In this patient population, the variables of age and hyperlipidemia did not significantly predict the presence of CAD; however, an ABI at rest of ≤0.90 versus other cardiovascular risk factors was an independent predictor of the presence of CAD and 3-vessel and left main CAD, but not of 1- or 2-vessel CAD. Cardiovascular risk factors were not predictive of CAD in this population, and this may be explained by the small sample size of the population in our study.[1,2] An abnormal ABI predicts the overall presence of CAD; however, the results of the logistic regression and receiver-operating characteristic curve analysis suggest that this is driven largely by the identification of patients with severe CAD, including 3-vessel and left main disease.

Limitations of our study included the small sample size and possibility of bias due to selection of a high-risk group, because all patients were referred for coronary angiography. However, this should apply to all groups of patients in the study. The results of this study also cannot be extrapolated to the general population, because we enrolled only African-Americans with risk factors; our hypothesis was that the ABI would be of value in screening selected intermediate-risk patients who may benefit from coronary angiography for risk stratification. Our population thereby meets these criteria and provides useful information to physicians treating these patients.

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Computerized QT Dispersion Measurement and Cardiovascular Mortality in Male Veterans

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We examined the prognostic value of computerized measurements of QT dispersion in 37,579 male veterans. The results of our study showed that QT dispersion is a poor independent predictor of cardiovascular mortality. ©2004 by Excerpta Medica, Inc.

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It has been proposed that QT dispersion, which is evident on a surface electrocardiogram (ECG), reflects underlying heterogeneity of ventricular repolarization and is a marker of vulnerability to ventricular arrhythmias. Despite intensive interest and research on this topic over the past decade, the prognostic significance and clinical utility of this measurement remains unclear. This has led to an ongoing debate as to whether QT dispersion should be regarded as the electrophysiologic "Holy Grail"[1] or as "the greatest fallacy in electrocardiography in the 1990s."[2] There has been a resurgence of interest in the prognostic implications of the ECG at rest in patients with structural heart disease. It has not been more evident than in patients receiving implantable cardioverter-defibrillators for primary prevention of sudden cardiac death and in patients receiving biventricular pacing systems for medically refractory heart failure. There is obvious appeal to using an inexpensive, noninvasive risk-stratification tool like the ECG to allocate costly therapies in these 2 patient groups. For instance, the new Medicare guideline for coverage for implantable cardioverter-defibrillator placement includes QRS duration as part of the criteria.[3] The purpose of the present study was to assess whether QT dispersion is a predictor of cardiovascular mortality.

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All initial ECGs of consecutive male veterans who obtained ECGs for any reason at the Palo Alto Veterans Administration Medical Center (Palo Alto, California) from April 1987 to December 1999 were examined. Because of the disproportionately small numbers of women who receive Veterans Administration care (98% men), women were excluded. Patients with electrocardiographic evidence of atrial fibrilla-

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TABLE 1 Characteristics of the Total Study Cohort and Results of Statistical Testing Between Those Surviving and Those Who Died of Cardiovascular Causes

	Total (n = 37,579)	Cardiovascular Death (n = 2,945)	Survived (n = 34,634)
Age (yrs)	55.7 ± 14.2	66.7 ± 11.2	54.7 ± 14.0*
Height (m)	1.76 ± 0.08	1.75 ± 0.08	1.76 ± 0.08*
Weight (kg)	84.5 ± 17.6	81.6 ± 16.6	84.7 ± 17.6*
Heart rate (beats/min)	73.1 ± 14.9	75.7 ± 15.3	72.9 ± 14.8*
Left ventricular hypertrophy	1,723 (4.6%)	356 (20.7%)	1,367 (79.3%)*
Right ventricular hypertrophy	89 (0.2%)	5 (5.6%)	84 (94.4%)†
ST depression	5,129 (13.6%)	811 (15.8%)	4,318 (84.2%)*
Intraventricular conduction delay	1,088 (2.9%)	123 (11.3%)	965 (88.7%)*
T-wave abnormalities (Minnesota code 5s)	8,268 (22.0%)	1453 (17.6%)	6,815 (82.4%)*
Pathologic Q waves	4,490 (11.9%)	740 (16.5%)	3,750 (83.5%)*
QRS duration (ms)	93.1 ± 10.9	96.2 ± 12.6	92.8 ± 10.7*
Mean QT (ms)	386.4 ± 38.4	390.5 ± 40.5	386.1 ± 38.2*
Mean QTc (ms)	420.0 ± 24.2	432.1 ± 27.4	419.0 ± 23.6*
Mean QT dispersion (ms)	31.3 ± 20.3	34.3 ± 22.6	31.0 ± 20.0*
Mean QTc dispersion (ms)	32.9 ± 21.2	36.7 ± 24.2	32.6 ± 20.9*

