) and in overweight/obese subjects (median 90 ms and mean 90.5 ms). (**d**) Normal weight subjects are shown in red, overweight/obese subjects in azure. There is a perfect coincidence of the means of the two groups which are both 0.22, with maximum values which do not exceed, in either case, 0.28. Also the value of the median coincides in the two distributions.

Table 3 Mean values of repolarization markers in the normoweight group, in the overweight group and in the obese group

Groups	QT	QTc	QTd	QTapex-d	Tpe	Tpe-d	Tpe/QT
BMI ≤24.9	340.2 ± 25.1	373.8 ± 25.9	29 ± 16.2	23.5 ± 14.6	87.3 ± 12.8	26.5 ± 16.8	0.22 ± 0.03
25 < BMI ≤ 29.9	360.0 ± 29.4	384.2 ± 28.8	31.2 ± 13.6	27.9 ± 16.8	92.0 ± 10.4	24.1 ± 15.6	0.22 ± 0.03
BMI ≥30	365.8 ± 26.4	414.7 ± 34.8	38.8 ± 19.0	34.2 ± 14.5	88.5 ± 19.5	30.8 ± 17.9	0.21 ± 0.04
P	<0.01	<0.01	>0.01	>0.01	>0.01	>0.01	>0.01

QTc, corrected QT; QTd, QT dispersion; QTapex-d, QT apex dispersion; Tpe, Tpeak-Tend; Tpe-d, Tpeak-Tend dispersion; Tpe/QT, Tpeak-Tend/QT ratio.
Data are shown as means ± s.d. *P* values were calculated by an ANOVA *F*-test.

vs. obese subjects. Mean QTd values in the normal weight group and in the overweight group were very close (29 ms vs. 31.2 ms), with a perfect coincidence of the median (30 ms); in the obese group the mean (38.8 ms) and the median (40 ms) diverged compared to the other two groups with a difference between the averages that reached almost the statistical significance comparing obese to normal weight subjects (38.8 ms vs. 29.0 ms). QTapex-d assumed a behavior very similar to that showed by QTd, with means of 23.5, 27.9, and 34.2 ms and medians of 20, 20, and 40 ms in the three groups. Regarding Tpe, Tpe-d and Tpe/QT ratio, no significant difference was evident among the groups ($P = 0.282$ for the first parameter, $P = 0.305$ for the second one and $P = 0.487$ for the third one).

DISCUSSION

A complex relationship exists among sedentary life, obesity, risk factors such as dyslipidemia, hypertension, diabetes mellitus, and cardiovascular morbidity and mortality (21–31); in particular, low levels of physical activity and elevated BMI result, independently, associated with high blood levels of cholesterol, fibrinogen, and C-reactive protein (22).

Obesity represents not only an independent risk factor for cardiovascular morbidity, but it is also a strong predictor of sudden cardiovascular death (23). Adipose cells product and release hormones, enzymes, and cytokines (24), which play a fundamental role in metabolic and cardiocirculatory homeostasis provoking a prothrombotic state (25) and a sub-clinical inflammation (26), which predispose to the onset of cardiovascular diseases. Even in absence of comorbid conditions and underlying organic heart pathologies, obesity is able to provoke alterations both in the morphology and in the electrophysiological properties of myocardial cells. Cardiac alterations due to obesity predispose to an elevated arrhythmic risk: obese subjects show, in fact, an increased incidence of both atrial fibrillation (28) and ventricular ectopy (29). Prevalence of premature ventricular beats in obese individuals with eccentric ventricular hypertrophy is 30 times greater than in normoweight subjects (29), with a positive correlation between BMI and ventricular ectopy (30). In patients with left ventricular dysfunction obesity leads to a worse postmyocardial infarction prognosis, increasing the risk of sudden death (31). In addition to left ventricular hypertrophy, both autonomic dysfunction

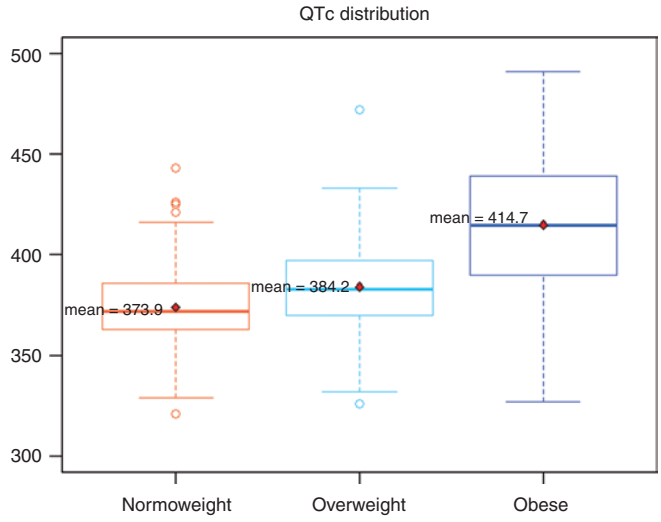

Figure 4 Normal weight subjects are shown in red, overweight subjects in azure, obese subjects in blue. QTc is distributed across higher values, passing from normal weight to overweight and from overweight to obese subjects. QTc is above normal range in the obese group.

with a reduced heart rate variability due to sympathetic predominance (32) and obstructive sleep apnea syndrome (very often associated with overweight and cardiac rhythm disturbances) (33), contribute to raise the risk of developing arrhythmias.

