Table-1

Age (n)	35-44 (1309)	45-54 (988)	55-64 (887)	65-74 (572)	≥75 (193)	Total	
ECG pattern	n (female: n) % in age group					n	%(overall)
Type 2	6(2) 0,5%	4(0) 0,4%	5(0) 0,6%	4(2) 0,7%	2(0) 1%	21(4)	0,5 %
Type 3	14(3) 1,1%	14(1) 1.4%	14(2) 1,6%	4(2) 0,3%	3(0) 1,6%	47(8)	1,2 %
BTEP	20(5) 1,5%	19(2) 1,9%	19(2) 2,1%	8(4) 1,0%	5(0) 2,6%	71(13)	1,7%
ER inferior	40(15) 3,1%	30(10) 3,0%	23(7) 2,6%	17(8) 3,0%	2(0) 1,0%	112(40)	2,8%
ER lateral	12(9) 0,9%	8(6) 0,8%	7(2) 0,8%	3(3) 0,5%	1(1) 0,5%	31(21)	0,8%
ER inferolateral	15(4) 1,1%	1(1) 0,1%	4(3) 0,5%	1(1) 0,2%	0 0%	21(9)	0,5%
ERV	67(28) 5,1%	39(17) 3,9%	34(12) 3,8%	21(12) 3,7%	3(1) 1,6%	164(70)	4,1%

ERV: Early Repolarization Variant BTEP: Brugada Type ECG Pattern n: Number of subjects

OP-155

Usefulness of Maximal Exercise-Corrected QT as a Predictor of Coronary Artery Disease: A Comparison of Simpler Heart Rate Corrections

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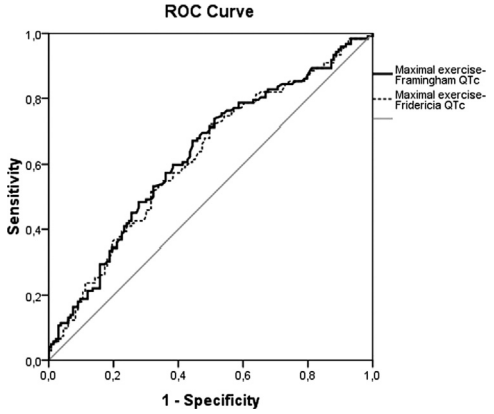
Background: The relationship between QT prolongation and myocardial ischemia is known. Due to the limited value of ST depression, we aimed to evaluate, by using four simpler heart rate corrections (Bazett, Framingham, Fridericia and Hodges), the value of maximal exercise-QTc prolongation in the diagnosis of coronary artery disease's (CAD) presence and severity.
Methods: We enrolled 234 subjects (mean age 57.3±9 years, 143 men) who underwent exercise testing and coronary angiography due to suspicion of CAD in the study. The coronary artery was considered diseased if stenosis in a major epicardial coronary artery was ≥50 % diameter. Evaluating CAD severity with Gensini scoring, the CAD group (n=122) and controls with non-CAD were compared in terms of corrected QT duration and at maximal exercise.The corrected QT was calculated by using the following formulae; Bazett's formula QTc= QT/RR1/2, Framingham QTc= QT+ 0.154(1-RR), Fridericia formula QTc= QT/RR1/3 and Hodges QTc= QT + 0.175 (HR-60), respectively.
Results: Age, gender, hypertension, dyslipidemia, smoking, exercise duration, resting and peak heart rate were similar between the two groups (All p > 0.05). The CAD group have higher raw QT values than the controls (268 [169- 438] vs. 240 [168-348], p<0.001).While Bazett and Hodges formulae were not appropriate at maximal heart rates for diagnosing of CAD presence, Framingham QTc of ≥350 ms and Fridericia QTc of ≥340 ms were seen to be useful. Maximal exercise-QTc Bazett (r=0.163, p=0.01), Framingham (r=0.239, p=0.001) and Fridericia (r=0.206, p=0.001) equations were weakly positively correlated with Gensini scoring. There was no correlation between Hodges QTc at maxima exercise and Gensini scoring (p value>0.05).
Conclusion: Measuring with Framingham and Fridericia formulae, are better than Bazett and Hodges at peak heart rates, patients with CAD have longer QTc interval at peak heart rates during exercise; this finding provides further evidence supporting routine incorporation of QTc at peak heart rates into exercise test interpretation.