*$p < 0.0001$; †$p = 0.55$.

TABLE 2 Mean QT Dispersion and QTc Dispersion Measurements for the Study Cohort Divided by Normal ECG and Abnormal Electrocardiographic Status and by Survival Status, With Statistical Differences Between Those Who Died and Those Who Survived

Variables	Total (n = 23,605)	Cardiovascular Death (n = 978)	Survived (n = 22,627)
Cohort with Normal ECG			
QT dispersion	29.1 ± 17.8	29.1 ± 18.1	29.1 ± 17.8*
QTc dispersion	30.4 ± 18.4	30.7 ± 19.0	30.4 ± 18.4†
Variables	Total (n = 13,974)	Cardiovascular Death (n = 1,96)	Survived (n = 12,2007)
Cohort with Abnormal ECG			
QT dispersion	34.9 ± 23.4	36.9 ± 24.2	34.6 ± 23.2‡
QTc dispersion	37.2 ± 24.7	39.7 ± 25.9	36.8 ± 24.5‡

*$p = 0.97$; †$p = 0.61$; ‡$p < 0.0001$.

Values are expressed as mean ± SD.

Note: Wolff-Parkinson-White paced rhythm bundle branch blocks, and atrial fibrillation were excluded from the total, normal and abnormal populations. In normal population Q wave myocardial infarction, intraventricular conduction delay, left ventricular hypertrophy, right ventricular hypertrophy, T-wave abnormality, and ST depression were also excluded.

tion, Wolff-Parkinson-White preexcitation syndrome, bundle branch block, and a paced rhythm were excluded from all analyses.

Computerized 12-lead, 10-second electrocardiographic recordings at rest were digitally recorded on the Marquette MAC system (Menomonee Falls, Wisconsin). Only the initial ECG was considered for patients with multiple ECGs in the database. Electrocardiographic abnormalities were defined according to GEMS IT 12 SL Program of the GE/Marquette electrocardiographic analysis system (GE Medical Systems IT, Menomonee Falls, Wisconsin). As outlined in the 12 SL ECG Analysis Program's Physician's Guide, the basic interval measurements in this analysis program are made based on "median beats." These beats are developed using simultaneously sampled data that allow for them to be time-aligned synchronously. QRS onset is determined when a rapid increase in slope change is detected in the area just in front of the QRS complex. T-wave offset is determined by using the slope changes along with predetermined thresholds to sense the end of the T wave via reduced slope changes. Special logic contained in the algorithm allows determination of the presence of P waves occurring in the terminal portion of the T wave to adjust the offset appropriately. The QT interval reported in the computerized interpretation is calculated as the time from the earliest onset of the QRS to the latest offset of the T wave across all 12 leads. This system has been validated and previously described.[4,5] The QT interval is corrected for heart rate using Bazett's formula[6] (QTc). QT dispersion was calculated as the difference between the maximum and minimum QT intervals as measured electronically in all 12 leads.

An abnormal ECG was defined as the presence of ≥1 of the following symptoms: pathologic Q waves, right or left ventricular hypertrophy, or abnormal ST segments and T waves. All remaining ECGs were classified as "normal." Additionally, patients with left or right bundle branch block were excluded because of limitations in the computerized measurements, which resulted in a recorded QT dispersion of zero in a predominance of these ECGs. QT dispersion measurements of zero were excluded from the study cohort.

