Psychophysiology, 40 (2003), 1–6. Blackwell Publishing Inc. Printed in the USA.

Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals

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Abstract

Respiratory sinus arrhythmia (RSA) is an index of the vagal control of heart rate that is associated with emotion regulatory capacity. To examine RSA in depressed and nondepressed participants in the context of an emotion-regulatory challenge, we presented a sad film to induce crying, a behavior associated with heightened parasympathetic activation. We predicted that nondepressed persons who cried would show elevations in RSA during the onset and the resolution of crying. By contrast, we predicted that depressed individuals who cried would fail to exhibit increased RSA over the course of their crying episodes. As hypothesized, nondepressed participants exhibited RSA increases that accompanied the resolution of tearful crying, consistent with a homeostatic function for crying, whereas depressed subjects who cried did not exhibit increased RSA. Results suggest that the physiological self-regulatory mechanisms invoked by crying are compromised in depression.

Descriptors: Respiratory sinus arrhythmia, Crying, Major Depressive Disorder, Emotion regulation, Homeostasis

Major Depressive Disorder (MDD) is characterized by persistent sad mood or a loss of interest or pleasure in daily activities, as well as by several associated symptoms, such as weight loss, sleep disturbance, fatigue, and feelings of guilt (American Psychiatric Association, 1994). Depression has increasingly been conceptualized as a disorder of emotion dysregulation (Gross & Muñoz, 1995). It is not clear, however, precisely which aspects of emotion regulation are disordered in depression. One promising construct in this regard is respiratory sinus arrhythmia (RSA), a noninvasive measure of the vagal control of heart rate. RSA is a dynamic parameter that has been linked to a variety of self-regulatory processes (e.g., Porges, 1995). In several studies, depressed individuals have exhibited lower resting levels of RSA than normal controls (e.g., Rechlin, Weis, Spitzer, & Kaschka, 1994), but there are also a number of published null results in this literature (e.g., Moser et al., 1998). It is possible that an examination of contexts where RSA fluctuates would produce a clearer pattern of compromised RSA functioning in depressed individuals. To address this issue, we examined changes in RSA levels among depressed and nondepressed individuals before, during, and after exposure to a sad film.

The authors express their appreciation to Adrine Biuckians, David Hardisty, Megan McCarthy, Sadia Najmi, Kate Reilly, and Miguel Sanchez for their help in conducting this study. This research was supported by National Institute of Mental Health (NIMH) Grant MH59259 awarded to Ian H. Gotlib and, in part, by MH56094 (FW), MH58147 (JG), and an NIMH predoctoral fellowship in affective science awarded to Jonathan Rottenberg.

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Resiratory Sinus Arrhythmia and Emotion Regulation

The parasympathetic nervous system is concerned with growth and restorative functions (i.e., facilitating digestion, slowing heart rate; cf. Berntson et al., 1997). Parasympathetic functioning is often monitored via the outflow of a branch of the vagus nerve that regulates the chronotropic control of the heart via nucleus ambiguus efferent projections to the sino-atrial node. Vagal influence on the heart may be quantified noninvasively by measuring respiratory sinus arrhythmia (RSA), the rhythmic increase and decrease in heart period that coincides with the respiratory frequency. Whereas the heart period decrease is associated with phases of inspiration when respiratory mechanisms in the brainstem attenuate the vagal efferent action on the heart, the heart period increase is associated with phases of expiration when the vagal efferent influence to the heart is reinstated.

A number of investigators have related compromised parasympathetic functioning to greater stress vulnerability and lowered emotion regulatory capacity. For example, Porges (1995, 1997) and others have highlighted the role of brainstem areas such as the nucleus ambiguus in regulating behavioral and physiological reactivity to stress. Porges (1995) contends that a

reduction in RSA reflects deficient regulatory capacity over both attentional and emotional systems. For example, infants who exhibited deficient modulation of RSA were found to exhibit behavioral problems as preschoolers (Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). In older children, low RSA has also been related to both internalizing and externalizing psychopathology (Pine et al., 1998).

