Depressed Mood Is Related to High-Frequency Heart Rate Variability During Stressors

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Objective: The purpose of this study was to examine the relationships between depressed mood and parasympathetic control of the heart in healthy men and women at rest and during two stressors. **Methods:** Fifty-three healthy college students completed a laboratory stress protocol that included a baseline resting period, a challenging speech task, and a forehead cold pressor task. Depressed mood was assessed using the Beck Depression Inventory (BDI). Parasympathetic cardiac control was measured as the high-frequency (0.12–0.40 Hz) component (HF) of heart rate variability using power spectrum analysis. Blood pressure, respiration rate, and respiration amplitude were measured simultaneously. **Results:** Participants were categorized as having a high or low depressed mood on the basis of median splits of their BDI scores. Those in the high depressed mood group had significantly greater reductions in HF during the speech task and significantly smaller increases in HF during the forehead cold pressor task than those in the low depressed mood group. Women had significantly greater reductions in HF during the speech task and smaller increases in HF during the forehead cold pressor task than men. However, gender and depressed mood did not interact to predict changes in HF. **Conclusions:** Depressed mood is related to the magnitude of decrease in parasympathetic cardiac control during stressors in healthy men and women. These findings extend those of previous studies, in which a similar phenomenon was observed among patients with cardiac disease. Because the participants in this study were healthy, the relationship between depressed mood and parasympathetic cardiac control does not seem to be secondary to cardiovascular disease. **Key words:** heart rate variability, parasympathetic tone, depression, cardiovascular mortality, stress, spectral analysis.

ANOVA = analysis of variance; ANS = autonomic nervous system; BDI = Beck Depression Inventory; BMI = body mass index; DBP = diastolic blood pressure; HF = high-frequency component of heart rate variability; HRV = heart rate variability; MI = myocardial infarction; MMPI-D = Minnesota Multiphasic Personality Inventory Depression scale; SBP = systolic blood pressure; STAI = Spielberger State-Trait Anxiety Inventory.

INTRODUCTION

Depression is an independent risk factor for mortality after MI (1–5). For example, in a prospective study, depression was a significant predictor of death due to cardiac disease and arrhythmic events 6 months after MI (5); this effect remained marginally significant after controlling for all other measured risk factors. Similarly, in a clinical trial of antiarrhythmic medications, depression predicted death and/or cardiac arrest 12 months after MI, after controlling for disease severity (1). Perhaps the strongest demonstration of this relationship is documented in a set of investigations in which depression consistently predicted mortality at 6, 12, and 18 months after MI independent of disease severity (2–4). Despite the relatively consistent findings for depression and cardiac mortality, the reasons for the association remain unexplained.

Several potential mechanisms linking depression and increased risk of mortality after MI have been proposed (6, 7). Some investigations have shown that depressed patients may have increased sympathetic and/or decreased parasympathetic nervous system functioning, implicating ANS dysfunction in post-MI risk. Altered ANS functioning can be a risk factor for secondary cardiac events among patients with coronary artery disease because of the increased probability of cardiac arrhythmia (8). Supporting this hypothesis is the observation that depression and premature ventricular contractions interact to predict disease risk (3). Specifically, this investigation noted the increased probability of death among mildly to moderately depressed patients exhibiting significant premature ventricular contractions.

Parasympathetic tone is an important factor in the development of cardiac arrhythmia. Among healthy individuals, parasympathetic tone helps to prevent ventricular arrhythmias by maintaining the electrical stability of the heart. When parasympathetic tone is decreased, cardiac arrhythmias are more likely to occur (9). For example, pharmacological blockade of parasympathetic influences on the heart greatly increases vulnerability to stress-induced arrhythmias in dogs (10). Alternatively, pharmacological stimulation of the parasympathetic nervous system reduces vulnerability to ventricular fibrillation in pigs (11).

Although stress decreases parasympathetic tone, under normal conditions enough parasympathetic tone remains during stress to prevent ventricular ar-

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Received August 16, 1999; revision received April 3, 2000.

