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The Effect of Acute Psychological Stress on QT Dispersion in Patients with Coronary Artery Disease

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Abstract

Background—Acute psychological stress can precipitate ventricular arrhythmias and sudden cardiac death in patients with coronary artery disease (CAD). However, the physiologic mechanisms by which these effects occur are not entirely clear. Mental stress-induced myocardial ischemia (MSIMI) occurs in a significant percentage of the CAD population. It is unknown if the proarrhythmic effects of psychological stress are mediated through the development of myocardial ischemia.

Objectives—To examine the effects of psychological stress on QT dispersion (QTd) among CAD patients and whether these effects are mediated via the development of myocardial ischemia.

Methods—Psychological stress was induced using a public speaking task. Twelve-lead electrocardiograms (EKG) were recorded at rest, during mental stress and during recovery. QTd was calculated as the difference between the longest and the shortest QT interval in the 12 lead EKG. Rest-stress myocardial perfusion imaging was also performed to detect mental stress-induced myocardial ischemia.

Results—Mental stress induced a significant increase in QTd compared to the resting condition ($p < 0.001$). This effect persisted beyond the first 10 minutes of recovery ($p < 0.001$). QTd was significantly associated with the development of mental stress ischemia with ischemic patients having significantly higher QTd during mental stress than non-ischemic patients ($p = 0.006$). This finding remained significant after controlling for possible confounding factors ($p = 0.01$).

Conclusion—Acute psychological stress induces a significant increase in QTd which persists for more than 10 minutes after cessation of the stressor. This effect seems to be, at least partially, mediated by the development of mental stress-induced myocardial ischemia.

Keywords

Psychological Stress; Coronary Artery Disease; Myocardial Ischemia; Ventricular arrhythmias; QT dispersion

A large body of evidence links acute psychological stress with the precipitation of cardiac arrhythmias and sudden cardiac death.¹ This has been documented in the setting of natural disasters as well as in laboratory settings.^{1–8} Additional studies have confirmed that acute psychological stress can lower the threshold for induced ventricular arrhythmias and precipitates spontaneous arrhythmic events in patients with coronary artery disease (CAD) and implantable cardioverter defibrillators (ICDs).^{9–11} The physiologic mechanisms by which these effects occur are not entirely clear. Psychological stress is associated with sympathetic nervous system activation which can induce transient myocardial ischemia in a significant proportion of the CAD population.^{12–17} Adrenergic stimulation alone, has the potential for altering the electrophysiologic properties of the myocardium and inducing inhomogeneous influences on myocardial repolarization.^{11, 18–21} However, it is unclear whether or not the pro-arrhythmic effects of mental stress are mediated via the development of myocardial ischemia.

Indices of spatial heterogeneity of myocardial repolarization, such as the inter-lead variability of the QT interval (QT dispersion; QTd), predict susceptibility to ventricular arrhythmias and sudden cardiac death.^{22–27} QT dispersion (QTd) is defined as the difference between the longest and the shortest QT interval on a 12 lead electrocardiogram (EKG).²² Very little is known about the effects of psychological stress on QTd. To date, only one study has evaluated these effects.²⁸ The study reported that mental stress induced significant prolongation of QTd in patients with CAD but not in healthy volunteers, suggesting that this effect was mediated via the development of myocardial ischemia. However myocardial ischemia was not measured in this study. To further investigate these findings, we measured QTd during rest and mental stress. We also examined for the occurrence of mental stress-induced ischemia using myocardial perfusion imaging.

Methods

Subjects

Participants in this study were recruited from outpatient clinics affiliated with university based medical centers. Eligibility criteria included age above 18 years and a documented clinical diagnosis of CAD supported by: 1) angiographic evidence of >50% stenosis in one or more coronary arteries or previous percutaneous coronary intervention (PCI) or coronary artery bypass graft surgery (CABG), 2) previous myocardial infarction (MI) documented with elevated troponin level in the range typical of MI, Q-wave abnormalities on ECG, or fixed, non-artifactual perfusion abnormalities on nuclear scan, or 3) a positive radionuclide pharmacologic or exercise stress test. Patients were excluded if they had unstable angina or acute MI within the two months preceding enrollment, a severe co-morbid medical problem restricting life-expectancy to less than 5 years, pregnancy or body weight over 400 lbs. The study protocol was approved by the University of Florida Institutional Review Board. Informed consent was obtained from all participants.