RSA and Major Depressive Disorder

Findings concerning the nature of the association between RSA and clinical depression are mixed. For example, several investigators have found that depressed patients have lower RSA than do nondepressed controls (e.g., Carney et al., 1995; Rechlin et al., 1994). Consistent with these findings, there is also evidence that successful treatment of depression is associated with increases in RSA (e.g., Balogh, Fitzpatrick, Hendricks, & Paige, 1993; but see also Schultz, Anderson, & van de Borne, 1997). In contrast, however, Moser et al. (1998) found that although depressed subjects exhibited higher heart rates than did nondepressed controls, they were not characterized by lower RSA. In fact, other researchers have also reported no differences in RSA between depressed subjects and nondepressed controls under baseline conditions (e.g., Lehofer et al., 1997; Yeragani, Pohl, Balon, & Ramesh, 1991).

One possible explanation for the conflicting findings regarding RSA in depression involves the concept of fluctuation in RSA. It is now well established that RSA levels fluctuate with changes in psychological and behavioral states (e.g., Grossman & Svebak, 1987). Indeed, it appears that the magnitude of fluctuation in RSA levels may provide an important index of difficulties in self-regulation. For instance, anxious individuals have been found to exhibit less change in RSA than do their nonanxious counterparts during and after emotion challenges (e.g., Cohen et al., 2000; Lyonfields, Borkovec, & Thayer, 1995), suggesting that anxiety is accompanied by deficits in autonomic flexibility. RSA modulation has not yet been examined in depressed persons, and it is possible that clearer evidence of compromised RSA functioning in depression would be obtained by probing RSA when strong emotions are evoked.

The Present Study

In the present study, we elicited tearful crying in depressed and nondepressed participants and examined the course of RSA changes associated with this behavior. Several factors motivated the selection of tearful crying for study. First, crying is well known to be a potent signal of organismic distress (Darwin, 1873). Indeed, theorists have argued that crying functions to alleviate distress by motivating the self to action (Tomkins, 1963) or by motivating others to engage in prosocial behaviors (Cornelius, 1997). Second, excessive crying in depressed persons has been noted in clinical contexts (cf. Beck, Rush, Shaw, & Emery, 1979) and may be reflective of the high levels of distress that are seen in this disorder. The Diagnostic and Statistical Manual for Mental Disorders (DSM-IV; American Psychiatric Association, 1994) also includes crying behavior as a characteristic of depressed persons, though other observers have noted that severely depressed individuals may not exhibit this pattern (e.g., Patel, 1993). Our third motivation to examine crying is that this behavior is known to be associated with changes in physiological functioning. For example, crying is associated with increased activation of the sympathetic branch of the autonomic nervous system (Gross, Frederickson, & Levenson, 1994). Importantly, the onset of psychogenic crying is parasympathetically mediated. More specifically, the innervation of the lacrimal gland indicates that parasympathetic fibers of the seventh cranial nerve provide a principal stimulus for the reflex secretion of tears (Werb, 1983). Although it has been assumed that crying also leads to changes in systemic parasympathetic activation (Gross et al., 1994), the nature, precise timing, and the detectability of these changes at target sites such as the sino-atrial node of the heart are all unknown.

Whether and how crying raises systemic parasympathetic tone is also an important theoretical question in light of psychological theories that have considered crying behavior to be functional and beneficial in reducing tension (e.g., Efran & Spangler, 1979; Sadoff, 1966; for a review, see Cornelius, 1997). To date, laboratory measurements of the acute physiological consequences of crying have been inconsistent with the view that crying performs cathartic or homeostatic functions (e.g., electrodermal activity during crying is increased; Kraemer & Hastrup, 1988). It is worth noting, however, that investigators have not measured the physiological *aftereffects* of crying, and it is possible that the physiological sequalae of crying episodes might reveal evidence of homeostatic functions for crying. Of course, RSA measures are especially important in this regard, given the strong conceptual relationship between RSA and organismic homeostasis.

