Ia antiarrhythmic drugs. Studies to date have been in resting patients, however, and the effects of heart rate increases on QT dispersion have not been studied. To evaluate the latter, we measured QT dispersion on a beat-to-beat basis in 13 normal subjects (8 male, 5 female, mean age = 30 yrs) before, during and after graded supine bicycle exercise. The 12-lead ECG was digitized at 500 samples/sec/lead and recorded directly to a PC hard disk for later analysis. The ECG was recorded at rest for 15 min, during exercise to exhaustion, then during recovery for 15 min. The times from Q onset to T wave peak (QTp) and end (QTe) were automatically measured on every beat in each lead using custom software. Median values for QTp and QTe were obtained during specified 10 msec R-R interval windows (e.g., between 695 and 705 msec for the 700 msec values). QTd (longest QT minus shortest QT) was calculated for all 12 leads (12L) and for the 6 precordial leads only (VL). Average QTd values for the 13 subjects (all intervals in msec; $\bar{X}$ = average [n = 7], SD = standard deviation, CV = coefficient of variation):

QTd using:	R-R Intervals							X	SD	CV
	450	500	600	700	800	900	1000			
QTp in 12L	39.6	36.4	37.9	41.6	37.3	38.2	34.9	38.0	2.2	5.7%
QTp in VL	28.8	28.5	24.3	27.9	27.0	29.2	27.1	27.5	1.7	6.0%
QTe in 12L	37.0	35.4	34.1	28.8	27.4	27.8	29.5	31.4	4.0	12.6%
QTe in VL	24.8	24.1	22.3	17.4	20.7	19.1	21.7	21.4	2.6	12.3%

Regression analysis revealed no significant changes in QTd with changes in R-R intervals, except for QTe in 12L; examination of those data revealed problems with identifying the end of the T wave in standard leads at short R-R intervals. QT intervals shortened normally with exercise in all leads. *Conclusions:* QT dispersion does not change with exercise-induced heart rate increases in normal subjects. QT dispersion is smaller in the precordial leads than in all 12 leads. QT dispersion is greater, and is measured with less variance, using QTp. These data provide a base for comparison with disease states such as myocardial infarction.

2:15

801-2 The Electrocardiographic Ventricular Repolarization During Stress from Awakening on Alarm Call

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Ambulatory electrocardiograms of 30 physicians were recorded during alarm calls at night while on duty in hospital. Men/women were 21/9, mean age was 37 ± 7 years, all were healthy. At the arousal from sleep, T-wave inversion (TWI) in lateral chest lead, ST-segment depression (STD) ≥0.1 mV, heart rate (HR) and QT interval (QT) were determined before and 5, 10, and 30 sec after signs of reaction in electrocardiogram, and at peak HR coinciding at 17 see on average. For comparison, QT was also measured outside the arousal episodes at corresponding HR levels but while the rate had remained stable (QTs) for at least 1 min. Number of subjects (N) or mean ± SD values were:

	Before	5 sec	10 sec	17 sec	30 sec
TWI (N)	0	0	10	12	4
STD (N)	0	0	5	7	1
HR (/min)	55 ± 7	92 ± 12	100 ± 15	112 ± 18	97 ± 24
QT (msec)	428 ± 28	397 ± 23	390 ± 40	362 ± 41	352 ± 40
QTs (msec)	410 ± 18	334 ± 16	323 ± 15	303 ± 15	327 ± 14
QT − QTs (msec)	+18	+63	+67	+59	+25

Overall, short-lasting episodes of TWI occurred in 18 (60%) and STD in 8 (27%) subjects, signifying sudden sympathetic overactivity. During the early part of the arousal, till the time of peak HR, QT markedly exceeded QTs, but at 30 sec the difference had almost disappeared. In conclusion, commonly encountered events like a wake-up call can evoke changes in ventricular repolarization, associated with a delay in its adaptation to heart rate. Same phenomena in electrically unstable hearts may mediate stress-provoked arrhythmias.