0033-3174/00/6206-0796

rhythmias. However, when autonomic influences on the heart are compromised, such as by damage to neural fibers due to MI, extreme stress may provoke lethal arrhythmias and sudden death. Consistent with this reasoning, reduced HRV, an index partially determined by ANS functioning, has been associated with increased mortality after MI (12, 13). Therefore, after MI, depression may contribute to a potentially dangerous reduction in parasympathetic tone, which can predispose a patient to lethal arrhythmic events.

Several investigators have examined the relationship between depression and parasympathetic tone by measuring HRV among depressed patients with cardiac disease. Despite the fact that some studies have examined post-MI patients who are clinically depressed and others have tested cardiac patients with mild dysphoria or depressed mood, the results are consistent. Overall, the findings suggest an association between depression or depressed mood and reduced HRV in patients with coronary artery disease (14–16). For example, Carney et al. (15) found that clinically depressed patients had lower HRV than did nondepressed patients matched for age, sex, and smoking status. Similarly, Krittayaphong et al. (16) reported that patients with higher scores on the MMPI-D had higher heart rates and lower HRV than patients with lower depression scores.

One difficulty in interpreting these data is that reduced HRV is a frequent consequence of MI independent of depression or depressed mood (12, 17); thus, depression and reduced HRV occur simultaneously after MI but may not be causally related. Therefore, tests of the relationship between depression and HRV involving patients without cardiac disease may be particularly informative because they control for the effects of coronary heart disease on HRV.

A number of studies comparing resting HRV in groups of healthy depressed or mildly dysphoric participants and groups of nondepressed participants have recently been published. Of these, only one (18) reported that healthy women with higher depressed mood (MMPI-D) had lower resting HRV than did healthy women with lower depressed mood. None of the remaining studies of healthy populations have demonstrated an effect of clinical depression on resting HRV in healthy men and women (19–22). Thus, although there are some intriguing data suggesting that depression and/or dysphoria may be associated with parasympathetic tone even among healthy individuals, the differences at rest are not robust and therefore are not consistent across investigations.

More robust effects might be apparent during psychological stress, especially because stress has predictable effects on parasympathetic tone. One study examined HRV during mental stressors among dysphoric patients with cardiac disease. The results of this study indicated that during mild psychological provocation, patients with higher depression scores (MMPI-D) had more profound HRV changes than patients with lower depression scores, indicating greater reductions in parasympathetic cardiac control (23). Another study examined HRV during two psychological stressors among healthy dysphoric women. In contrast to the investigation of patients with cardiac disease, this study reported no differences in reactivity of HRV between the dysphoric and control groups (18). It is unclear whether the discrepant findings in patients with cardiac disease and healthy control subjects are due to patient status or gender because no study has investigated HRV in healthy depressed men during psychological stressors.

A portion of the discrepant findings may also be attributable to different methods of indexing parasympathetic tone. Many investigations estimated HRV using the standard deviation of all normal R-R intervals during Holter monitoring because of the ready availability of these data (15, 16). These measures of HRV are not optimally interpretable as indices of parasympathetic activity because they also reflect sympathetic and nonautonomic influences (24, 25). Although measurement and interpretation of HRV are somewhat controversial, it is generally accepted that HF (0.12–0.40 Hz), analyzed using spectral analysis, is a specific measure of parasympathetic cardiac control (24). A number of highly correlated measures of parasympathetically mediated HRV can be obtained using a variety of methods (24). For simplicity, HF is used throughout this article to refer to all of them. In addition, the interpretation of HF as reflecting parasympathetic cardiac control requires measurement of respiratory parameters, which have been shown to influence measures of HRV (26).

Together, these data point to the plausibility of a relationship between measures of parasympathetic control of the heart and depression or depressed mood. In concert with the data implicating psychological stress in cardiac arrhythmias and sudden death (27) and the data linking altered autonomic functioning to vulnerability to stress-induced arrhythmia (10), the investigation of depressed mood and HRV during stressors is warranted. However, to date there are no studies relating depression or depressed mood and HRV in healthy young men and women at rest and in response to stress tasks. The purpose of this study was to examine the relationship between depressed mood and HRV in a sample of patients without cardiac disease at rest and during two unique stressors using HF, a HRV measure that is specific to parasympathetic

influences. A secondary purpose was to extend previous research to include both men and women, given the recent suggestion of possible gender differences in the relationship between depressed mood and HRV (28). Finally, two stress tasks were chosen to obtain different patterns of cardiovascular response.

