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The Effect of Lower and Higher Calorie Meal on the Parameters of Ventricular Repolarization in Healthy Subjects

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Keywords: Meal consumption; Ventricular repolarisation; High-calorie intake

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Abstract

BACKGROUND: Cardiovascular modulation following meal consumption has been known. Little and conflicting data is present regarding electrocardiographic QT and QTc intervals after a meal, and status of ventricular repolarization following meal is not known comprehensively.

AIM: To inquire the electrocardiographic status of ventricular repolarisation thoroughly after lower and higher calorie meal consumption in a comparative manner.

METHODS: A group of 61 healthy individuals were studied before and after lunch. They were divided into two groups according to the calorie consumed (higher calorie and lower calorie; median 1580 and 900 kcals, respectively). Calorie consumed was estimated using dietary guidelines. Data was collected from 12-lead ECG both in a fasted state and 2nd postprandial hour for each participant. Parameters of ventricular repolarization, namely, JTp, Tp-e, QT, QTc intervals and their ratios, as well as RR intervals, were compared between fasted and postprandial states for every participant.

RESULTS: Tp-e and QTc intervals, and Tp-e/QTc ratio do not significantly change after both higher- and lower-calorie meals. JTp and QT intervals significantly shorten in both groups, regardless of the calorie consumed. While JTp shows a positive correlation with RR interval both before and after a meal in lower calorie intake group, no correlation was found with RR interval after a meal in higher calorie group. Logistic regression analysis revealed that higher calorie intake during a meal is a predictor for greater shortening in JTp and QT, compared to lower calorie meal.

CONCLUSION: Our study may guide future studies on ventricular repolarisation, particularly those conducted on various disease conditions or drug effect of cardiac electrophysiology.

Introduction

A vast realm of significant bodily modulations regarding cardiovascular and neuroendocrine systems has long been recognised following meal consumption. The mesenteric arterial system requires a boost in its blood flow for digestive and absorptive purposes during the postprandial period compared to the previous fasting state [1]. To attain this, accordingly, the heart responds to a meal by increasing its output, rate and stroke volume [2], [3], despite confounded findings regarding the latter [4]. Moreover, both glucose and insulin levels escalate, together with alterations in autonomic nervous and other relevant endocrine systems [4], [5], [6].

Electrocardiographically, ventricular repolarisation is represented by QT interval, and its prolongation was suggested to be associated with higher risk of ventricular arrhythmias in a variety of clinical settings [7], [8], [9]. Moreover, the QT interval can be divided further into three parts as such: QRS width; JTp interval, which stands for the time period between the end of QRS and the peak of the T wave; and T peak to T end interval (Tp-e interval), which stands for the time period in between the peak and the end of the T wave. In general, JTp comprises the major part of a QT interval and can roughly be

regarded as an early phase of ventricular repolarization, whereas Tp-e interval may represent the late phase of ventricular repolarization. Repolarization in the cardiac ventricles commence very soon after the onset of ventricular activation. Hence QT interval provides insight about the overall repolarization status of the ventricles. Tp-e interval, on the other hand, appears as an index for transmural dispersion of ventricular repolarisation [10] and is associated with varying durations of the action potential in endocardium, myocardium and epicardium. Just as is the case in QT interval, evidence suggests that prolongation of Tp-e interval has been associated with the ventricular arrhythmogenic potential of different degrees [11], [12], [13].

More recently, effect of food intake on some electrocardiographic parameters appeals more and more to the investigators, since accumulating evidence points out that meal consumption of varying composition and calories are likely to have effect on QT and QTc intervals [14], [15], [16], [17], [18]. This especially holds true when it comes to the development of new drugs and drug regiments in thorough QT/QTc studies, where food intake is a likely challenge in anticipation of drug-related proarrhythmic potential [17], [19]. Previous studies have yielded conflicting results on the effect of food intake on QT and QTc. There appears to be no data regarding changes in such different parts of QT interval as JTp, Tp-e, JT and their ratios with each other, namely, Tp-e/QT, Tp-e/QTc, Tp-e/JTp, Tp-e/JT and JTp/JT following meal consumption.

In the present study, we aimed to evaluate these ventricular repolarisation parameters comparatively between lower- and higher-calorie meal consumption in healthy individuals.