2:30

801-3 Differential Effect of d-Sotalol on QT Dispersion in Acute Drug Responders Versus Non-Responders

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QT interval dispersion measured as interlead variability of QT, is a marker of dispersion of ventricular repolarization and, hence, of cardiac electrical instability. The influence of the new class III agent d-sotalol on QT dispersion (QT disp) was investigated in 20 patients (pts) with recurrent sustained ventricular tachycardia (VT) and/or ventricular fibrillation (VF). All pts had inducibile VT/VF at baseline and were electrophysiologically studied under oral d-sotalol (460 ± 94 mg/day). In 10 pts d-sotalol suppressed the induction of VT/VF, whereas 10 pts did not respond. QT and RR intervals of 3 consecutive beats were measured in 6 simultaneously recorded precordial leads (V1–V6) by two independent blinded investigators. Under oral d-sotalol, the QT disp was reduced in 9/10 responders but only in 4/10 non-responders, despite comparable doses of the drug in both groups. The relative changes between control and under d-sotalol therapy in responders and non-responders of corrected maximal QT (QTc max.), QT disp., dispersion of the QTc (QTc disp.) and adjusted QTc (adj. QTc disp) are presented as means ± SD.

	responder	non-responder	p
Δ QTc max.	32.9 ± 69.2	27.5 ± 41.4	NS
Δ QT disp.	−30.3 ± 16.8	20.3 ± 38.2	0.001
Δ QTc disp.	−34 ± 31	18.5 ± 37.8	0.003
Δ adj. QTc disp.	−13.3 ± 12.9	7.5 ± 15.6	0.004
Dosage	480 ± 103	440 ± 84	NS

The new class III agent d-sotalol produced a significant reduction of QTc dispersion in responders in contrasts to non-responders.

Conclusion: QT dispersion may be potentially useful in the assessment of the efficacy of class III antiarrhythmic drugs. This effect may be due to homogenization of repolarization in both normal and diseased myocardium representing the arrhythmogenic substrate.

2:45

801-4 Prognostic Implications of QT and QU Interval Measures in Acute Myocardial Infarction

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Prolongation of the QT interval corrected using Bazett's formula (QTc) has been reported as a marker for increased risk of arrhythmic events after acute myocardial infarction (AMI). However, the QU interval changes have not been examined. At the same time, QU interval may be of clinical significance, especially in the light of recent experimental evidence linking the U wave with the subpopulation of the so-called M cells within myocardial wall. To evaluate prognostic significance of QT and QU interval measures in AMI, we studied 512 survivors of acute phase of their first myocardial infarction. Patients with conduction defects and drugs likely to affect QT measures were not included into the analysis. The following intervals were estimated in all the measurable leads on a standard predischarge 12-lead ECG (25 mm/sec paper speed) using a digitizing pad — mean RR, mean and max QT, and mean QU. All QT and QU intervals were subsequently corrected for heart rate using Bazett's formula. At one year follow-up, 23 patients (Group I, 19 male, mean age 58.7 ± 8.9 years) suffered arrhythmic events (VT/VF or sudden cardiac death). This subset of patients was compared with arrhythmia-free group of 489 subjects (Group II, 385 male, mean age 56.1 ± 9.2 years). Statistical analysis was performed using unpaired t-test and ANOVA, results are expressed as mean ± SD.

Group	QT mean	QTc mean	QT max	QTc max	QU mean	QUc mean
I	358.7 ± 31.5	426.6 ± 30.7	396.5 ± 38.5	472.8 ± 40.3	459.5 ± 58.7	535.2 ± 41.3
II	387.3 ± 44.1	423.9 ± 24	421.7 ± 51.5	467.9 ± 79.1	552.0 ± 73.9	585.7 ± 55.1
p <	0.002	NS	0.02	NS	0.001	0.01

The significant difference in QU and QUc, but not in QT intervals persisted even after elimination of the effect of heart rate (ANOVA: $p < 0.007$ and 0.011, respectively).

Conclusion: The differences in the QT but not QU interval measures in the 2 groups can be explained by differing heart rates. Shorter QU interval seemed to identify patients at risk of arrhythmic events after AMI. The pathophysiological basis for this finding is not clear, but could be related to differences in the subpopulation of M cells within myocardial wall.

3:00

801-5 Dynamic QT Behaviour After Myocardial Infarction and Influence of Beta-blocker Therapy

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The influence of myocardial infarction on dynamic QT behaviour is not known but could be important in arrhythmogenesis. Therefore we studied the dynamic QT behaviour in 43 patients with and 22 patients without (AMI+ and AMI−) beta-blocker therapy after myocardial infarction in comparison with dynamic QT behaviour in normal subjects. For dynamic QT measurements a 24 hour ECG was used and 4 segments of 6 hours: Morning (6–12), Day (12–18), Evening (18–24), Night (0–6). For each 30 second period a mean RR and a mean QT interval (QTend) was measured automatically with validated software developed by ELA Medical, France. The slope of the regression line relating the QT interval to the RR interval was used for dynamic QT