METHODS

Participants

Fifty-three college students (25 men and 28 women) with a mean age of 18.7 years (SD = 1.53 years, range = 17–27 years) participated in this study in exchange for partial course credit. All participants were healthy, nonsmokers, normotensive (<90 mm Hg resting DBP), and not taking medication affecting cholesterol or cardiovascular functioning. All participants were within 20% of their ideal body weight. Basic demographic information according to gender is reported in Table 1.

Physiological Measures

The electrocardiographic signal was sampled continuously at 500 Hz using three disposable spot electrodes placed in a three-lead configuration using the Minnesota impedance device (Instrumentation for Medicine, model 304B) and custom software. R-wave detection was performed using custom software that locates peaks and troughs using low-pass–filtered versions of the first and second derivatives of the electrocardiographic signal. Each minute of electrocardiographic data was visually inspected for errors in signal detection. After R-wave detection, an interbeat interval series was generated using a previously described algorithm (29). A heart period time series was created from the interbeat interval series using a "weighted" beat algorithm (30). This algorithm also detected sharp transitions in the heart period time series, which were removed by smoothing. These sharp transitions were removed because they do not have implications for HF and can distort fast Fourier transform power spectrum analyses. There were no differences between men and women or between those in the high and low depressed mood groups in the number of transitions removed. To remove low-frequency trends (including the DC component) from the input signal, a linear (first-order) polynomial was fit to, and subtracted from, the heart period time series (31). The heart period time series was then band-pass–filtered using an interpolated finite impulse response filter (32). The mean heart rate was calculated as 60,000 divided by the mean of the heart period time series. The power spectrum of the heart period time series was calculated using a fast Fourier transform, which decomposes the variance in the frequency domain (ms^2/Hz). The HF value was calculated as the natural log of the area under the power spectrum (ms^2) within the corner frequencies of the band-pass filter.

Respiratory amplitude signals were sampled continuously at 500 Hz using an EPM Systems respiration band (Slymar, CA). The effective sampling frequency of the signals was then reduced to 250 Hz for analysis. The respiratory signal was detrended linearly and band-pass–filtered using the same interpolated finite impulse response filter described above. The respiratory power spectrum was calculated using a fast Fourier transform and scaled to U^2/Hz. The mean respiratory rate and amplitude were calculated from the respiratory power spectrum using waveform moment analysis (33).

SBP and DBP were measured every minute using a Dinamap model 1846 oscillometric blood pressure monitor (Critikon Inc., Tampa, FL).

Body Composition Measures

Height (cm) and weight (kg) of each participant were measured at the beginning of the protocol using a standard balance beam scale. These measurements were used to calculate BMI.

Psychosocial Measures

Before physiological monitoring, all participants provided information about their demographic characteristics, health behaviors, general health status, depressed mood, and anxiety. The health behaviors and general health status measures consisted of a questionnaire designed to assess compliance with instructions regarding what substances and activities to avoid before participating (eg, caffeine and exercise) and a medical history questionnaire designed to screen for participants with high blood pressure or known cardiovascular disease. The purpose of the medical history questionnaire was to screen and exclude individuals with high blood pressure, elevated cholesterol level, diabetes, or any known chronic

TABLE 1. Demographic Information by Gender and BDI Median Split

Variable[a]	Men		Women	
	High	Low	High	Low
N	14	11	14	14
Ethnicity (%)				
White	18	11	17	19
African American	2	4	6	2
Other	6	6	4	6
Age (y)	18.4 ± 0.6	18.7 ± 1.4	18.4 ± 0.8	18.8 ± 2.5
Height (m)	1.76 ± 0.05	1.74 ± 0.06	1.64 ± 0.08	1.65 ± 0.09
Weight (kg)	74.9 ± 6.1	79.8 ± 12.7	63.8 ± 7.5	62.2 ± 7.5
BMI (kg/m^2)	24.3 ± 2.0	26.3 ± 3.7	23.7 ± 2.8	22.8 ± 3.5
BDI scores				
Values	12.2 ± 7.2	2.6 ± 1.7	10.6 ± 3.2	3.4 ± 2.0
Range	6–31	0–5	6–16	0–5

[a] Values for age, height, weight, BMI, and BDI scores are mean ± SD.

disease. Demographic characteristics are presented in Table 1. Depressed mood was measured using the BDI (34). State and trait anxiety were measured using the STAI (35).

