la 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 digi-
tized 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 (QT6) were automatically measured on every beat
in each lead using custom software. Median values for QTp and QT6 were
obtained during specified 10 msec R-R interval windows (e.g., between 695
and 705 msec for the 700 msec values). QT6 (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; X = average
in [n = 7]; SD = standard deviation; CV = coefficient of variation).

<table>
<thead>
<tr>
<th>QTp using</th>
<th>R-R Intervals</th>
<th>450</th>
<th>500</th>
<th>600</th>
<th>700</th>
<th>800</th>
<th>900</th>
<th>1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>QTp in 12L</td>
<td>33.6 ± 3.4</td>
<td>37.9</td>
<td>41.6</td>
<td>37.2</td>
<td>38.2</td>
<td>34.9</td>
<td>38.0</td>
<td>3.2</td>
</tr>
<tr>
<td>QTp in VL</td>
<td>28.8 ± 2.5</td>
<td>24.3</td>
<td>27.0</td>
<td>27.0</td>
<td>29.2</td>
<td>27.2</td>
<td>27.1</td>
<td>2.7</td>
</tr>
<tr>
<td>QT6 in 12L</td>
<td>37.0 ± 3.4</td>
<td>34.1</td>
<td>28.8</td>
<td>27.4</td>
<td>29.6</td>
<td>29.5</td>
<td>21.4</td>
<td>4.0</td>
</tr>
<tr>
<td>QT6 in VL</td>
<td>24.8 ± 2.4</td>
<td>22.3</td>
<td>17.4</td>
<td>20.7</td>
<td>19.1</td>
<td>21.7</td>
<td>21.4</td>
<td>2.6</td>
</tr>
</tbody>
</table>

Regression analysis revealed no significant changes in QTd with changes
in R-R intervals, except for QT6 in 12L: examination of 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. Con-
clusions: QT dispersion does not change with exercise-induced heart rate in-
creases in normal subjects. QT dispersion is smaller in the precordial leads
than in all 12 leads. QT dispersion is greater, and is measured with
more variance, using QTp. These data provide a base for comparison with disease
states such as myocardial infarction.

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

Laure Toivonen, Kirsu Helenius, Matti Viiitasalo. Helsinki University Central Hospital, Helsinki, Finland

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 (TII) in lateral chest lead, S1-segment depression (STD) ≥ 10% of 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:

![Table](image)

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 re-
polarization, associated with a delay in its adaptation to heart rate. Same phenomena in electrically unstable hearts may mediate stress-provoked ar-
rhythmias.

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

Daniel F. Lighazan, Corina Ipec, Thomas Hilbert, Wolfgang Kühles, Johannes Brachmann. Division of Cardiology. University of Heidelberg, Germany

QT interval dispersion measured as interlead variabiliy 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 in-
ducible VT/VF at baseline and were electrophysiologically studied under oral
of 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 blind 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 cor-
rected maximal QT (QTc max), QT disp., dispersion of the QTc (QTc disp) and adjusted QTc (adj. QTc disp) are presented as means ± SD.

<table>
<thead>
<tr>
<th>Group</th>
<th>QTc max</th>
<th>QTc disp</th>
<th>QTc disp.</th>
<th>QTc disp.</th>
<th>QTc disp.</th>
<th>QTc disp.</th>
<th>QTc disp.</th>
<th>QTc disp.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responder</td>
<td>423 ± 92</td>
<td>277 ± 41</td>
<td>4.1 ± 0.0</td>
<td>1000</td>
<td>423 ± 92</td>
<td>277 ± 41</td>
<td>4.1 ± 0.0</td>
<td>1000</td>
</tr>
<tr>
<td>Non-responder</td>
<td>687 ± 185</td>
<td>103 ± 28</td>
<td>0.001</td>
<td>0.001</td>
<td>687 ± 185</td>
<td>103 ± 28</td>
<td>0.001</td>
<td>0.001</td>
</tr>
</tbody>
</table>

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

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

Ravish Kishore, Josef Kautzner, John A. Caron, Marek Malik. St. George's Hospital Medical School, London, United Kingdom

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 disorders and drugs likely to affect QT measures were not included into the analysis. The following intervals were estimated in all the measur-
able 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, mean age 56 ± 8.2 years). Statistical analysis was performed using unpaired t test and ANOVA, results are expressed as mean ± SD.

