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BRIEF REPORT

Can the Distress-Signal and Arousal-Reduction Views of Crying Be Reconciled? Evidence From the Cardiovascular System

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Theorists have staked out two ostensibly opposing views of human crying as either an arousing behavior that signals distress or a soothing behavior that reduces arousal after distress. The present study examined whether these views of crying might be reconciled by attending to physiological changes that unfold over crying episodes. Sixty female students watched neutral and cry-eliciting films while autonomic physiology, including respiratory sinus arrhythmia and pre-ejection period, was assessed. Crying participants exhibited heart rate increases that rapidly subsided after crying onset. Crying onset was also associated with increases in respiratory sinus arrhythmia and slowed breathing. All crying effects subsided by 4 minutes after onset. It is possible that crying is both an arousing distress signal and a means to restore psychological and physiological balance, depending on how and when this complex behavior is interrogated.

Keywords: crying, autonomic arousal, heart rate, respiratory sinus arrhythmia

Crying is a very common and universal form of human emotional expression. Although no other species has the ability to shed emotional tears, humans of all ages and from all cultures cry on certain occasions to express their emotions (Vingerhoets & Cornelius, 2001; Vingerhoets, Cornelius, Van Heck, & Becht, 2000). Despite the obvious significance of crying, scientific understanding of this behavior is at an early phase.

Historically, theorists have advanced apparently opposing claims about human crying as either (1) an arousing behavior that signals distress or (2) a soothing behavior that reduces arousal after distress (Gross, Fredrickson, & Levenson, 1994; Vingerhoets et al., 2000). According to the first view, crying functions to communicate the need for help and to stimulate others to provide comfort and social support (Frijda, 1997; Kottler, 1996; Nelson, 2000). This distress-signal view predicts that crying is a costly signal associated with increased physiological activation (Gross et al., 1994). According to the second view, crying is a soothing behavior that reduces organismic distress and arousal. This arousal-reduction view posits that crying facilitates the recovery of psychological and physiological homeostasis after distress in a crying individual (Bindra, 1972; Efran & Spangler, 1979).

Importantly, emotion scientists have generally overlooked the possibility that these two views of crying are, in fact, reconcilable. Crying is a response that invokes a cascade of psychobiological changes that is not well understood (Vingerhoets et al., 2000). Whether observed crying effects are consistent with the distress-signal or arousal-reduction view may depend critically on how and when one probes this behavior. Here we put forward one novel idea for reconciling these positions involving the timing of crying-related changes in the autonomic nervous system: During a crying episode, early changes might correspond with the distress-signal view (e.g., cardiac acceleration), whereas later changes might correspond with the arousal-reduction view (e.g., cardiac deceleration).

Past work offers limited guidance on whether distress-signal and arousal-reduction views could be reconciled in this manner. Most empirical studies have examined the short-term autonomic correlates of crying within a sad-film viewing paradigm (Gross et al., 1994; Kraemer & Hastrup, 1988; Rottenberg, Gross, Wilhelm, Najmi, & Gotlib, 2002; Rottenberg, Wilhelm, Gross, & Gotlib, 2003; Sakuragi, Sugiyama, & Takeuchi, 2002). Results to date have generally found short-term psychobiological changes consistent with the distress-signal view (e.g., reports of distress, heart rate acceleration; Gross et al., 1994; Kraemer & Hastrup, 1988; Rottenberg et al., 2002). Only Rottenberg and colleagues demonstrated findings consistent with the arousal-reduction view. Criers

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The present study has been approved by the medical ethical committee of the TweeSteden Hospital in Tilburg, the Netherlands.

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exhibited higher levels of respiratory sinus arrhythmia, a parasympathetic parameter theoretically linked to homeostasis (Rottenberg et al., 2003; but also see Sakuragi et al., 2002). Consistent with the idea that crying may restore physiological balance only as crying episodes wear on, Rottenberg et al. found elevated respiratory sinus arrhythmia only late in their recording protocol. In general, the course of crying effects has been little studied. In fact, to our knowledge, the duration of crying effects has never been assessed.