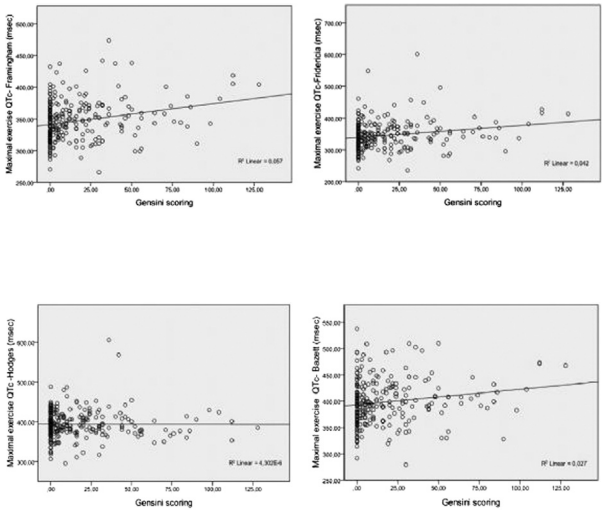


Table 1. Baseline characteristics, biochemical results and Gensini scoring for the both groups.

Variables	CAD group n=122	Control group n=112	P value*
Age, years	55.5± 9.1	54.9± 8.6	0.776
Male, n (%)	74(60.6)	69 (61.6)	0.144
Female, n (%)	48(39.4)	43 (38.4)	0.156
BMI, kg/m2	29(23-41)	29(19-45)	0.235
DM, n (%)	28 (22.9)	24(21.4)	0.441
Smoking, n (%)	25 (20.4)	21 (18.7)	0.108
HT, n (%)	38 (31.1)	34 (30.3)	0.138
LDL, mg /dl	137±41	135±41	0.477
HDL, mg/dl	42(26-70)	47(25-87)	<0.001
TG, mg/dl	170 (64-440)	124(58-330)	0.008
Uric acid, mg/dl	5.2 (1.1-8.3)	4.7(2.4-9.4)	0.009
Creatinine, mg /dl	1.0 (0.7-1.4)	0.9(0.3-1.3)	0.568
Hemoglobin, mg /dl	13.2±1.0	13.4 ±0.9	0.678
METs, unit	8.7±2.3	8.8±2.4	0.569
ST depression ≥1 mm, n (%)	95 (77.8)	38 (33.9)	<0.001
Gensini scoring	25(10-128)	2 (0- 4)	<0.001

Data are presented as mean±SD, median (minimum-maximum) values and number/ percentage, *Chi-square, Mann Whitney U test and unpaired Student's t-tests, BMI- body mass index, CAD - coronary artery diseases, DM - diabetes mellitus, HT-hypertension, LDL - low - density lipoprotein, METs– peak metabolic equivalents of exercise test,TG – triglyceride

Table 2. Resting and maximal exercise testing findings for the both group.

Variables	CAD group n=122	Control group n=112	P value*
Resting heart rate, bpm	81(56-101)	85(50-102)	0.118
Resting QT, ms	339 ± 34	332 ± 36	0.101
Resting QTc, ms			
Bazett	390 (311-587)	393 (295-488)	0.865
Framingham	378 ± 25 a1	374 ± 27 b1	0.373
Fridericia	376(302- 491) a2	374 (279-458) b2	0.409
Hodges	394 (311-517) a3	389 (297-490) b3	0.995
Resting systolic BP, mmHg	121 ± 15	118± 17	0.158
Resting diastolic BP, mmHg	81± 6	78 ± 8	0.244
Resting to peak HR time, min	6.9±2.6	7.1 ±2.5	0.376
Peak HR, bpm	149 ±19	150 ± 20	0.861
Max. exercise QT, ms	268 (169- 438)	240 (168-348)	<0.001
Max. exercise QTc, ms			
Bazett	405 (290-556)	391 (291-537)	0.015
Framingham	360 ± 34 a1	340 ± 29 b1	<0.001
Fridericia	350 (235-515) a2	331 (242- 461) b2	0.001
Hodges	411 (331-514) a3	409 (325-497) b3	0.601
Peak systolic BP, mmHg	190 ± 27	195 ± 26	0.123
Peak diastolic BP, mmHg	95±12	98 ± 11	0.156

bpm- beat per minute, BP- blood pressure, HR- heart rate, min-minute, ms- millisecond, * Chi square, Mann Whitney U test and unpaired Student's t-tests, paired t test and Wilcoxon rank test.a1,a2,a3 between resting and maximal exercise in the CAD group($p < 0.001$), b1,b2,b3 between resting and maximal exercise in the control group ($p < 0.001$).