Mental stress procedure

Study procedures were performed in the morning after an over night fast. Beta-blockers, calcium-channel blockers and long acting nitrates were withheld the night before testing. Demographic and psychosocial characteristics were obtained prior to study procedures.

Patients were initially rested for 30 minutes in a reclined position in a temperature-controlled, dark and quiet room. Heart rate (HR), blood pressure and 12-lead EKG were recorded at baseline. Mental stress was then induced via a public speaking task performed in front of a small audience, as in prior research.^{29, 30} Participants were given a scenario describing a real life stressful event and asked to make up a realistic story around it. Participants were given two minutes to prepare their speech and three minutes to speak. They were told that their speech would be video-taped and later judged by the research staff on measures of its content, quality and whether it lasted for the entire 3 minutes. Hemodynamic and 12-lead EKG measurements were recorded at baseline, at every minute during the stress and at 10 minutes into the recovery period.

Myocardial perfusion imaging

Myocardial perfusion imaging with 99m-Tc-sestamibi was used. At one minute into the speech a total dose of 20–30 mCi (based on patient's body weight) was injected. This timing is based on previous reports that maximal heart rate, blood pressure and neurohormonal responses to mental stress usually occur at or near onset of the stressful task and ischemic abnormalities are induced relatively rapidly during this process.^{31–33} Stress perfusion images were acquired 30–60 minutes later using single photon emission computed tomography (SPECT) conventional methodology (64 projections over a circular 180 degree orbit, with the gamma camera set at a 140 keV energy peak with a 20 percent window).³⁴ A high resolution collimator and two dimensional Butterworth filter were used and trans-axial tomograms were reconstructed using back projection with a ramp filter. Resting images were obtained within one week of the stress test. The studies were interpreted by an experienced nuclear cardiologist (DSS) blinded to the condition (rest vs. stress). Rest and stress images were displayed on a 20 segment model and visually compared for number and severity of perfusion defects using a scoring method from zero to four; with zero being normal uptake and four no uptake. A summed difference score was calculated as the difference between summed stress and rest scores. Ischemia was defined as new or worsening perfusion defects during mental stress as compared to the resting baseline images with a summed difference score of ≥ 3 .

QT dispersion measurement

Standard 12-lead EKGs were recorded at baseline after a resting period, at every minute during the stress and at 10 minutes into the recovery period. The QT interval was manually measured by one observer (AM) from the onset of the QRS complex to the end of the T wave (defined as the return to the T-P isoelectric line).^{22, 23, 27, 28} If a U wave was present, the QT interval was measured to the nadir between the T and the U waves. If the T wave could not be clearly defined that lead was excluded. Only EKGs with >8 analyzable leads were included. QTd was calculated separately for each EKG as the differences between the shortest and the longest QT intervals. The maximum QTd during stress was compared to the resting and the recovery QTd. A second observer (MH) blindly performed QTd measurements on a randomly selected sample of EKGs ($n=40$). Measurements by the two observers were highly correlated, the concordance correlation coefficient (95% CI) was 0.792 (0.642 to 0.883).

Statistical Analysis

Results were expressed as means $\pm$ standard deviations for continuous variables and frequencies and percentages for categorical variables. Statistical significance was considered as $p < 0.05$. Univariable and multivariable analyses were used to examine the relationship between mental stress ischemia and QTd. In addition to the stress QTd and ischemia status, the latter analysis included, as covariates, the resting QTd, history of co-morbid conditions (diabetes and hypertension), medication status (beta blockers and calcium channel blockers)

and CAD severity factors (history of MI, CABG or PCI). Log transformation of the QTd measurement was conducted to satisfy the Gaussian distribution assumption of the model. All analyses were done using SAS statistical software (SAS Inc., Cary, NC).

Results

Patient characteristics and baseline data

A total of 70 patients were studied, of which 41 (59%) were males and 29 (41%) were females. The majority was Caucasian (80%) and the mean age was 63 ± 9 years. All participants had CAD. Demographic and clinical characteristics of the study population are described in Table 1.