The present study was designed to examine RSA changes that accompany crying in depressed and nondepressed individuals. Crying was induced by the presentation of a sad film, and the fluctuations in RSA that accompanied the onset and the resolution of crying episodes were measured. We addressed two questions in this study: (1) Do nondepressed individuals exhibit increased parasympathetic activation coincident with the onset and/or resolution of crying episodes? (2) Is evidence of compromised parasympathetic functioning observed when crying is provoked in depressed individuals? We predicted that: (a) crying among nondepressed participants would be accompanied by increases in RSA during the film and postfilm periods; and (b) crying among depressed participants would not be associated with increases in RSA for either period.

Method

Participants

Twenty-five depressed and 31 nondepressed nonpsychiatric control participants took part in the study. All depressed participants met DSM-IVcriteria for Major Depressive Disorder using the Structured Clinical Interview for DSM-IV Axis I (SCID-I; First, Spitzer, Gibbons, & Williams, 1995). All participants were female, fluent English speakers, and were between the ages of 18 and 60 (depressed age: $M = 31.7$, $SD = 10.2$; nondepressed age: $M = 33.5$, $SD = 11.0$; $t(54) < 1$). None of the depressed participants was taking psychotropic medications at the time of testing. All participants provided written informed consent and were paid $25 per hour. Nondepressed participants were interviewed using the same general and medical criteria that were used for the depressed participants. In addition, they were interviewed to exclude those with lifetime diagnoses of any Axis-I disorder.

Film Stimuli

The cry-eliciting film was 170 s in length and depicted a boy who was distraught at the death of his father (Gross & Levenson,

1995). A second film was used to provide a neutral baseline period. This film lasted 60 s and depicted coastal landscape scenery.

Equipment

Films and instructions were presented on a 20-in. television monitor at a viewing distance of 1.75 m. An SA Instruments 12-channel bioamplifier was used to condition physiological responses. Signals were sampled at 400 Hz using a Data Translation 3001 PCI 12-bit 16-channel analog to digital converter.

Measures

Behavioral. A remotely controlled camera positioned behind darkened glass unobtrusively videotaped participants. Crying was judged to be present when three independent raters agreed that a participant displayed visible tears in at least one eye. In addition, crying duration was coded by recording the time of cry onset (when tear presence began) and cry offset (when observable tears were not present). Reliabilities for cry onset and cry offset were $\alpha = .71$ and $\alpha = .70$, respectively.

Physiological. (1) An electrocardiogram was recorded using Beckman miniature electrodes, placed in a bipolar configuration on opposite sides of the participant's chest. The heart period (HP) was calculated as the interval (in milliseconds) between successive R waves. (2) Two channels of respiration were measured with inductive plethysmography bands (Ambulatory Monitoring, Ardsley, NY) placed around the chest and abdomen. Calibration against fixed volume bags was accomplished by the least-squares method. Respiratory rate and tidal volume were calculated breath-by-breath using customized programs.

Procedure

Participants were assessed individually. They were greeted and were then positioned in a comfortable chair facing a video monitor in a quiet, well-furnished laboratory room and connected to physiological monitoring devices. The viewing of the sad film was preceded by a number of experimental tasks not reported here. Immediately prior to viewing the sad film, participants viewed the 60-s neutral film. Participants were simply instructed to watch each film carefully. The sad film was followed by a 90-s postfilm period, during which participants were instructed to sit comfortably.

Data Reduction

A customized computer program written in MATLAB (Wilhelm, Grossman, & Roth, 1999) was used for computation of RSA. The beat-by-beat values of HP were edited for outliers due to artifacts or ectopic myocardial activity, linearly interpolated, and converted into instantaneous time series with a resolution of 4 Hz. HP time series were linearly detrended, and the power spectral densities derived for each period using the Welch algorithm, which ensemble averages successive periodograms (overlapping 256-point segments were Hanning windowed and subjected to fast Fourier transform, and estimates of power were adjusted to account for attenuation produced by the Hanning window). RSA was computed by summing power spectral density values over the frequency band associated with respiration (0.15–0.50 Hz), and resulting values were normalized using the natural logarithm. Period averages for physiological measures were computed for three epochs: baseline, sad film, and postfilm.