Methods

Study design and participants

Our longitudinal study included a total of 61 (36 men and 25 women; aged between 18 and 50) consecutive and healthy individuals who admitted to our cardiology out-patient clinic for a routine cardiologic check-up between May 2018 and July 2018. All the participants were in an overnight fasted state for their anticipation of a probable routine blood chemistry screen. Thorough physical examinations, and electrocardiographic (ECG) and transthoracic echocardiographic scans were implemented in each of the participants. Further, each was subjected to a comprehensive interrogation for any past and/or present medical disease, and smoking and alcohol habits. Body-mass index (BMI) was calculated as weight in kilograms divided by the square of the height in meters. Blood samples were obtained through venipuncture for routine laboratory analysis. Then, the participating individuals were asked to eat their lunch as ordinarily as in their previous daily lives, but to abstain from caffeine-containing and alcoholic beverages as well as smoking. Two hours after the onset of their lunch [17], they were asked to re-admit to the out-patient polyclinic to have their postprandial ECG recordings taken. Subsequently, the participants were interrogated about what they had eaten at lunch, recording the composition and the volume of the food consumed, and then we calculated the estimated total calorie intake. Among the exclusion criteria were renal, gastrointestinal or hepatic dysfunction, alcoholism, any known History of cardiac disease, malignancy, diabetes mellitus, and medication use of any kind, endocrine pathology, hypertension, supplementation therapy of any kind, and having a complex meal that prevents appropriate estimation of energy content of the relevant meal. Informed consent was obtained from every participant, and the local committee approved our study (Kirsehir Ahi Evran University Ethical Committee, No: 2018-35/135, Date: 27.04.2018).

Calculation of basal metabolic rate and estimated caloric intake

The daily required total calorie intake to maintain the study participants' basal metabolic rate was calculated specifically by the use of Harris and Benedict equation [20], and the preliminary result obtained by the equation was multiplied by the physical activity level to obtain the ultimate result. As for the physical activity level, it was estimated using the Baecke Questionnaire [21]. Moreover, the estimation of the energy content of meals by the composition and the volume that the participants consumed at lunch were done using ''Turkey Dietary Guidelines'' [22].

ECG measurements

In a supine position following at least 10 minutes rest, 12-lead ECGs were obtained from each of the participants 1-2 hour before the lunch and 2 hours after the onset of lunch using a standard ECG system (Nihon Kohden, Tokyo, Japan) at a paper speed of 50 mm/s. All of the ECG papers were scanned and transferred to the digital media, and the digital records were analysed under x300% magnification in a personal computer. RR interval, QT interval, JT interval and JTp interval and Tp-e interval, together with such ratios as Tp-e/QT, Tp-e/QTc, Tp-e/JT, Tp-e/JTp and JTp/JT were calculated using the precordial lead V3 [23], [24], [25]. There are two commonly used methods of measuring Tp-e interval: tangent method and tail method [26], [27]. We used in our study the tangent method, which refers to the time interval between the peak of the T wave and the point

where the tangent of the steepest down-slope of the T wave intersects with the isoelectric line (T wave offset) [26]. Respective QT, JT and JTp intervals were measured as follows: from the Q onset to T wave offset; from the S wave offset to T wave offset; and, from the S wave offset to the peak of T wave. Although variable methods were applied as methods of correction for heart rate in different studies [14], [15], [16], [17], the decision of heart rate correction for QT and JTp intervals in the present study was made to implement the Bazett's formula [28].

On the other hand, no incentive was felt in favour of heart rate correction for Tp-e interval, as Tp-e was suggested to be heart-rate independent [29]. Moreover, three consecutive beats were averaged to obtain the ultimate measurement for each parameter. All the ECG parameters were assessed by a single experienced cardiologist blinded to the study design to avoid inter-observer variability, especially in the detection of S wave offset [30].

Statistical analysis

Statistical analysis of the study data was performed using SPSS Version 22.0 (SPSS Inc., Chicago, IL, USA). Numbers, percentage, mean ± standard deviation, median, minimum (min), maximum (max) and 25-75 percentiles were used for the descriptive statistics. Independent sample t-test and Mann-Whitney U test were used for the comparison of the demographic characteristics between the two groups. Wilcoxon Signed Ranks test was utilised to compare differences of ECG parameters of interest between fasted and postprandial states in lower and higher calorie-intake groups. Moreover, Spearman's rank correlation analysis was used to determine the correlation of the ECG parameters with RR interval, and multivariate linear logistic regression analysis (variables at a $p < 0.10$ significance level in the univariate analysis) was applied to evaluate the independent association of calorie intake (high-low calorie) with the ECG parameters of interest. A p-value < 0.05 was accepted to be statistically significant.