A relationship between QT parameters and ventricular tachyarrhythmias has been widely demonstrated, QT and QTd have been reported to be increased in obesity and appear influenced by autonomic tone (3,4,34). These two parameters give different kinds of information about repolarization. The QT interval reflects the total duration of ventricular myocardial depolarization and repolarization. Prolongation of QT on a standard 12-lead electrocardiogramm is caused by lengthening of the action potential of ventricular myocytes. QT dispersion, instead, measures indirectly the heterogeneity of repolarization, caused by regional variations in the duration of the action potentials.

Both QTc and QTd are correlated in a significant manner with cardiovascular and all cause mortality (35). A QTc interval lengthening (5,6) and a QTd increase (7–10) are associated with a greater risk of ventricular tachyarrhythmias and sudden death in many pathologies.

A prolonged QTc interval has been reported in obese subjects with a positive correlation between BMI and QTc (3). Other researches found in obese compared with nonobese women a nonpathological lengthening of QTc and QTd, which showed a positively correlation with circulating plasma levels of free fatty acids (36).

It seems that there is a relationship between free fatty acids, cardiac sympathetic nervous activity and repolarization abnormalities; the autonomic dysfunction with a sympatho-vagal imbalance is a potential mechanism underlying QT and QTd prolongation in obese subjects (34).

Our study shows the presence, in overweight and obese subjects, also without associated comorbid conditions, of a longer QTc compared to that of normal weight subjects, over 440 ms in the 10% of the sample. On the contrary QTd was, in both groups, always equal or lower than 60 ms with a single obese subject showing QTd equal to 70 ms; the values showed were not different from those obtained in healthy subjects from previous researches (in particular, in 8,455 healthy subjects from 54 different studies, Malik observed QTd values ranging between 10.5 ± 10.0 ms and 71 ± 7 ms) (37).

Our data are, moreover, compatible with a previous study that highlighted no significant difference as regards QTd between normal weight subjects and obese/overweight subjects, not affected by comorbid conditions such as ischemic cardiomyopathy, hypertension and diabetes mellitus (38).

Just like QTd also QT apex dispersion did not show significant changes between the two groups. However, it must be noted how QTc, QTd, and QTd-apex present a tendency toward increase, comparing normal weight to overweight subjects, and overweight to obese subjects; though these differences reach statistical significance, only for QTc, the only parameter that, unlike the others, may exceed the normal cut-off.

Beyond QTd (18), other electrocardiographic variables involving the terminal part of QT are able to reflect dispersion of ventricular repolarization, as assessed by monophasic action potentials recordings; in particular, the QT peak/QTend ratio from a single lead (V3) and the distance between the peak and the end of the T wave (Tpe interval) reflect dispersion of ventricular repolarization, with equally good correlation than QTd (39). Maximum Tpe interval was shown to be greater in inducible patients compared to noninducible patients during electrophysiological study (40) and, together with its dispersion, it was associated to an increased incidence of life-threatening ventricular arrhythmias in patients affected by Brugada syndrome (12). Another electrocardiographic parameter, the Tpe/QT ratio, was recently indicated as a new marker of arrhythmogenesis (15), both in patients affected by hypertrophic cardiomyopathy (13) and in patients affected by long QT syndrome (14). In particular, in LQTS, a Tpe/QT ratio in V5 >0.28 was demonstrated to be an efficacious predictor of ventricular arrhythmias (torsades de pointes).

In our study, no one of these new parameters showed significant differences between the two groups; Tpe-d did not show the tendency to increase with increasing BMI and Tpe_{max} and Tpe/QT ratio were not dissimilar between normal weight and overweight/obese subjects, in particular mean Tpe/QT ratio coincided in the two groups. A clear influence of BMI on QT parameters was demonstrated only for QT and QTc.

After all the only parameters that changed in a statistically meaningful way between normal weight and overweight/obese subjects were represented by QT and QTc; therefore, it could be assumed that in uncomplicated obesity some subjects begin to show a delayed ventricular repolarization demonstrated by a QTc increase in absence, however, of interregional variations of repolarization as the normal parameters of global dispersion (QTd and QTd-apex) and transmural dispersion (Tpe, Tpe-d, and Tpe/QT ratio) show.

If obesity is truly uncomplicated (lack of comorbid conditions such as coronary artery disease, hypertension, diabetes), it is not associated with any dispersion of repolarization but its prolongation may be present.

In a small percentage of overweight/obese subjects (10% in our study), even in absence of underlying heart diseases, cardiomyocytes show some abnormalities in their electrophysiological properties, characterized by a lengthening of action potential.

The exact mechanism involved in QT prolongation is unknown, but it seems to influence ion channel properties causing only an increase of APs duration but not an amplification of spatial dispersion of repolarization.

Conclusions

Uncomplicated obesity and overweight are not associated with a statistically significant increase in QTd, QTd-apex, Tpe, Tpe-d, and Tpe/QT ratio; a lengthening over physiological limit for QTc may occur. Also if there is a tendency toward increase of QTd and QTd-apex in obese subjects, this does not achieve statistical significance and does not exceed the pathological threshold. Further studies are necessary to demonstrate whether this tendency towards an increase for QTd and QTd-apex, already present in uncomplicated obesity, could be even more emphasized in complicated obesity extending into the pathological range and if, in this case, increases in Tpe, Tpe-d, and Tpe/QT ratio may be also present.

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DISCLOSURE

The authors declared no conflict of interest.

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