Results

Statistical Analysis

The primary between-subjects variables were depression status (depressed, nondepressed) and cry status (criers, noncriers). The within-subject variable was time (baseline, film, postfilm). Our primary analysis was a three-way repeated-measures analysis of variance (ANOVA) conducted on RSA. In this analysis, a significant interaction of depression status, cry status, and time would indicate that the parasympathetic changes induced by depressed and nondepressed crying were divergent in their course. Where appropriate, p values were corrected for nonsphericity using the Greenhouse–Geisser ε.

Crying Behavior

Crying behavior was elicited in 20 participants (36%). Of those who cried, 8 were nondepressed (26% of this group), and 12 were depressed (48% of this group). The two groups of criers cried for a similar amount of time in both the film and postfilm periods (sad-film period: 93.6 s depressed vs. 101.3 s nondepressed, $t(18) < 1$; postfilm period: 45.3 s depressed vs. 42.8 s nondepressed, $t(18) < 1$). In all 20 cases, crying was judged to have resolved before the end of the postfilm period.

RSA

The three-way (Depression Status × Cry Status × Time) repeated measures ANOVA conducted on RSA yielded a significant main effect only for time, $F(2,51) = 11.33$, $p < .001$, $\varepsilon = .883$. This main effect was subsumed, however, by the predicted three-way interaction of cry status, depression status, and time, $F(2,51) = 4.16$, $p < .05$, $\varepsilon = .883$. To examine this interaction, we conducted separate two-way ANOVAs (Cry Status × Time) for the depressed and the nondepressed participants. The interactions of cry status and time for the depressed and nondepressed participants are plotted in Figure 1. This interaction was significant for the nondepressed participants, $F(2,28) = 7.65$, $p < .005$, $\varepsilon = .929$, but not for the depressed participants, $F(2,22) < 1$, indicating that the effects of crying on RSA varied as a function of time only for the nondepressed participants.

To examine this significant two-way interaction for the nondepressed participants, we conducted one-way ANOVAs (by time) separately for nondepressed criers and noncriers. The effect for time was significant for nondepressed criers, $F(2,6) = 12.65$, $p < .005$, $\varepsilon = .739$, but not for nondepressed noncriers, $F(2,19) = 2.64$, $p > .05$, $\varepsilon = .967$. Follow-up paired t tests indicated that this significant effect for time was due to the nondepressed criers exhibiting higher levels of RSA in the postfilm period than in both the sad-film, $t(7) = 4.12$, $p < .005$, and the baseline, $t(7) = 3.67$, $p < .01$, periods, which did not differ significantly from each other, $t(7) < 1$. In fact, this pattern was observed in all 8 nondepressed criers. In sum, consistent with both hypotheses, compared to their noncrying counterparts, nondepressed participants who cried exhibited increased RSA coincident with the resolution of crying, whereas crying had no impact on RSA among depressed individuals.

The Physiological Context of RSA Differences

To understand the broader physiological context of the RSA effects observed among the nondepressed participants, we conducted analyses on heart rate, respiration rate, and tidal volume. Means and standard deviations for these data, broken down by cry status and time are depicted in Table 1 (depressed