The Present Study

Our primary goal was to examine whether a reconciliation of the distress-signal and arousal-reduction views of crying might be possible by examining the nature and duration of crying effects on autonomic physiology. To achieve this goal, we sought to overcome three methodological challenges. First, crying in the laboratory has a low base rate ($\sim$ 20%) when sad films are used as stimuli. This low base rate raises questions concerning whether past work reveals the physiology of highly cry prone individuals rather than the physiology of crying per se. To increase the base rate of crying, we used a longer, more emotionally complex film than those previously used in past studies. Second, prior work has not obtained sound physiological indicators from both branches of the autonomic nervous system. Pre-ejection period and respiratory sinus arrhythmia were measured here to permit disentangling of sympathetic and parasympathetic influences on cardiovascular activity (e.g., De Geus & Van Doornen, 1996). Moreover, measures of parasympathetic functioning are particularly important for assessing the arousal-reduction hypothesis, especially in view of theorized links between parasympathetic activation and tear production (Van Haeringen, 2001). Third, the timing of key moments during crying episodes has often been imprecise. In the current study, participants pressed a button when they began to cry. This button press defined crying onset, which was used to define preonset, onset, and postonset periods for each crier.

Method

Participants

Sixty female psychology students from Tilburg University participated (age range 18–32; $M = 20.4$, $SD = 2.8$). Participants had no chronic physical or psychiatric illnesses, were not pregnant, and did not use medications other than hormonal contraceptives. Forty participants received course credit and 20 participants received a financial reward (30 euro) for study participation. All participants provided signed informed consent.

Film Stimuli

A National-Geographic documentary lasting 15 minutes (Nixon, 1994) was used as neutral film to provide an assessment of baseline physiology. "Once were warriors" (OWW) was selected as the emotional, cry-eliciting film (Scholes & Tamahori, 1994). The original OWW was edited for poignancy to create a 70-min version. OWW depicts the life of a Maori family in New Zealand that is tyrannized by the father and contains dramatic scenes of extreme violence, rape, and suicide.

Ascertainment of Crying Onset

Crying was defined to participants as all tearful behavior, such as tears welling up in the eyes, as well as tears running out of the eyes and sobbing. Participants were instructed to press a button at each onset of tears during their viewing of OWW. This button press denoted crying onset for each crier. Self and observer measures of crying have shown excellent correspondence in past work (Choti, Marston, Holston, & Hart, 1987; Kraemer & Hastrup, 1988; Marston, Hart, Hileman, & Faunce, 1984).

Physiological Assessment

Equipment. The Ambulatory Monitoring System device (VU-AMS, Vrije Universiteit, Amsterdam) was used to record the electrocardiograph (ECG) and impedance cardiograph (ICG) signals for determining respiration rate, respiratory sinus arrhythmia and pre-ejection period (Klaver, De Geus, & De Vries, 1994). The ECG signal, the blood-pressure signal, and electrodermal activity were digitized by a 12-bit AD-converter at 1000 Hz and stored for later off-line processing.

Heart rate (HR). The ECG signal was measured using three disposable pregelled Ag/AgCl electrodes. A customized computer-program identified R-waves and calculated inter-beat-intervals (IBIs). IBIs were transformed into HR in beats per minute.

Diastolic (DBP) and systolic blood pressure (SBP). The blood-pressure signal was obtained with the Portapres device (Model-2, Finapres Medical Systems BV, Arnhem, the Netherlands), which provides a continuous measurement of finger arterial blood pressure on a beat-to-beat basis (for calculation methods see Peñáz, 1973; Wesseling, De Wit, Van der Hoeven, Van Goudoever, & Settels, 1995). DBP and SBP were derived for each heartbeat as the minimum and maximum blood pressure, respectively. DBP and SBP were lacking for two persons due to equipment failure.

Skin conductance level (SCL). Electrodermal activity was recorded using a constant voltage (.5 V) coupler with two Ag/AgCl electrodes (contact area of 8 mm^2) placed on the right hand. The SCL was set to zero before starting the recording for each participant. Due to equipment failure, SCL was absent for six participants.

Respiration rate (RR), respiratory sinus arrhythmia (RSA), and pre-ejection period (PEP). By means of six disposable pregelled Ag/AgCl electrodes, ECG and ICG signals were recorded (see De Geus & Van Doornen (1996) for more details). The AMS device samples DZ/dt values for 512 ms around each R-wave. The PEP (in ms) was defined as the interval from the R-wave peak to the B-point, minus a fixed Q-R interval of 48 ms (Sherwood et al., 1990; Willemsen, De Geus, Klaver, Van Doornen, & Carroll, 1996). The starting points of inspiration and expiration were used to compute RR in breaths per minute. Using the respiratory intervals, RSA was computed for each breath using the peak-to-trough method (De Geus & Van Doornen, 1996; Grossman, Van Beek, & Wientjes, 1990). As a result of equipment failure, RR, RSA, and PEP were missing for five individuals. RR and RSA could not be computed for two participants due to a noisy respiratory signal, and RSA was absent for one individual because of deviant IBIs.