Tasks

We used two separate stressors to elicit two distinct patterns of physiological reactivity (36). An impromptu videotaped speaking task was chosen because it generally elicits a reduction in HF (37), and a forehead cold pressor task was chosen because it generally elicits an increase in HF (38, 39). The forehead cold pressor task mimics the diving reflex to result in a reduction in heart rate mediated by the parasympathetic nervous system (38, 39).

The 5-minute speech task required participants to mentally prepare and then deliver a brief videotaped speech about a hypothetical situation. The situation described was one in which the participant had been falsely accused of shoplifting and had to defend himself or herself to a police officer in the mall's security office. The speeches were videotaped on a closed-circuit system, and participants were told that they would later be rated for poise, articulation, and appearance. The 3-minute forehead cold pressor task consisted of having each participant place a 1.5-liter ice bag filled with 800 ml of crushed ice and 200 ml of water on their forehead. The temperature of the ice water was 4°C. Participants sat upright during both tasks and were instructed to rest quietly without moving. They were taught how to apply the ice to their forehead in a standard way (with the face tilted slightly upward) so that their posture remained constant.

Procedure

Participants completed the laboratory protocol in a sound-attenuated laboratory. Written informed consent was obtained before the onset of the study, after which participants completed the psychosocial questionnaires. Instruments were then placed on participants for collection of electrocardiographic, blood pressure, and respiratory signals. After instrumentation, participants relaxed in a comfortable chair for 10 minutes to acclimate to the experimental environment. During the acclimation phase, they listened to music and completed questionnaires while their blood pressure was measured every minute. The acclimation period was followed by a 10-minute baseline period and then the two stress tasks, which were administered in a counterbalanced order. Each task was followed by a 10-minute recovery period, during which participants again listened to music and rested quietly. During each phase of the laboratory protocol, blood pressure measurements were taken each minute and electrocardiographic and respiration signals were recorded continuously. After completing the tasks and final rest period, participants were debriefed and excused.

Analytic Strategy

To increase reliability, each physiological measure was averaged over the last 5 minutes of the baseline period, every minute of the stress tasks, and the last 5 minutes of the recovery period. Groups were defined as high (High) or low (Low) in depressed mood on the basis of median splits of BDI scores (see Table 1).

Depressed mood and gender effects on initial baseline values of each physiological measure were tested using 2 × 2 ANOVAs (Depressed Mood [High vs. Low] by Gender [Women vs. Men]). Next, we wished to verify that the physiological parameters changed significantly from the initial baseline period to the stressor period. This was tested by a series of one-way ANOVAs on the repeated factor (Phase).

For each physiological parameter, reactivity scores were calculated by subtracting initial baseline values from task values. To test group differences in reactivity, a series of 2 × 2 × 2 ANOVAs (Gender [Women vs. Men] by Depressed Mood [Low vs. High] by Task [Speech vs. Cold Pressor]) on reactivity scores were calculated. All analyses involving HF were also performed using changes in respiration rate and amplitude as covariates.

For all analyses involving more than two levels of a repeated factor, probability values were adjusted for violations of the sphericity assumption using the Greenhouse-Geisser correction for degrees of freedom. Greenhouse-Geisser statistics are reported for each analysis as ϵ statistics. Main effects and interactions with a probability value of $<.05$ were considered statistically significant. All analyses were repeated with BMI as a covariate. In no cases did these analyses result in different findings. Post hoc tests were conducted using the Tukey Honestly Significant Difference test statistic (40).