<table>
<thead>
<tr>
<th>Group</th>
<th>QT mean</th>
<th>QU mean</th>
<th>QUc mean</th>
<th>QUc mean</th>
<th>QUc mean</th>
<th>QUc mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>418.7 ± 41</td>
<td>103 ± 28</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Group II</td>
<td>456.9 ± 45</td>
<td>185 ± 37</td>
<td>0.003</td>
<td>0.003</td>
<td>0.003</td>
<td>0.003</td>
</tr>
</tbody>
</table>

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 QT inter-
val 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.

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

Rene Tavernier, Luc Jordaens, Veronique Schiettekatte, Denis L. Clement. University Hospital, Ghent, Belgium

The influence of myocardial infarction on dynamic QT behaviour is not known but could be important in arrhythmogenesis. Therefore we studied the dy-
namic 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 val-
dlicated software developed by ELA Medical, France. The slope of the regres-
sion line relating the QT interval to the RR interval was used for dynamic OT
measurements in each 6 hour segment. Mean slopes for each segment are presented.

<table>
<thead>
<tr>
<th></th>
<th>Morning</th>
<th>Day</th>
<th>Evening</th>
<th>Night</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normals</td>
<td>VA</td>
<td>NS</td>
<td>59 ± 18</td>
<td>18 ± 5</td>
</tr>
<tr>
<td>AMI</td>
<td>58 ± 18</td>
<td>18 ± 5</td>
<td>63 ± 21</td>
<td>18 ± 4</td>
</tr>
<tr>
<td>AMI+</td>
<td>46 ± 14</td>
<td>14 ± 4</td>
<td>51 ± 12</td>
<td>15 ± 5</td>
</tr>
</tbody>
</table>

*p < 0.05; **p < 0.005; ***p < 0.0005

We conclude that myocardial infarction has an important influence on dynamic QT slope. The relationship was stronger during the day, the evening an the night are more pronounced compared to slopes in normal individuals. Beta-blocker therapy results in a significant decrease of QT-slope after myocardial infarction and tends to normalize the rate dependent changes of the QT interval.

**802-1** A New Noninvasive Test to Predict Inducible Ventricular Tachycardia in Patients with Unexplained Syncope: QT Dispersion

Leigh A. Hutchinson, Fidalsa Moreno, Frederick A. Ehrilt, Jonathan S. Steinberg, Sr. Luke's-Roosevelt Hospital Center and Columbia University, New York, NY; LDS Hospital, Salt Lake City, UT

Regional disparities in ventricular repolarization are known to promote reentrant ventricular arrhythmias; recent work suggests that this may be assessed noninvasively using QTI/QT dispersion on the surface ECG. However, very little data is available on its clinical use. We studied pts with unexplained syncope to determine if inducible VT could be predicted by QT dispersion (QTD, defined as QTmax-QTmin), QT standard deviation (QTDs), and equivalent QT intervals. Values were also corrected using Bazett's formula. We enrolled 38 pts (22 M, age = 59 ± 18 yrs, 29% prior MI) who underwent programmed electrical stimulation (PES). Standard ECGs were recorded prior to PES and analyzed by a computerized analyzer/digitizer. Results: Of the 38 pts, 9 (24%) had inducible sustained VT and 29 pts did not.

Standard QRS, QT and QTC, and corrected dispersion intervals were not significantly different between pts with and without inducible VT.

Conclusion: Confirming the relationship of dispersion of ventricular repolarization and ventricular reentry, several ECG parameters of dispersion were associated with inducible VT in patients with unexplained syncope. QT dispersion is a novel and noninvasive measure of risk of VT.