ORALS

OP-156

Relationship between Scar Size and Characteristics by ce-CMR and Tpeak-Tend Interval in Post-MI Patients with Relatively Preserved LV Functions and Nonsustained Ventricular Tachycardia

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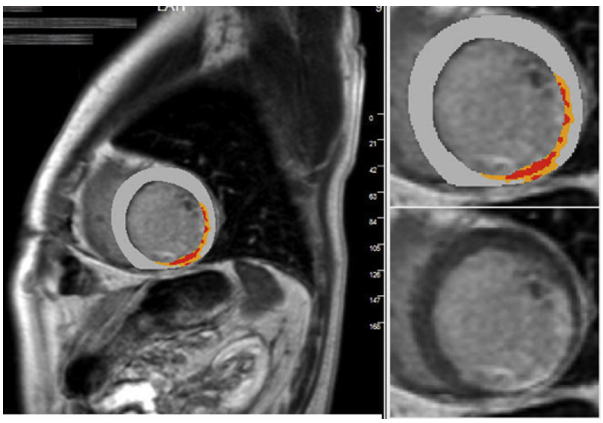
Introduction: The Tpeak-Tend (Tpe) interval is an electrocardiographic index of transmural repolarization dispersion and has been reported to predict life- threatening arrhythmias in the long QT syndrome, Brugada Syndrome, hypertrophic cardiomyopathy and systolic dysfunction. Infarct tissue size and heterogeneity characterized by cardiac magnetic resonance (CMR) has been shown to be associated with arrhythmogenic substrates and sudden cardiac death. Although Tpe intervalis not considered to reflect structural heart disease, Tpe interval was found to be correlated with extent of non-transmural scar in post-MI patients. However the relationship between peri-infarction zone and scar core percent has never been studied. In this study, we aimed to study the relation between Tpe interval and scar size and characteristics assessed with CMR in post-MI patients.
Methods: This study was enrolled 28 post-MI patients with non-sustained ventricular tachycardia. Tpe interval was defined as the peak or nadir of T wave to the end of T wave and was measured in each precordial lead by a single blinded observer. All patients underwent both cine and contrast enhanced CMR imaging. Left ventricular ejection fraction (LVEF), scar core, peri-infarct zone and total scar masses were assessed and these values to LV mass ratios were obtained (Figure). The correlation analysis was performed to determine the relationship between Tpe interval and infarct parameters.
Results: All of the patients had evidence of scar on late enhanced images. Mean left ventricular ejection fraction was 44.3±4.2%. Mean scar core (>3SD SI of remote myocardium) was 3.34±0.86%, mean peri-infarct zone (2-3 SD SI of remote myocardium) 22.48±8.51% and mean total scar; 25.82±8.81% respectively. Correlation analysis showed no correlation between scar core percent (r=0.233, p=0.24), peri-infarct zone percent (r=0.144, p=0.57) and total scar percent (r=0.123, p=0.54) respectively.
Conclusion: There was no association between scar size, infarct heterogeneity and Tpe interval. Our data suggest that these two modalities may reflect different arrhythmogenic mechanisms.



OP-157

Comparison of Electrocardiographic Criteria in Hypertrophic Cardiomyopathy Patients with and without Apical Aneurysm

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Background: Apical aneurysms in patients with hypertrophic cardiomyopathy(HCM) represent an underrecognized but clinically important subset of HCM patients,often requiring a high index of suspicion.It is commonly associated with adverse clinical course,including sudden cardiac death and embolic stroke;however it may be frequently missed by echocardiography because of poor image quality of left ventricular apex, particularly between the apical hypertrophic cardiomyopathy (ACM) and mid-ventricular obstructive hypertrophic cardiomyopathy (MVO-HCM). Previous descriptions of an association of apical aneurysm with ST segment elevation (STE) have been confined to case reports or small patient series.
Objectives: We aimed to compare electrocardiographic STE in HCM patients with and without apical aneurysm.
Methods: We developed this clinical review using an extensive MEDLINE review of the literature and data from our laboratories. By means of 12-lead electrocardiography, we measured the maximal voltage of negative T-wave in all leads, and categorized the repolarization abnormalities as ST-segment depression, STE or isoelectric ST segment (Figure 1).
Results: There were 29 ACM patients without apical aneurysm (Group 1; 52.6 ± 17.7years, 69% male) and 28 MVO-HCM patients with apical aneurysm (Group 2; 59.6±13.2years, 57% male). The STE in V4-6 derivations were statistically more frequent in patients with apical aneurysm compared to those without aneurysm (93% vs 7%, p<0,001).There was a positive correlation between the presence of the STE in V4-6 derivations and the presence of the apical aneurysm (Spearman's ρ=0.895,P<0.001).
Conclusions: Clinicians and specifically echocardiographers must pay special attention on the electrocardiography to correctly detect the frequently overlooked apical aneurysm in HCM patients, and should be careful for apical aneurysm in the presence of STE in V4-6 derivations.