Procedure

Participants were tested individually. After connecting monitoring devices, participants viewed neutral and negative films. The neutral film was shown before the negative film in order to familiarize participants with the experimental setting and to provide a baseline period for physiological recording. Participants were instructed to press a button every time they cried during the negative film. Mood was measured before and after each film. Eighteen mood indicators were rated on a Likert scale varying from 1 (*not at all*) to 10 (*very much*). Positive indicators included relaxed, happy, relieved, under control, and cheerful. Negative indicators included powerless, pitiful, disgust, sad, astonished, angry, guilty, tense, fearful, restless, bad tempered, touched, and nervous.

Data Reduction and Analysis

Automatic scoring was verified by visual inspection and signals were screened for artifacts and outliers. The VU-AMS software package extracted RR, RSA, and PEP (De Geus & Van Doornen, 1996) and computed ensemble averages over 30-s periods. The twelfth minute of the neutral film was used to define baseline values and three continuous periods during the film OWW were used to define preonset, onset, and postonset periods. To better isolate the effects of crying onset, the period defined by the button press was kept as short as possible, more precisely, a 30-s period containing one ensemble average. To evaluate the short-term effects of crying, preonset, and postonset epochs were defined as 60-s periods before and after the onset period, respectively. When a respondent pressed the button more than once, only the first crying episode was selected for analysis. Individuals who did not cry were randomly matched to crying counterparts. Using the timing of the button press of the crying participants, the physiological responses of the noncrying participants were partitioned into three time periods equivalent to those of the crying participant with whom they had been matched. Primary analyses compared crying and noncrying participants over the preonset, onset, and postonset periods. Secondary analyses explored additional minutes of post onset data to ascertain the full duration of any crying effects.

Results

Elicitation of Crying

Twenty-seven participants reported crying during OWW. The most common scene to precipitate crying depicted the mother of the Maori family finding her daughter dead by suicide.

Manipulation Check: Reported Mood

Average scores were calculated for positive mood and negative mood reported before and after the film OWW. GLM repeated measure analyses were performed with Time Period as a within-subjects factor and Crying as a between-subjects factor. For positive mood, no significant effects were observed, Fs < 3.30, ps $> .08$, *partial* η^2s $< .06$. For negative mood, there was a significant effect of Time Period, $F = 220.97$, $p < .001$, *partial* $\eta^2 = .82$. Consistent with the intended manipulation, higher negative-mood scores were reported after ($M = 4.16$, $SD = 1.38$) than before the film OWW ($M = 2.36$, $SD = 1.05$). No other effects were observed, all Fs < 3.79, ps $> .06$, *partial* η^2s $< .07$. Thus, both crying and noncrying participants reported increased negative mood to the OWW film.

Baseline Physiology

Both a multivariate analysis on all the physiological variables and univariate analyses on each physiological variable separately revealed that crying and noncrying individuals were physiologically indistinguishable during baseline, all Fs < 1.

Acute Effects of Crying

A GLM repeated measure multivariate analysis on the physiological variables was performed with Time Period as a within-subjects factor and Crying as a between-subjects factor. This analysis revealed a trend for Crying to influence physiological responses, $F(1, 37) = 2.91$, $p < .10$, *partial* $\eta^2 = .07$, and a main effect of Time Period, $F(3, 35) = 12.03$, $p < .001$, *partial* $\eta^2 = 0.51$. Importantly, these main effects were qualified by a significant Time Period × Crying interaction, $F(3, 35) = 3.45$, $p < .05$, *partial* $\eta^2 = .23$, indicating that crying altered the trajectory of autonomic responding.

To decompose this interaction, GLM repeated measures univariate analyses were conducted for each physiological variable with Time Period as a within-subjects factor and Crying as a between-subjects factor. Table 1 presents the means and standard deviations for all variables and time periods for noncrying and crying participants.

Blood pressure, skin conductance level and pre-ejection period. For DBP, SBP, SCL and PEP the only significant effect was Time Period, all Fs ≥ 7.36, ps $< .01$, *partial* η^2s $> .25$. Because the time periods were defined idiographically by each crier, effects of Time Period are not easily interpreted.