QT dispersion during mental stress

Mental stress induced a significant increase in QTd, compared to the resting condition. The mean resting QTd was 24 ± 18 ms, the mean stress QTd was 43 ± 17 ms ($p < 0.001$). QTd was still significantly above baseline at 10 minutes into recovery with a mean value of 30 ± 16 ms (p value was < 0.001 for the difference between the mean rest and the recovery QTd). QT dispersion and hemodynamic responses to mental stress are shown in Table 2.

QT dispersion and mental stress-induced myocardial ischemia

Mental stress-induced myocardial ischemia occurred in 24 (35%) patients. Patients with mental stress-induced ischemia developed significantly higher increases in QTd during mental stress compared to patients without ischemia ($p = 0.006$). The mean QTd during stress (controlling for the resting QTd) was 50 ms (95% CI=44–56) in patients with mental stress ischemia compared to 39 ms (95% CI=35–44) in patients without ischemia (see Figure 1). This finding remained significant ($p = 0.012$) after controlling for possible confounding factors (see the statistical analysis section for listing of these factors). Additionally the severity of ischemia as measured by the summed difference score (SDS) was significantly correlated with the degree of increase in QTd from rest to stress ($r^2 = 0.40$; $p = 0.017$). The adjusted and unadjusted stress QTd by mental stress ischemia groups is shown in table 3.

Discussion

In this study we examined the effects of psychological stress on QTd, a measure of spatial heterogeneity of myocardial repolarization. Our findings indicate that psychological stress induces a significant increase in the QTd from the resting state. This effect seems to persist for at least 10 minutes after cessation of the stressful event. Furthermore patients with mental stress-induced ischemia had significantly higher increases in QTd during stress compared to non ischemic patients, suggesting that ischemia mediated the effects of psychological stress on QTd. The findings of this study provide further mechanistic insight into the proarrhythmic effects of psychological stress.

We explored this relationship because of existing evidence linking psychological stress with ventricular arrhythmias and sudden cardiac death.^{1–10} Epidemiologic and case-based studies suggest that acute forms of psychological stress are associated with a rise in heart attacks, sudden death, and other cardiac events in the general population.^{2–6} Studies in coronary artery disease patients have also confirmed the observation that mental stress is a potent trigger of myocardial ischemia in both the laboratory and the field.^{12–17} Mentally stressful tasks can provoke transient myocardial ischemia in a significant proportion (40–70%) of patients with CAD.^{13–17} 35 Several studies have shown that the development of myocardial ischemia in this setting predicts ischemia during daily life and is a poor prognostic factor linked to fatal and nonfatal cardiac events in this population.^{13–17} 35

This study provides further evidence for the detrimental effects of psychological stress on ventricular electrophysiological properties and the potential for increased susceptibility to arrhythmic events and sudden cardiac death under these influences. These observations are possibly caused by a direct effect of adrenergic stimulation on the cardiac ionic channels.^{18–21} However it is intriguing to consider the potential role of myocardial ischemia in this setting. In our analysis we observed that the effect of psychological stress on QTd was augmented by the presence of myocardial ischemia. Furthermore, we also observed a significant correlation between the severity of ischemia and the degree of increase in QTd. These findings suggest that ischemia mediated, at least partially, the effect of psychological stress on QTd.

The relationship between QTd and myocardial ischemia induced by other stressors such as exercise and atrial pacing has been documented.^{36–39} Ischemia alters the electrophysiologic properties of the myocardium.⁴⁰ Given that ischemia is in most instances a regional phenomenon, it creates repolarization heterogeneity across the myocardium which manifests as QTd and increased susceptibility for arrhythmic events. The findings of this study improves our understanding of the mechanistic pathways underlying these influences and may help in designing future therapeutic interventions for mental stress-related arrhythmic events.