RESULTS

Baseline Analyses

Results of the series of 2 × 2 ANOVAs (Depressed Mood by Gender) on the baseline values revealed significant main effects of Depressed Mood on SBP ($F(1,49) = 4.47$, $p < .05$). Inspection of the means indicated that high depressed mood participants had significantly higher SBP than did low depressed mood participants. There were no other significant main effects or interaction terms involving depressed mood (all F values < 2, all p values $> .16$).

As expected, several significant main effects were apparent for Gender. Specifically, there was a main effect of Gender on SBP ($F(1,49) = 11.57$, $p < .01$). Inspection of the means demonstrated that men (mean = 115.9 mm Hg) had significantly higher baseline SBP than women (mean = 103.2 mm Hg). There was also a trend for a Gender effect on baseline heart rate ($F(1,49) = 3.63$, $p = .06$). Inspection of the means illustrated that the source of this marginal effect was due to men (mean = 70.2 beats/min) having slightly lower baseline heart rate than women (mean = 75.2 beats/min).

No other main effects or interaction terms were observed for the analyses involving the baseline values. Mean baseline values of blood pressure, heart rate, and HF for the high and low depressed mood groups are presented in Table 2.

Phase Analyses

Results of the series of one-way ANOVAs revealed significant effects of phase on each physiologic variable (all values of $F(4,208) > 13$, all p values $< .0001$, all ϵ values ≥ 0.46). Subsequent inspection of the means and post hoc testing indicated that HF significantly decreased during the speech task, increased during the cold pressor task, and returned to near baseline values during the recovery periods (all p val-

TABLE 2. Baseline Values (mean ± SD) of Cardiovascular Measures by Gender and BDI Median Split

Variable	Men		Women	
	High	Low	High	Low
N	14	11	14	14
SBP (mm Hg)	119.7 ± 10.2	111.2 ± 23.2	106.0 ± 7.0	100.4 ± 9.2
DBP (mm Hg)	58.2 ± 5.2	53.8 ± 6.5	57.7 ± 5.9	57.4 ± 5.3
Heart rate (beats/min)	69.2 ± 8.6	71.5 ± 9.5	75.8 ± 6.5	74.6 ± 12.3
HRV [ln(ms^2)]	6.8 ± 0.7	6.1 ± 1.9	6.9 ± 0.5	6.7 ± 1.3

ues < .05). SBP and heart rate increased significantly during the speech task and returned to near baseline levels during the speech task recovery period, cold pressor task, and cold pressor task recovery period (all p values < .01). Post hoc tests also demonstrated that DBP increased during the speech and cold pressor tasks and returned to near baseline levels during the recovery periods (all p values < .01). Mean values of HF, blood pressure, and heart rate at each phase of the laboratory protocol by BDI median split are presented in Table 3.

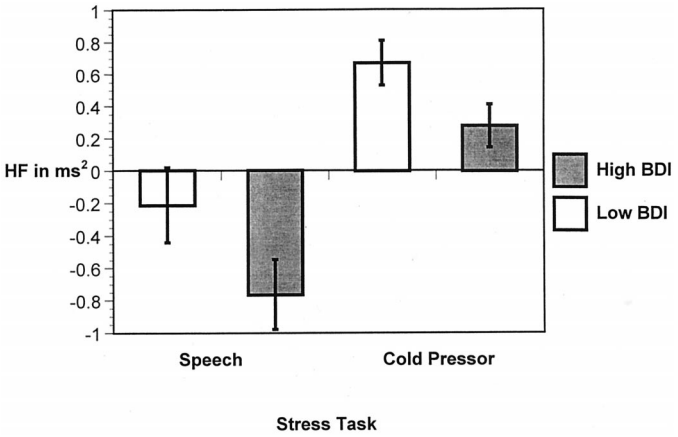

Fig. 1. Changes in HF (with standard errors) according to depressed mood group.

Reactivity Analyses

The change score analyses involving HF revealed a significant main effect for Depressed Mood ($F(1,49) = 6.43$, $p = .01$). Post hoc tests demonstrated that those high in depressed mood had significantly larger decreases in HF during the speech task and significantly smaller increases in HF during the cold pressor task (p values < .05) than did those low in depressed mood. Changes in HF according to depressed mood group are shown in Figure 1.