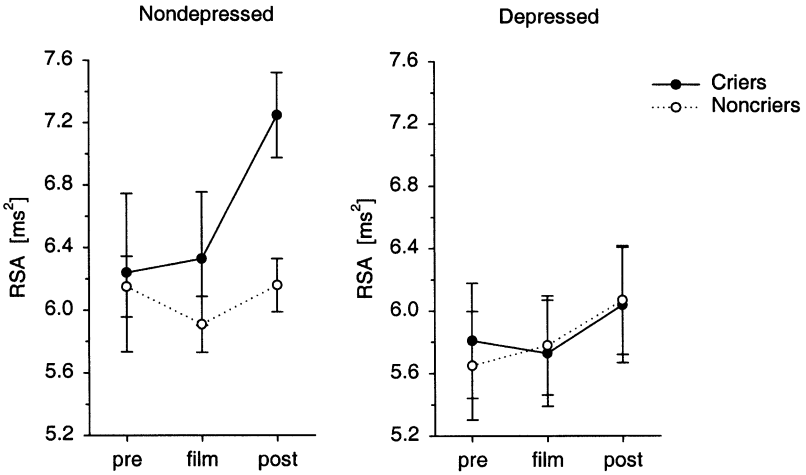

Figure 1. RSA means and standard errors for each experimental epoch, broken down by diagnosis and cry group.

participants are included in the table for comparative purposes).[1] Because crying onset has been associated with heart rate acceleration in previous work (Gross et al., 1994), we wished to examine whether crying among the nondepressed participants in response to a sad film led to cardiac acceleration in the present study. Relatedly, we wished to examine whether heart rate acceleration among the nondepressed criers—presumably due to increased sympathetic drive to the heart—was reversed during resolution of crying, as would be expected if parasympathetic activation overrode sympathetic influences on the heart (Berntson et al., 1997). To examine heart rate reactivity, we computed change scores for the film and postfilm epochs by subtracting prefilm from film and postfilm heart rate values. We then contrasted the heart rate reactivity of the nondepressed criers with the reactivity of the nondepressed noncriers.

As expected, during the sad film, criers exhibited greater cardiac acceleration than did noncriers (criers: $M = 2.51$, $SD = 3.50$; noncriers: $M = -1.90$, $SD = 2.35$; $t(29) = 4.03$, $p < .001$). The two groups no longer differed during the postfilm period, $t(29) = 1.33$, $p > .05$, a pattern consistent with a vagal braking of heart rate among participants who cried. It is important to note, however, that criers did not exhibit cardiac deceleration during the postfilm period in an absolute sense; in fact, criers had higher heart rates during the postfilm period than they did at baseline (mean increase = 3.42, $t(7) = 4.48$, $p < .005$). This evidence of cardiac acceleration among the criers suggests that crying led to strong sympathetic drive to the heart during both the film and postfilm periods, which was only partially counteracted by increased vagal outflow to the heart in the postfilm period.

Analysis of respiratory parameters is also important to a more complete understanding of our RSA effects. It is well known that RSA is affected by within-subject/between-task variation in respiratory rate and tidal volume (e.g., Grossman, Karemaker, & Wieling, 1991). For example, crying has been found to be associated with respiratory changes, such as slowed respiration rate (Gross et al., 1994), that might independently lead to increases in RSA. Consequently, it was critical that we examine whether changes in respiratory parameters might account for the increased RSA during crying resolution among the nondepressed participants. A two-way (Cry Status × Time Epoch) repeated measures ANOVA conducted on respiratory rate yielded a main effect for time epoch, $F(2,27) = 20.68$, $p < .001$, $\varepsilon = .964$, which was qualified by a significant interaction of cry status and time, $F(2,27) = 3.61$, $p < .05$, $\varepsilon = 0.964$. Follow-up one-way ANOVAs by cry status conducted separately for the three time epochs indicated that crying was associated with a tendency towards slower respiratory rates during the sad film, $F(1,28) = 3.49$, $p = .07$, but not during the other epochs (prefilm period: $F(1,28) < 1$; postfilm period: $F(1,28) = 1.65$, $p > .15$). A similar analysis of tidal volume for these participants yielded only a main effect for time epoch, $F(2,27) = 19.02$, $p < .001$, $\varepsilon = .941$; neither the main effect for cry status $F(1,28) = 3.47$, $p > .05$, nor the Cry Status × Time interaction, $F(2,27) = 1.68$, $p > .05$, $\varepsilon = .941$, was significant. Paired t tests conducted to examine the effect for time revealed that the tidal volumes in each experimental epoch differed significantly from one another (all ps $< .05$), with the largest tidal volumes occurring during the postfilm period, followed by the baseline period tidal volumes, followed by the film-period tidal volumes. Finally, RSA analyses were repeated using respiratory rate and tidal volume as changing covariates. The pattern of results was unaffected, indicating that the elevated levels of RSA among nondepressed criers were mediated vagally, and were not simply respiratory artifacts.