The California Health Department Service was used to ascertain vital status as of December 31, 2000. The cause of death was available from death certificates, and all-cause death and cardiovascular death were used as end points. Mortality status was available for all patients in the database.

Number Crunching System Software (Kaysville, Utah) was used for all statistical analyses after transferring the data from a Microsoft ACCESS (Redmond, Washington) database. The *t* test and chi-square analysis were used to compare variables as univariates. Cardiovascular mortality was used as the end point for survival analysis. Those who underwent intervention could not be removed from observation because data regarding subsequent interventions were not available for all patients.

The final cohort consisted of 37,579 male veterans (mean age 55.7 ± 14.2 years) whose initial ECGs were considered in this study. The demographics and prevalence of selected electrocardiographic abnormal-

ities are described in Table 1. The mean QT dispersion and QTc dispersion were significantly shorter in the survivors than in those who died from cardiovascular causes (31 ± 20 vs 34 ± 23 ms, $p < 0.0001$ and 33 ± 21 vs 37 ± 24 ms, $p < 0.0001$, respectively). There were 23,605 patients (62.8%) with normal ECGs. The mean QT dispersion, mean QTc dispersion, and mortality statistics for patients with normal and abnormal ECGs are listed in Table 2. Patients with abnormal ECGs had greater mean QT dispersion ($p < 0.0001$) and QTc dispersion ($p < 0.0001$) values than patients with normal ECGs. QT dispersion and QTc dispersion were greater in those with abnormal ECGs and in those with abnormal ECGs who died from cardiovascular causes.

There were a total of 2,945 deaths (7.8%) from cardiovascular causes after a mean follow-up of 6.0 ± 3.8 years. The average annual mortality in the total population was 1.2%/year. The average annual mortality in the subsets with normal and abnormal ECGs was 0.5%/year and 2.3%/year ($p < 0.0001$), respectively. In the Cox regression analyses, QT dispersion, adjusted for age and heart rate, and age-adjusted QTc dispersion were significant independent predictors, with patients in the 90th percentile having a 1.9% annual cardiovascular mortality and a hazard ratio of 1.6 ($p < 0.0001$).

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Considerable research has been done to develop a simple and inexpensive screening test to identify patients at increased risk for ventricular arrhythmias and cardiovascular death. QT dispersion, defined as the difference between the maximum and minimum QT interval measurements on a standard 12-lead ECG, has received much attention over the last decade as such a potential tool. Unfortunately, the role, if any, of QT dispersion measurement in clinical practice still remains uncertain because of conflicting findings. A simple PubMed literature keyword search of "QT dispersion" yields >750 citations on the topic in the last 10 years alone. Therefore, the lack of a consensus on the prognostic significance and clinical utility of this measurement is not from lack of effort.

Factors such as small sample sizes and the use of different patient populations may explain differences in some studies. However, much of the discrepancies among these studies may be explained by the difficulty in accurately measuring reproducible QT intervals. Some of the harshest critics argue that QT dispersion reflects nothing more than random measurement error. Despite studies reporting the prognostic value of QT dispersion measurements in a variety of patient populations, the fundamental premise that QT dispersion reflects regional repolarization variation at all continues to be challenged.