In addition, a significant main effect of Gender was also apparent for HF ($F(1,47) = 4.9$, $p = .031$). Subsequent inspection of the means indicated that women had larger decreases in HF during the speech task and smaller increases in HF during the cold pressor task than did men (p values < .05). Importantly, there was no significant interaction term involving gender, indicating that the relationship between HF and depressed mood is not accounted for by gender.

All analyses involving HF were also performed using respiration rate and amplitude as covariates. In no case were the results of these analyses substantially altered.

Because state and trait anxiety, as measured by the STAI, were significantly correlated with BDI scores, we repeated the change score analyses involving HF

TABLE 3. Mean Values (±SD) of Cardiovascular Measures at Each Phase by BDI Median Split

Variable	Baseline	Speech	Recovery	Pressor	Recovery
SBP (mm Hg)					
High BDI	112.8 ± 11.0	130.4 ± 16.0	112.9 ± 14.0	115.9 ± 12.2	111.1 ± 11
Low BDI	105.2 ± 17.3	121.2 ± 16.4	107.3 ± 15.1	111.0 ± 16.6	103.5 ± 16
DBP (mm Hg)					
High BDI	58.0 ± 5.4	72.5 ± 14.7	56.8 ± 13.2	62.8 ± 12.7	57.3 ± 12
Low BDI	55.8 ± 6.0	67.8 ± 7.4	55.9 ± 6.2	59.9 ± 7.2	54.5 ± 5.8
Heart rate (beats/min)					
High BDI	72.5 ± 8.2	88.8 ± 13.8	73.4 ± 9.6	73.4 ± 10.2	72.8 ± 8.9
Low BDI	73.2 ± 11.0	86.3 ± 15.4	74.8 ± 9.5	72.2 ± 8.5	71.0 ± 9.4
Heart rate variability [ln(ms^2)]					
High BDI	6.9 ± 0.6	6.1 ± 0.9	6.8 ± 0.7	7.1 ± 0.9	6.8 ± 0.6
Low BDI	6.4 ± 1.6	6.2 ± 1.1	6.2 ± 1.3	6.8 ± 1.5	6.5 ± 1.4

using state and trait anxiety as covariates. In no case were the results altered, indicating that the relationship between depressed mood and changes in HF was not a function of state or trait anxiety. In addition, a series of 2 × 2 × 2 ANOVAs (Anxiety Score Median Split by Gender by Task) on HF change scores revealed no main effects for state or trait anxiety.

A series of 2 × 2 × 2 ANOVAs (Depressed Mood by Gender by Task) on heart rate change scores revealed a main effect of Depressed Mood ($F(1,49) = 5.27$, $p = .03$). Inspection of the means indicated that those in the high depressed mood group had greater increases in heart rate during stressors than those in the low depressed mood group. In addition, there was a main effect of Gender on heart rate change scores ($F(1,49) = 5.54$, $p = .02$). Inspection of the means indicated that the source of the effect was due to women having larger increases during the speech task and smaller reductions during the cold pressor task than men. No other effects involving the cardiovascular reactivity variables were significant.

DISCUSSION

In this investigation, we found that participants with higher scores on the BDI had significantly different patterns of HF in response to stressors than participants with lower BDI scores. Specifically, individuals with higher depressed mood exhibited greater decreases in HF to the speech task and smaller increases in HF to the cold pressor task than individuals low in depressed mood. The results of this study suggest that mildly depressed but otherwise healthy individuals experience altered parasympathetic responses to stressors. These findings extend the results of previous investigations of parasympathetic stress responses among patients with coronary artery disease and depressed mood (23) to healthy men and women. Because the men and women in the current study were young and healthy, the results suggest that altered ANS activity during stressors in depressed individuals is not a function of underlying cardiac disease.

