1184 Clinical Electrocardiographic Aspects of Drug Therapy

Wednesday, April 1, 1998, 9:00 a.m -11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m.–11:00 a.m.

1184-89 The Extent of CTG Array Repeat Expansion Predicts Electrocardiographic Abnormalities in Myotonic Dystrophy

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In myotonic dystrophy (DM) the severity of neuromuscular disease correlates with genomic CTG array repeat expansion (CTG-N). To evaluate whether cardiac conduction disease correlates with CTG-N we obtained 12-lead electrocardiograms in 22 consecutive individuals with diagnosed DM participating in the Arrhythmia in DM Study. The age was 42 ± 10 years (mean ± SD) with duration of diagnosis of DM for 13 ± 10 years. All individuals had prolonged CTG-N of greater than 50 repeats (640 ± 450, range 66–1456). The 12-lead electrocardiogram was abnormal in 16 of the 22 individuals. Quantitative markers of conduction disease including a prolonged PR interval (PRI) >200 ms and/or a QRS duration (QRSd) >100 ms was present in 15. The PRI was 195 ± 32 ms, range 140–284 and QRSd was 108 ± 26 ms, range 80–200. Using Spearman Rank Correlation PRI and QRSd correlated with age and duration of diagnosis ($P < 0.01$) but not CTG-N. With stepwise multiple regression and correcting for age, CTG-N correlated with QRSd ($P < 0.05$) but not PRI.

Conclusion: Electrocardiographic abnormalities of cardiac conduction in DM as measured by increasing PRI and QRSd progress with increasing age. If age is corrected for, then increasing CTG-N predicts worsened distal conduction disease as reflected in increasing QRSd. The PRI does not correlate with CTG-N after age correction, possibly reflecting the less specific nature of this measurement for distal conduction disease. These findings suggest a direct effect of CTG-N on the severity of distal cardiac conduction disease in DM.

1184-90 The ECG has Limited Value for Detecting Left Atrial Enlargement

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Background: Electrocardiography is used commonly for assessing left atrial enlargement (LAE), but has not been carefully evaluated against echocardiographic left atrium (LA) area measurements.

Methods: 149 patients with first myocardial infarction had ECG and Echo within 48 hrs of discharge and at 18 months, providing 298 observations. LA volume was determined by planimetry of an end-diastolic echo frame in the long axis parasternal view. LA waveforms were measured from ECGs by a single author blinded to echo results. LAE was determined by standard published criteria: 1) negative phase of P wave in lead V1 > 0.04 sec width, 2) negative phase of P wave in V1 > 1 mm depth, 3) P-terminal force in V1 > 0.04 mm/sec, 4) notched P wave with inter-peak interval >0.04 sec in lead II, 5) P wave duration ≥0.12 sec in lead II.

Results: Of the 149 patients, 42 had LAE [defined as echo LA area ≥20 cm2]. The accuracy of ECG criteria for echocardiographically defined LAE is shown in the table. Linear regression was performed using each ECG criterion plotted against echo LA area, and did not significantly correlate (all $p > 0.1$). 48 of 87 patients with falsely positive ECG criteria for LAE had left ventricular hypertrophy (posterior & septal thickness ≥24 mm).

ECG Criteria	1	2	3	4	5	1 & 3	2 & 5
Sensitivity, %	44	10	38	14	8	44	4
Specificity, %	53	91	64	90	93	52	99

Conclusion: ECG criteria for left atrial enlargement have very limited sensitivity but high specificity for echocardiographically determined left atrial enlargement.