Results

Demographic Characteristics

Baseline demographic characteristics of the patients are presented in Table 1. Higher calorie intake group was composed of 35 participants [15 female (42.8%)], whereas lower calorie intake group was composed of 26 participants [10 female (38.4%)]. Both groups were similar regarding age, gender and blood analysis. BMI (kg/m^2), however, was only slightly greater in the lower calorie intake group compared to the higher calorie intake group, which was statistically significant [24.5 (23.8-25.2) vs 23.8 (23-24.7), respectively; $p = 0.045$]. As for the echocardiography, both systolic and diastolic functions, as well as cardiac dimensions were normal in both groups.

Calculated Basal metabolic rate and Estimated Caloric Intake

One-third of the caloric intake required for the maintenance of the basal metabolic rate, correspondingly to lunch, by Harris and Benedict formula was similar in both groups [860 Kcal (830-880) vs 850 Kcal (820-860), respectively; $p = 0.415$]. Moreover, median calorie consumption at lunch was estimated to be 900 Kcal (850-930) for the lower calorie intake group, while it was 1580 Kcal (1500-1670) in the higher calorie intake group ($p < 0.001$).

Table 1: Demographic characteristics of the study groups

	Lower Calorie Intake Group (median, IQR 25-75) (n = 26)	Higher Calorie Intake Group (median, IQR 25-75) (n = 35)	p
Age (years)	35.5 (22-43)	34 (25-41)	0.502
Gender, female, n(%)	10 (38.4%)	15 (42.8%)	0.730
BMI, kg/m2	24.5 (23.8-25.2)	23.8 (23-24.7)	0.045
Total Cholesterol (mg/dL)	178 (165-189)	187 (170-203)	0.113
Hemoglobin (g/dL)	14 (13.5-14.5)	14.1 (13.2-14.6)	0.832
ALT (U/L)	18 (15-21)	18 (16-20)	0.861
Glucose (mg/dL)	84.5 (81-88)	89 (82-92)	0.111
WBC (10^3/uL)	7.8 (6.4-8.6)	7.9 (6.5-9)	0.366
C-reactive protein (mg/dL)	0.09 (0.05-0.4)	0.19 (0.09-0.29)	0.290
E/E'	5.7 (5.4-6.0)	5.8 (5.3-6,2)	0.563
LVEF (%)	64.5 (60-67)	64 (61-68)	0.583
LVEDD (mm)	45 (43-48)	45 (44-49)	0.596
Left atrial diameter (mm)	34 (32-36)	34 (33-37)	0.513
IVS thickness (mm)	9.4 (9.1-10)	9.3 (9.1-9.8)	0.918
PWT (mm)	8.9 (8.7-9.2)	9.0 (8.7-9.3)	0.558
Calorie Consumption (Kcal)	900 (850-930)	1580 (1500-1670)	< 0.001
(Calorie required for BMR)* (Kcal)	860 (830-880)	850 (820-860)	0.415

Values were given in median (25-75 IQR). E/E' = the ratio of transmitral early filling velocity to tissue Doppler early diastolic lateral mitral annular velocity. ALT = alanine aminotransferase; LVEDD = left ventricle end-diastolic diameter; LVEF = left ventricle ejection fraction; IVS = interventricular septum. PWT = posterior wall thickness; BMR = basal metabolic rate. BMI = body-mass index; WBC = white blood cell count; * Calorie corresponding to one-third of total daily basal metabolic rate, expected to consume at lunch.