Altered HF during stressors may be particularly relevant to the theoretical model relating depressed mood and HRV to post-MI mortality. Dogs susceptible to ventricular fibrillation have greater HF decreases during exercise than nonsusceptible dogs (41). In addition, vagal reflexes, as measured by baroreceptor reflexes, predict susceptibility to ventricular fibrillation during myocardial ischemia in animals (42). Although speculative, to the extent that stressors elicit dangerous decreases in parasympathetic control of the heart, the finding that depressed mood is related to the magnitude of stressor-related reductions in HF may help to explain the relationship between depression and increased risk of mortality after MI (3).

In the current study, there were no significant baseline differences in HF between the high and low depressed mood groups. This finding is consistent with the majority of previous research among healthy individuals (19–22), although a relationship between depressed mood and baseline HF among healthy women has been reported (18).

In contrast to finding no group differences at baseline, most investigations of patients with coronary artery disease have reported a relationship between depression or depressed mood and 24-hour measures of HRV (14–16). More relevant to the current study, however, are the two investigations of HRV during psychological stressors among depressed individuals. Although the investigation of healthy women indicated no task-associated changes in HRV in women with higher depressed mood compared with those with lower depressed mood (18), a study among post-MI patients indicated that patients with higher depressed mood had greater reductions in parasympathetic cardiac control (23). The inconsistent findings may be due to the effects of heart disease on HRV, different methodologies for estimating HRV, or the difficulty in interpreting measures of HRV for between-subjects comparisons (25).

There were no gender differences in HF at baseline in our study, a finding that is consistent with the results reported for two other recent studies (43, 44). A main effect of gender on HF changes during stressors was observed in the current study, but there was no interaction of gender and depressed mood. The lack of an interaction of gender and depressed mood in this study stands in contrast to a previous study (28), which reported gender differences in the relationship between depressed mood and HF. Specifically, the results of this earlier study indicated a positive relationship between depressed mood as measured by the BDI and several measures of HRV in women but a negative relationship in men. However, Light et al. (45) reported the reverse relationship between HRV and depressed mood for women. When these findings are considered together, it is clear that more research on the nature of the gender difference in HF is necessary to clarify the inconsistencies in the literature.

There are several potential limitations of this study. The sample size was relatively small, and a larger sample would have provided greater power to illustrate effects and would have allowed us to test potential nonlinear relationships. However, the fact that this study found a statistically significant relationship between depressed mood and HF during stressors suggests that the effect size is robust. Another potential

limitation is that the range of BDI scores was somewhat restricted, and the mean BDI score in the high depressed mood group indicated that these participants had depressed mood rather than major depressive disorder. However, in one prospective study of depression and post-MI mortality, the optimal point at which to dichotomize BDI scores was shown to be only 10 (3). In addition, in the Cardiac Arrhythmia Pilot Study (1), the mean BDI score for patients who died or experienced cardiac arrest was only 12.15. In the current study, a significant relationship between depressed mood and HF changes was detected despite the relatively low mean BDI scores. It is intriguing to speculate that a stronger relationship may be present with higher BDI scores. Women in this study were not tested in a consistent phase of the menstrual cycle. Although it is possible that menstrual cycle phase can sometimes alter mood in susceptible women, it is unlikely that there were influences of phase on HF because a recent investigation directly tested this question and found no effects (46). A final limitation is that respiratory amplitude measures were not calibrated for each subject and therefore represented arbitrary units. However, respiratory rate has been shown to be more important than amplitude in determining HF values (24), and uncalibrated respiration amplitudes provide reasonable statistical control for individual differences in respiratory amplitude changes during the laboratory protocol (47).

CONCLUSION

Depressed mood was related to the magnitude of changes in parasympathetic cardiac control during stressors in healthy men and women, extending the findings of a similar study of patients with cardiac disease. To the extent that stressors elicit dangerous decreases in parasympathetic control of the heart, this finding may help to explain the relationship between depressed mood and increased risk of mortality after MI.

This work was supported in part by an Ohio State University fellowship (to J.W.H.) and by the National Institutes of Health (Grant HL48363 to C.M.S. and Grant MO1-RR00034 to the Ohio State University General Clinical Research Center). The authors thank the study participants and Dr. Gary Berntson for technical guidance.

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