1184-91 Extent of Early ST Segment Electron Resolution in Acute Myocardial Infarction and Rowing Ventricular Dissinergy and Stress Induced Myocardial Limb and Viability

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Recent studies demonstrated the prognostic value of the extent of ST segment elevation resolution (ST%-↓) after fibrinolytic therapy in patients (pts) with AMI. The aim of the study was to evaluate the relation between the extent of ST%-↓ 3 hours after fibrinolytic therapy for AMI and resting wall motion abnormalities (WMA) and stress induced myocardial Schema (MI) and viability (MV) in infarct related artery perfusion territory. One-hundred ninety seven one pts (mean age 61 ± 9 years) with fast AMI, treated with fibrinolytic therapy within 12 hours of symptom onset, underwent: 1) 12-lead ecg evaluation before and 3 hours abter fibrinolytic drug administration, 2) Peak creatine-kinase (CK) level 3) Dobutanune echocardiographic test (DET) 10 ± 2 days after AMI to evaluate DET-induced homozonal MI, MV and the extent of resting WMA. Homozonal MI was defined as worsening or new WMA in the infarcted artery perfusion territory. MV was defined as improving resting WMA in at least two infarct segments during low dose DET. The extent of WMA was derived calculating the resting wad motion score index (WMSI). Pts were classified in 3 groups according to the extent of ST%-↓: Group 1: ST%-↓ ≤35%; Group 2: 35% < ST%-↓ > 70%; Group 3: ST%-↓ ≥ 70%.

	Group 1 (50 pts)	Group 2 (63 pts)	Group 3 (84 pts)	p
CK (UI/l)	3113 ± 2475	3088 ± 1952	2239 ± 1929	0.018
WMSI	1.62 ± 0.47	1.68 ± 0.41	1.43 ± 0.34	0.0001
MI (pts)	8 (16%)	6 (9%)	12 (14%)	0.56
MV (pts)	16 (32%)	18 (28%)	14 (17%)	0.09

Conclusions: Pts with hiker eat of ST%-↓ 3 hours after fibrinolytic therapy have a less extent of resting WMA but a similar incidence of stress induced homozonal myocardial ischemia and viability than pts with lower extent of ST%-↓.

1184-92 Limits to the 12-Lead Electrocardiographic Diagnosis of Acute Myocardial Infarction: Can we Improve on ST Elevation?

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Background: ST elevation (STE) on the 12-lead ECG remains central for diagnosis of acute myocardial infarction (AMI), despite 30–50% of patients presenting with atypical ECG changes. Previous studies have found use of additional ECG features may improve diagnosis but most studies have been small or subject to selection bias.

Methods: We recruited 1041 unselected consecutive *patients* with chest pain (201 AMI with STE, 134 AMI with atypical ECG, 706 not AMI) and 149 *controls* without chest pain. A single, blinded observer recorded abnormal ECG features including Q waves (≥0.03 ms & >25% Rwave), STE (≥1 mm), ST depression (≥1 mm), and T-inversion (≥1 mm). AMI diagnosis was made by WHO criteria. Subjects were randomly divided into a training (T) set (587) and validation (V) set (603). Logistic regression models based on features predictive of AMI in the Tset were prospectively tested in the Vset. Models included (i) STE in any lead (ii) site specific STE (iii) any abnormal feature in any lead (iv) any site specific abnormal feature.

Results: STE models had reasonable specificity but poor sensitivity for AMI. Use of 1 mm STE in leads V1–V4 instead of 2 mm, ≥2 contiguous leads, or reciprocal changes did not improve overall classification. Both simple and complex feature models only improved specificity with further loss of sensitivity.

	Tset %sens	spec	Vset %sens	spec
STE: any lead	64	83	67	81
STE: specific site	43	95	56	94
Feature: any lead	48	93	54	96
Feature: specific	48	94	57	94

Conclusion: The ECG is a sub-optimal tool for diagnosis of AMI. Use of ECG features in addition to STE does not improve overall diagnosis. A rapid, accurate non invasive method for early diagnosis of AMI is still required.

1184-93 Diltiazem Can Prevent the "Reverse-Use Dependence" Characterizing the Class III Antiarrhythmic Effect of Sotalol

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Background: The prolongation of the refractory period by class III antiarrhythmics is attenuated at short cycle lengths. Ibis is thought to be related to the increase of the intracellular Ca^{++} at rapid heart rates. We investigated whether Diltiazem (D) had any effect on the QT I segment prolongation exerted by Sotalol (S) at different heart rates.

Methods: 10 pts with ventricular arrhythmias but without evident structural heart disease were ECG monitored for 24 hours first in basal conditions and