Electrocardiographic Repolarization Parameters

Comparison of the ECG parameters of ventricular repolarization is presented in Table 2. Respective JTp, JTpc and QT intervals significantly decreased in the postprandial 2nd hour compared to the fasting state in both of lower and higher calorie intake groups [194 ms (177.2-209.9) vs 179.9 ms (161.6-189.5), $p < 0.001$; and, 201 ms (190.2-217.9) vs 179.7 ms (157.2-193.2], $p < 0.001$], [203 ms (196-211.1) vs 198.8 ms (180.1-208.6), $p = 0.003$; and, 216.6 ms (206.9-232.9) vs 205.7 ms (189.2-226), $p = 0.004$], [361.2 ms (345.52-378.57) vs 351.8 ms (334.72-369.30), $p = 0.001$; and, 388 ms (364-404) vs 364 ms (340-378), $p < 0.001$]. Respective Tp-e

interval, QTc interval and Tp-e/QTc ratio, however, did not change postprandially compared to the fasting state in both of the lower and higher calorie intake groups [76.05 ms (66.60-79.92) vs 75.25 ms (65.50-82.75), p = 0.440; and, 71.9 ms (63.6-79.2) vs 73.5 ms (67.4-80), p = 0.051], [391.5 ms (373.95-403.50) vs 391.5 ms (378.75-411.65), p = 0.139; and, 412 ms (391-428) vs 419 ms (399-430), p = 0.148], [0.195 (0.178-0.205) vs 0.193 (0.170-0.209), p = 0.77; and, 0.172 (0.155-0.190) vs 0.178 (0.158-0.192), p = 0.987]. Furthermore, respective Tp-e/QT, Tp-e/JTp and Tp-e/JT ratios increased significantly, whereas JTp/JT ratio decreases significantly in the postprandial 2nd hour both in lower calorie and higher calorie intake groups [0.198 (0.183-0.226) vs 0.214 (0.188-0.228), p = 0.004; and, 0.183 (0.163-0.202) vs 0.197 (0.182-0.225), p < 0.001], [0.376 (0.338-0.451) vs 0.404 (0.372-0.479), p < 0.001; and, 0.345 (0.296-0.397) vs 0.400 (0.347-0.469), p < 0.001], [0.273 (0.252-0.311) vs 0.288 (0.271-0.324), p < 0.001; and, 0.257 (0.228-0.284) vs 0.286 (0.258-0.319), p < 0.001], [0.726 (0.688-0.747) vs 0.711 (0.675-0.728), p < 0.001; and, 0.742 (0.715-0.771) vs 0.713 (0.680-0.741), p < 0.001]. Lastly, RR interval decreased significantly due to increase in hearth rate in the postprandial period in both of the lower and higher calorie intake groups [882 ms (787.5-967.1) vs 779.2 ms (703.2-892.5), p < 0.001; and, 880 ms (778-974) vs 742 ms (682-808), p < 0.001].

Table 2: ECG parameters of ventricular repolarisation compared between the two groups

		Fasting ECG Parameters	Postprandial ECG parameters	P
Lower Calorie Intake Group	Tp-e (ms)	76.05 (66.60-79.92)	75.25 (65.50-82.75)	0.440
	JTp (ms)	194 (177.27-209.90)	179.95 (161.62-189.55)	< 0.001
	JTPpc (ms)	203 (196-211.15)	198.85 (180.10-208.67)	0.003
	QT (ms)	361.2 (345.52-378.57)	351.8 (334.72-369.30)	0.001
	QTc (ms)	391.5 (373.95-403.50)	391.5 (378.75-411.65)	0.139
	Tp-e/QT	0.198 (0.183-0.226)	0.214 (0.188-0.228)	0.004
	Tp-e/QTc	0.195 (0.178-0.205)	0.193 (0.170-0.209)	0.77
	Tp-e/JTp	0.376 (0.338-0.451)	0.404 (0.372-0.479)	< 0.001
	Tp-e/JT	0.273 (0.252-0.311)	0.288 (0.271-0.324)	< 0.001
	JTp/JT	0.726 (0.688-0.747)	0.711 (0.675-0.728)	< 0.001
	RR interval (ms)	882 (787.5-967.17)	779.2 (703.25-892.50)	< 0.001
Higher Calorie Intake Group	Tp-e (ms)	71.9 (63.6-79.2)	73.5 (67.4-80)	0.051
	JTp (ms)	201.1 (190.2-217.9)	179.7 (157.2-193.2)	< 0.001
	JTPpc (ms)	216.6 (206.9-232.9)	205.7 (189.2-226)	0.004
	QT (ms)	388 (364-404)	364 (340-378)	< 0.001
	QTc (ms)	412 (391-428)	419 (399-430)	0.148
	Tp-e/QT	0.183 (0.163-0.202)	0.197 (0.182-0.225)	< 0.001
	Tp-e/QTc	0.172 (0.155-0.190)	0.178 (0.158-0.192)	0.987
	Tp-e/JTp	0.345 (0.296-0.397)	0.400 (0.347-0.469)	< 0.001
	Tp-e/JT	0.257 (0.228-0.284)	0.286 (0.258-0.319)	< 0.001
	JTp/JT	0.742 (0.715-0.771)	0.713 (0.680-0.741)	< 0.001
	RR interval (ms)	880 (778-974)	742 (682-808)	< 0.001

Values were given as median (IQR 25-75).