Discussion

Based on previous literature indicating abnormalities in RSA among depressed individuals, and on work suggesting that diminished self-regulatory capacity may be related to a lack of RSA responding when strong emotions are invoked, we hypothesized that the elicitation of tearful crying would reveal evidence of compromised parasympathetic functioning among depressed individuals. As we predicted, whereas tearful crying was associated with significant increases in RSA among nondepressed participants, crying had no impact on RSA among depressed participants. In fact, these differences in RSA functioning between depressed and nondepressed persons emerged only

[1]Exploratory repeated measures ANOVAs yielded no main effects or interactions involving diagnostic group for any of these three variables.

Table 1. *Means (SD) of Physiological Measures by Diagnosis and Cry Group*

	Nondepressed criers ($n = 8$)	Nondepressed noncriers ($n = 23$)	Depressed criers ($n = 12$)	Depressed noncriers ($n = 13$)
RSA				
Prefilm	6.24 (1.43)	6.15 (0.93)	5.81 (1.27)	5.65 (1.25)
Film	6.33 (1.28)	5.91 (0.87)	5.73 (1.17)	5.78 (1.14)
Postfilm	7.25 (0.77)	6.16 (0.81)	6.04 (1.29)	6.07 (1.26)
Heart rate				
Prefilm	75.57 (11.29)	74.89 (9.49)	81.14 (9.72)	74.18 (10.02)
Film	78.09 (12.27)	72.98 (8.64)	80.72 (13.66)	72.70 (9.89)
Postfilm	79.00 (10.93)	76.73 (10.17)	83.14 (11.85)	76.17 (10.41)
Respiration rate				
Prefilm	13.88 (2.52)	13.84 (2.66)	13.09 (2.78)	15.67 (3.66)
Film	15.19 (2.01)	17.57 (3.16)	14.50 (1.68)	18.20 (2.41)
Postfilm	13.28 (1.97)	14.51 (2.28)	12.93 (1.99)	14.63 (3.00)
Tidal volume				
Prefilm	392.72 (141.89)	342.01 (140.69)	427.73 (184.39)	416.28 (205.71)
Film	400.13 (106.71)	284.44 (130.27)	367.69 (168.44)	349.68 (131.82)
Postfilm	531.27 (162.02)	409.97 (119.84)	515.20 (258.91)	494.68 (157.76)

Notes: RSA: Respiratory sinus arrhythmia [Logarithm of high frequency heart period power/ms^2]; Heart rate [beats/minute]; Respiratory rate [breaths/minute]; Tidal volume [ml].

when crying was elicited—they were not observable during an emotionally neutral baseline period (e.g., Moser et al., 1998).

The data described in this report represent the first empirical evidence that tearful crying in nondepressed, psychiatrically healthy individuals is associated with parasympathetic activation that extends beyond the site of the lacrimal gland to reach target organs such as the heart. We anticipated that nondepressed individuals would exhibit increased systemic parasympathetic tone both during and after crying. Surprisingly, increases in parasympathetic tone were observed only with the resolution of crying episodes. There are several possible reasons why we obtained this temporal pattern of findings. First, it is possible that we did not have sufficient statistical power to detect increased parasympathetic tone during the film period among nondepressed individuals who cried. We should note, however, that there is little indication of increased RSA for nondepressed criers during the film period (see Table 1). Moreover, despite the relatively small sample of nondepressed criers, our statistical power was adequate to detect postfilm period differences in RSA.