Limitations

Measurement of the QTd is subject to inter-reader variability.^{22, 41} Various observers may have different definition for the end of the T wave, especially in the presence of a U wave and in situations where the T and P waves fuse. Various methods have been suggested to deal with this bias. In our protocol, whenever this situation occurred we used the method suggested by Day et al and Barr et al in which the QT interval was measured to the nadir between the T and the U waves.^{23, 27} Our QT interval measurements were done manually by a single observer who was blinded to ischemia status. Our protocol included a process, whereby a random sample of EKGs was analyzed by two independent observers and their measurements later correlated. There are some reports that manual measurements are as accurate as computer analysis in the hands of a careful observer.^{41, 42} Ischemia measurement using nuclear perfusion imaging is also subject to reader variability and may represent another confounding factor in this study.

Conclusion

Our study has demonstrated the detrimental effect of psychological stress on QTd, a measure of spatial heterogeneity of myocardial depolarization and a predictor of sudden cardiac death. We have also shown for the first time that this effect is significantly exacerbated by the concurrent development of myocardial ischemia in this setting. These findings provide mechanistic insight into the cardiovascular and proarrhythmic effects of psychological stress.

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Abbreviations list

CAD Coronary Artery Disease

QTd	QT dispersion
ECG	Electrocardiogram
CABG	Coronary Artery Bypass Graft surgery
PCI	Percutaneous Coronary Intervention
MI	Myocardial Infarction

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Figure 1.
The mean rest to stress increase in QTd in patients with and without mental stress-induced ischemia

Table 1

Demographic and clinical characteristics of study population

Variables	Frequency/Mean (n=70)
Mean age (years)	63±9
Gender (Males)	41 (59%)
Ethnicity (Caucasian)	56 (80%)
(African American)	10 (14%)
Previous MI	18 (26%)
Abnormal coronary angiogram	51 (73%)
Abnormal exercise or pharmacologic stress test	23 (33%)
Previous CABG	24 (34%)
Previous PCI*	19 (27%)
Smoking past or current	40 (57%)
Hypertension	48 (69%)
Diabetes	19 (27%)
History of angina	48 (69%)
Hyperlipidemia	64 (91%)
Beta Blockers	47 (67%)
ACE Inhibitors	37 (53%)
Calcium Channel Blockers	14 (20%)
Mean resting LVEF (%)	60±13
Mean Body Mass Index	30±7

Values expressed as either mean±standard deviation or frequency (%)

Abbreviations used: MI=myocardial infarction; CABG=Coronary Artery Bypass Graft; PCI=Percutaneous Coronary Intervention; ACE inhibitor=Angiotensin Converting Enzyme Inhibitor; LVEF=Left Ventricular Ejection Fraction.

Table 2

Comparison of QT dispersion and hemodynamic responses to mental stress

	Rest	Stress	P value
QT dispersion (all patients)	24±18	43±17	<0.001
QTd by MSIMI groups			
No Ischemia (n=44)	22±18	39±16	* p<0.001
MSIMI (n=24)	27±17	51±16	† p<0.001
SBP mm/Hg	120±18	162±26	<0.001
DBP mm/Hg	65±9	93±14	<0.001
HR beats/min	61±10	79±16	<0.001

Values expressed as mean±standard deviation.

QTd is expressed in ms

* P value for comparing the rest to stress increase in QTd among patients without MSIMI.

† P value for comparing the rest to stress increase in QTd among patients with MSIMI.

Abbreviations use: QTd=QT dispersion; MSIMI=Mental Stress-induced Myocardial Ischemia; SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; HR=Heart Rate.

Table 3

QT dispersion in patients with and without mental stress-induced ischemia

	Mean (95% CI)	P value
Stress QTd by MSIMI groups [‡]		
MSIMI (n=24)	50 (44–56)	* p=0.006
No Ischemia (n=44)	39 (35–44)	
Adjusted [†] stress QTd by MSIMI [‡]		
MSIMI (n=24)	49 (43–55)	† p=0.012
No Ischemia (n=44)	40 (36–44)	

QTd is expressed in ms

* comparing the stress QTd between MSIMI groups, controlling for the resting QTd.

[†] Controlling for the resting QTd, CAD severity factors (history of MI, CABG or PCI), co-morbid medical conditions (hypertension and diabetes) and cardiac medications (beta blockers and calcium channel blockers).[‡] Two patients were excluded due to poor imaging quality**Abbreviations use:** QTd=QT dispersion; MSIMI=Mental Stress-induced Myocardial Ischemia.