Correlations and Predictors

The correlation of RR interval with some ECG parameters of ventricular repolarisation is shown in Table 3. The JTp interval both in fasted and postprandial states in lower calorie intake group exhibits a significant correlation with respective RR intervals [(r = 0.708, p < 0.001; fasted state) and (r = 0.576, p = 0.002; postprandially)]. On the other hand, only fasted state JTp interval shows a statistically significant correlation with fasted state RR interval in the higher calorie intake group, while no statistically significant correlation was evident between the postprandial JTp and RR intervals in the same group [(r = 0.440, p = 0.008; fasted state) and (r = 0.213, p = 0.219; postprandially)].

Table 3: Spearman's Rank Correlation analysis comparing fasted-state and postprandial JTp and QT intervals with relevant RR intervals

		Fasting State RR interval (ms)		Postprandial RR interval (ms)	
		p	r	p	r
Lower Calorie Intake Group	Postprandial JTp (ms)			0.002	0.576
	Fasting JTp (ms)	< 0.001	0.708		
	Postprandial QT (ms)			< 0.001	0.644
	Fasting QT (ms)	0.001	0,605		
Higher Calorie Intake Group	Postprandial JTp (ms)			0.219	0.213
	Fasting JTp (ms)	0.008	0.440		
	Postprandial QT (ms)			0.001	0.534
	Fasting QT (ms)	0.002	0.512		

As for the QT interval, it shows a statistically significant correlation with RR interval in respective fasted and postprandial states in both of the groups [(r = 0.605, p < 0.001; fasted state) and (r = 0.644, p = < 0.001; postprandially); the lower calorie intake group], [(r = 0.512, p = 0.002; fasted state) and (r = 0.534, p = 0.001; postprandially); the higher calorie intake group].

Further in logistic regression analysis, greater increase in caloric intake at lunch was found to be predictor of greater decrease in JTp and QT intervals (β = 1.045, p = 0.034; β = 1.036, p = 0.018; respectively). Such an association, however, did not show up with the other ECG parameters of ventricular repolarization (Table 4).

Table 4: Multivariate linear logistic regression analysis

Variables	B	Exp (B)	p	95% CI	
				Lower	Upper
JTp (ms)	0.44	1.045	0.034	1.003	1.088
QT (ms)	0.36	1.036	0.018	1.006	1.067
Tp-e (ms)	-0.025	0.976	0.647	0.878	1.084
QTc (ms)	-0.001	0.999	0.942	0.947	1.025
Tp-e/QTc	-3.193	0.041	0.852	0	146

Discussion

The very first finding of our study indicated that Tp-e interval, QTc and Tp-e/QTc did not change significantly in the postprandial 2nd hour compared to the fasted state, irrespective of the total calorie consumed during a meal. On the other hand, QT, JTp and RR intervals decreased significantly in the postprandial 2nd hour compared to the fasted state in both of the higher and lower calorie intake groups, which was compatible with the findings of previous studies [15], [17], [19].

RR interval has long been known to decrease

following meal consumption since postprandial state pertains to hyperemia in the mesenteric vasculature to meet digestive requirements [1], [2], [31]. As stated in the introduction section, QT interval can roughly be divided into two as the early phase of the ventricular repolarisation represented by JTp interval and late phase of the ventricular repolarisation represented by Tp-e interval. Although QT interval is known to be heart rate dependent, the same does not hold true for the Tp-e interval. Hnatkova et al., [29] reported in their study that only a very small portion of Tp-e interval ($T_{95\%}$-Te) was affected by heart rate changes, hence that omission of heart rate correction of Tp-e does not incur any inaccuracy in the absence of dramatic heart rate changes. Accordingly, most of the heart rate dependency of QT interval is associated with the heart rate dependency of JTp interval [29]. According to the Spearman's correlation analysis in the current study, JTp interval exhibited a positive and significant correlation with RR interval both in fasted and postprandial states in lower calorie intake group. Thereby, shortening of JTp postprandially in lower calorie intake group cannot be ascribed to anything other than a normal response to the postprandial heart rate increase. Note, however, that postprandial JTp does not show any correlation with postprandial RR interval in higher calorie intake group, despite the persistence of positive correlation between fasted-state JTp and fasted-state RR intervals. Aside from heart rate increase, it may be reasonable to assume that this condition in part is due to more pronounced neuroendocrine and cardiovascular activation incited by higher calorie intake than metabolic requirements which particularly affects the early phase of the ventricular repolarisation in healthy subjects [31], [32]. Contrary to the more specific early phase of ventricular repolarisation, global ventricular repolarisation represented by QT interval still exhibits positive correlation with RR interval during fasted and postprandial states in both of the groups.