A second explanation for the temporal pattern of findings involves the role of attentional factors in the modulation of RSA. Findings from previous studies indicate that deployment of attention may be associated with vagal withdrawal (e.g., Mulder & Mulder, 1981; Suess, Porges, & Plude, 1994). If nondepressed participants cried as a function of their greater attentional deployment toward the sad film, vagal withdrawal associated with this deployment may have masked crying-induced increases in RSA until the removal of the sad film stimulus. To rule out this explanation in future work, it would be useful to use a cry-eliciting paradigm that involved less dramatic shifts in attentional deployment (e.g., shift from sad to neutral mental imagery).

Finally, a third explanation for why a delay in RSA activation was observed in the nondepressed criers is that crying is a part of a homeostatic mechanism that, once engaged, invokes a series of physiological changes. Indeed, consistent with prior work, crying onset was associated with heart rate acceleration (Gross et al., 1994). During the postfilm period, however, partial neutralization of crying-related arousal appears to have occurred. Criers did not continue to exhibit cardiac acceleration in the postfilm period relative to their noncrying counterparts, suggesting that higher vagal outflow partially neutralized the accelerative effects of sympathetic drive on heart rate (Berntson et al., 1997). Moreover, post-sad-film RSA levels in nondepressed criers were not only higher than levels during the sad film, but were higher than levels before the sad film, a pattern of rebound that suggests the operation of an active homeostatic mechanism.

Clinically depressed individuals failed to exhibit increased RSA during crying resolution. This finding has implications for both psychological and physical health. Within an emotion-regulation framework, this pattern of responding may reflect a compromise of self-regulatory mechanisms that facilitate recovery from negative emotion in depression. From a physical health standpoint, high RSA has been shown to protect patients diagnosed with coronary artery disease from exaggerated cardiovascular responses to psychological stress (Grossman, Watkins, Wilhelm, Manolakis, & Lown, 1996). Conversely, and consistent with a buffering effect of RSA, the low RSA observed in individuals diagnosed with MDD may mediate the increased risk for cardiac mortality and morbidity seen in this disorder (e.g., Carney et al., 1995; reviewed in Musselman, Evans, & Nemeroff, 1998). One interpretation of the present RSA findings is that crying provides cardiovascular benefits to nondepressed persons that are unavailable to depressed individuals. To comment more conclusively concerning the health consequences of crying in these subject groups, a longer-term assessment of RSA after crying will be needed. Nevertheless, it is noteworthy that the salutary effects of crying observed in the present study are consistent with findings that indicate that verbal and nonverbal disclosure of emotion are associated with improved subsequent health outcomes (e.g., Pennebaker, Barger, & Tiebout, 1989). To extend these findings concerning the possible benefits of crying in future work, it will be critical to measure concurrent changes in psychological state and to compare crying with other forms of negative emotion expression.

It is important to acknowledge two limitations of the current study. First, because we tested females, caution is warranted in generalizing our findings to males. Replication in male samples is also an important future direction in light of recent work that has reported gender differences in RSA among individuals experiencing depressive symptoms (Thayer, Smith, Rossy, Sollers, & Friedman, 1998). Second, in examining RSA differences between depressed and nondepressed participants, we controlled for tidal

volume and respiratory rate differences, but not for possible differences in central respiratory drive. Because central respiratory drive may also have an independent effect on RSA (Kawahara & Yamauchi, 1990), measurement of this central drive (e.g., end-tidal partial pressure of CO_2) is strongly recommended. Although more work is required to extend these encouraging findings and to delineate more precisely the parasympathetic regulatory abnormalities that appear to characterize clinically depressed individuals, the present finding that depressed persons exhibited evidence of parasympathetic compromise only when crying responses were elicited and not during an emotionally neutral task underscores the utility of using emotional challenges to probe RSA functioning in depression.

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(Received December 4, 2000; Accepted October 15, 2001)