The fundamental problem in the application of QT dispersion to clinical practice is the difficulty of accurately and reliably measuring QT intervals. Even if heterogeneity of repolarization can be detected on the surface ECG, there is growing doubt that these measurements can be obtained with the precision and reproducibility necessary for widespread clinical use. Both manual and computerized measurements have been shown to be potentially unreliable, with high interobserver, intraobserver, and even day-to-day intrapatient variabilities.[4–6] Even in the present study, computerized measurement of QT dispersion was hindered by the inability of the software to measure T-wave morphology in sufficient leads to measure QT dispersion, particularly in patients with bundle branch abnormalities. This lack of reliable reproducibility has been demonstrated not only in patients with abnormal ECGs, but in those with normal ECGs as well.[7] Experts have concluded that all QT measurements, whether obtained by hand or computer, are limited by low T-wave amplitudes,[8,9] superimposed U and P waves, and abnormal T-wave morphologies, all problems that are difficult to overcome. As a result of these problems, there is no consensus in published data on the reference values for QT dispersion in normal populations, let alone in diseased cohorts. Published studies of healthy subjects have reported mean QT dispersion values ranging from 10.5 to 71 ms.[10,11]

The prognostic power of QT dispersion measurement has specifically been studied in many focused populations, including post-myocardial infarction,[12] the elderly,[13] patients with left ventricular dysfunction,[14] diabetics,[15] those with structural heart disease,[16] and transplant patients,[17] as well as clinically healthy patients.[18] However, negative findings have been similarly found in other recent investigations. Okin et al[19] reported data from The Strong Heart Study, a community-based study of cardiovascular disease and risk factors in American Indians. In this population, using multivariate Cox regression analyses controlling for traditional cardiac risk factors, they concluded that although QT dispersion measurement was a significant predictor of cardiovascular mortality, it was not useful in predicting all-cause mortality. Similarly, Zabel et al,[12] using a QT dispersion cut-off value of 61 ms, found no relation between QT dispersion measurements and future arrhythmic or all-cause mortality in a prospective study of 280 infarct survivors.

The present study showed that in a large series of male patients, QT dispersion appears to be a weak predictor and is limited methodologically. It may just represent a measurement error most prevalent in patients with repolarization abnormalities that confound the recognition of the T wave.

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Comparison of Ability to Identify Left Atrial Thrombus by Three-Dimensional Tomography Versus Transesophageal Echocardiography in Patients With Atrial Fibrillation

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We sought to determine the potential use of recently introduced cardiac 3-dimensional computed tomography as an alternative to transesophageal echocardiography for examination of the left atrial appendage. Our data suggest that computed tomography is a potential alternative for assessing the anatomy of the left atrial appendage and for detecting thrombi.
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Recent advances in 3-dimensional (3-D) computed tomography (CT) of the cardiovascular system have introduced a new, exciting noninvasive modality that is gradually complementing other diagnostic tools available to the clinician. CT has been most effective in assessing cardiac morphology, such as identification of anomalous structures or cardiac masses.[1] Given that a reliable alternative imaging modality for evaluating the left atrium (LA) and left atrial appendage (LAA) is not available, we sought to determine the potential use of 3-D CT to visualize the LA and LAA for the detection or exclusion of thrombus.

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Thirty-one patients seen at the Cleveland Clinic Foundation with a history of persistent atrial fibrillation were evaluated in a comparison study of 3-D CT and transesophageal echocardiography (TEE). Twenty-eight patients were enrolled as part of an institutional review board–approved protocol for assessing the pulmonary venous ostium by 3-D CT and TEE before pulmonary vein ablation for atrial fibrillation. The remaining patients underwent clinically indicated CT scans and TEE studies (1 evaluation for constrictive pericarditis, 1 to rule out intracardiac mass, and 1 for evaluation of periaortic abscess).

Transesophageal echocardiographic examinations were performed using 5- to 7-MHz multiplane probes with an Advanced Technology Laboratories (ATL) HDI 5000 (Bothell, Washington) or Hewlett Packard Sonus (Andover, Massachusetts) system. Imaging was focused on the LA and LAA to obtain the maximal size and image quality. Multiple views, including a continuous sweep through the LAA from 0° to 180°, were obtained. A 45° view of the LAA at the mid-esophageal short axis of the aorta and a longitudinal view at the 90° mid-esophageal view with anterior and inferior left ventricular walls as landmarks were routinely obtained. Careful attention was paid to identify normal structures, such as the pectinate muscles in the LAA. A thrombus in the LA or LAA was

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