Previous studies yielded conflicting results concerning the effect of food intake on QT and QTc. Cirincione et al., [19] reported in their study that QT and QTcF intervals are subjected to the most dramatic decrease in 2-3 hours after a meal. The heart rate increase was also the most prominent 2 hours after the meal. Although they utilized Fridericia'sformula [33] for heart rate correction, we preferred to use the Bazett's formula due to lack of precise consensus regarding the most eligible formula to apply in clinical settings and to the fact that most of the currently-available formulas for heart rate correction yield almost equivalent results in resting heart rates within the range of 60-90 beats/min [34]. Taubel et al., [17] showed in their study that the maximum decrease in QT interval was 2 hours after the onset of a standardised breakfast composed of 617 kcal. However, they applied different correction formulas on QT for heart rate which yielded as follows: QTcF interval with maximum shortening at 2.5 hours; QTcIP interval with maximum shortening at 3.5 hours; and, QTcB with an initial slight increase, then returning to the baseline at 2 hours after the onset of breakfast. Nagy et al., [15] conducted a study inquiring RR interval, QT and QTcB at postprandial 1 hour and reported a decrease in postprandial QT at 1 hour. However, QTcB, they suggested, increased significantly 15 minutes after the meal, then gradually decreased to reach such a level that was still significantly greater than the baseline value at 1 hour. Sciot et al., [14] reported a significant increase in heart rate, a matched decrease in QT interval, but no difference either in QTcF or in QTcI intervals. The absence of change in QTcB in our study seems compatible with the findings of Taubel et al., [17] Multivariate linear logistic regression analysis revealed that higher calorie consumption during meal acts as a predictor for greater JTp and QT interval shortening, but not for the Tp-e and QTc intervals and Tp-e/QTc ratio.

The main strength of our study is that, apart from previous studies, we evaluated such recently more appealing parameters of ventricular repolarisation as Tp-e, JTp, QT, QTc intervals, and Tp-e/QTc ratio in a comparative manner after meal consumption between higher and lower calorie intake groups of healthy subjects. Moreover, to our knowledge, this study is the first in this regard.

Our study is likely to provide a new framework for further studies about ventricular repolarization, particularly those conducted on various disease conditions or drug effect of cardiac electrophysiology.

This study should be evaluated in light of some limitations. First, we did not provide a fixed lower and higher calorie meal for the study participants and relied solely on the information provided by them. Secondly, our study population is relatively small, and further studies with greater participation may reveal different results. Thirdly, we could not estimate the macronutrient portion (proteins, glucose, lipids) of the meals consumed, which may have exerted effects to some extent on the repolarisation parameters of interest.

In conclusion, Tp-e interval, QTc interval and Tp-e/QTc ratio do not change significantly at a 2nd postprandial hour, regardless of the amount of the calorie consumed. However, QT interval shortens, due almost solely to JTp interval shortening. Further, JTp interval shortening following higher calorie meal may not only be associated with increased heart rate but also higher calorie consumption during the meal, possibly through unproportional escalation in neuroendocrine and cardiovascular response. Lastly, higher calorie consumption at meal represents a predictor for greater JTp and QT shortening. We believe that the current study may provide a new insight for further studies about ventricular repolarization parameters, particularly those conducted on various disease conditions or drug effect on cardiac electrophysiology. Also, our study is

very likely to increase the awareness about the effect of the timing and the calorie of the meals consumed on the assessment of the parameters of ventricular repolarisation among the researchers in their future studies regarding noninvasive electrophysiology. However, further large-scale studies are warranted to justify our study results.

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