**802-2** Cardiovascular Risk Factors Levels and Changes Following Lifestyle Intervention in Families: The British Family Heart Study

David Wood, Stephen Pyke, Anni-Louise Kinmonth, Simon Thompson, British Family Heart Study Group, Department of Clinical Epidemiology, National Heart and Lung Institute, London, UK

2373 families were recruited to a national randomised controlled trial of cardiovascular screening and lifestyle intervention in 26 general practices in 13 towns across Britain. 2054 (97%) comprised both a male and female partner and amongst these 1477 (72%) were represented at screening by both partners. There was a significant association between reported cigarette smoking habits in partners (odds ratio 0.95% CI 4.6, 8.0) at baseline. Where both partners were initially smokers, men were 5 times and women were 10 times (p < 0.05) more likely to have quit where their partner had also quit. Body mass index (BMI) measurements were correlated (r = 0.21, p < 0.0001) at baseline and one year changes in BMI in partners were also significantly correlated (r = 0.26, p < 0.0001). Similar results were found for systolic blood pressure, blood cholesterol and blood glucose cross-sectionally (r = 0.14, 0.10, 0.13 respectively, p < 0.0005) and longitudinally (r = 0.14, 0.24, 0.19 respectively, p < 0.0001). Whilst the cross sectional inter-partner relationships are weak the longitudinal results demonstrate the scope for cardiovascular intervention programmes to capitalize on the tendency of partners to change together. Men with women most able to improve their risk factors over a one year period tend to have partners who also improve substantially. Therefore, for lifestyle intervention programmes in middle aged men and women, targeting a couple together rather than as individual patients may result in a greater reduction in cardiovascular risk factors through mutual reinforcement of lifestyle changes.

**802-3** Treatment of Ventricular Arrhythmias After the Cardiac Arrhythmia Suppression Trial (CAST): A Survey of US Physicians

Yves Rosenberg, Eleanor Schon, Mario Stylianou, Albert Parker. National Heart, Lung, and Blood Institute, Bethesda, MD

To determine how US physicians treat patients with asymptomatic ventricular arrhythmias we conducted in fall 1992, after the publication of all CAST results, a telephone survey of a randomly selected sample of 1072 American generalists (G) and cardiologists (C), as part of a survey designed to study how the results of three major cardiovascular clinical trials have influenced medical practice. 730 physicians responding treating patients with ventricular arrhythmias. The response rate was 63% (G: 65.3%, C: 60.9%). 44.2% of G, but only 22.1% of C would routinely use an antiarrhythmic drug (AAD) or device to treat a patient presenting ventricular premature depolarizations (VPD) who had a myocardial infarction (MI) in the past six months (p < 0.001). 68.2% of G and 40% of C would treat the same patient if he presented asymptomatic nonsustained ventricular tachycardia (NSVT), p < 0.001. For the former condition, the first choice drug would be a β-blocker (G = 53.5%, C = 74%, p < 0.002), but 28.3% of G and 23.5% of C would use a class I AAD (with <1% using a class IC). 38.9% of G and 16.1% of C would treat a patient with coronary heart disease and an ejection fraction <40% presenting asymptomatic VPD, 66.2% of G and 26.6% of C would treat asymptomatic NSVT, p < 0.001. For asymptomatic VPD, the first choice drug is a Class I AAD (G = 46.4%, C = 47.2%); 27.4% of G and 44.4% of C would prescribe a β-blocker; 11.9% of G but no C would prescribe a calcium-blocker. For the physicians treating NSVT, the first choice drug is:

<table>
<thead>
<tr>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>AAD</td>
<td>IC</td>
<td>IC</td>
<td>IC</td>
<td>AAD</td>
</tr>
</tbody>
</table>

G (n = 145) 49% 0% 0% 7% 17% 2% 9.1% 4.8% 11.7%
C (n = 91) 51.7% 12.1% 1.1% 29.7% 2.2% 2.2% 0% 1.1% 1.1%

Total 50% 8.9% 0% 22% 0.8% 6.8% 3% 7.8%