Heart rate. Significant effects were observed for Time Period, $F(3, 50) = 5.25$, $p < .05$, *partial* $\eta^2 = .24$, and Time Period × Crying, $F(3, 50) = 3.11$, $p < .05$, *partial* $\eta^2 = .16$. To decompose this interaction, separate repeated measures analyses on HR for each group with Time Period as a within-subjects factor revealed that Time Period effect was only significant for crying participants, $F(3, 24) = 4.77$, $p < .05$, *partial* $\eta^2 = .37$. Post hoc analyses of the Time-Period effect in crying participants revealed that HR accelerated over baseline during the preonset period and onset period, both $F(1, 26)$s > 5.44, ps $< .05$, *partial* η^2s $> .17$. In addition, HR was higher during the onset of crying than during the preonset and postonset period, both $F(1, 26)$s > 5.12, ps $< .05$, *partial* η^2s $> .16$ (see Figure 1a).

Respiration rate. Significant effects were obtained for Time Period, $F(3, 43) = 4.46$, $p < .01$, *partial* $\eta^2 = .24$, and Time Period × Crying, $F(3, 43) = 3.30$, $p < .05$, *partial* $\eta^2 = .19$. To decompose this interaction, follow-up analyses revealed that the effect of Time Period on respiration rate was only significant for crying participants, $F(3, 21) = 6.43$, $p < .01$, *partial* $\eta^2 = .48$. Relative to baseline and the preonset period, crying individuals exhibited lower levels of RR during the onset period and postonset period, all $F(1, 23)$s > 6.66, ps $< .05$, *partial* η^2s $> .22$ (see Figure 1b).

Table 1
Mean Scores on the Physiological Variables (With Standard Deviations in Parentheses) for Non-Crying Participants and Crying Participants

	Baseline		Pre-onset		Onset		Post-onset	
Variable	NC	C	NC	C	NC	C	NC	C
HR	70.0 (9.3)	67.7 (8.3)	70.0 (8.9)	71.5 (8.4)	69.9 (8.9)	73.6 (9.2)	68.9 (8.3)	69.8 (7.4)
DBP	52.7 (13.3)	52.6 (9.3)	64.6 (11.4)	67.6 (10.5)	64.8 (11.4)	69.0 (10.9)	64.7 (11.4)	69.3 (11.6)
SBP	118.6 (15.4)	115.3 (13.2)	133.4 (17.0)	137.8 (17.1)	132.9 (16.2)	139.2 (19.1)	134.2 (15.7)	139.4 (19.5)
SCL	−1.2 (3.3)	−1.0 (4.7)	3.6 (4.8)	5.0 (5.2)	3.8 (4.5)	5.5 (5.0)	3.6 (4.3)	5.5 (4.9)
RR	17.1 (2.1)	16.6 (2.1)	16.9 (2.3)	16.6 (2.9)	16.9 (2.7)	15.1 (2.0)	16.8 (2.1)	15.0 (2.4)
RSA	89.3 (56.0)	97.7 (45.7)	83.9 (70.9)	93.4 (33.3)	87.5 (67.2)	123.8 (50.0)	87.7 (58.2)	120.4 (71.4)
PEP	104.3 (23.3)	105.8 (18.1)	101.7 (22.6)	105.0 (17.7)	102.8 (22.7)	106.5 (18.6)	103.5 (22.8)	106.5 (18.7)

Note. NC = noncrying participants; C = crying participants; HR = heart rate (beats/min); DBP = diastolic blood pressure (mmHg); SBP = systolic blood pressure (mmHg); SCL = skin conductance level (μS); RR = respiration rate (breaths/min); RSA = respiratory sinus arrhythmia (ms); PEP = pre-ejection period (ms).

Respiratory sinus arrhythmia. RSA was significantly influenced by Time Period, $F(3, 41) = 3.84$, $p < .05$, *partial* $\eta^2 = .22$. Time Period × Crying interaction for RSA approached conventional levels of significance $F(3, 41) = 2.82$, $p < .10$, *partial* $\eta^2 = .17$. Because of the high theoretical significance of this effect, we proceeded with post hoc follow up analyses within each group, which again revealed that the Time Period effect was significant only for crying participants, $F(3, 19) = 3.55$, $p < .05$, *partial* $\eta^2 = .36$. Crying participants exhibited higher RSA during the onset period than during baseline, $F(1, 21) = 5.15$, $p < .05$, *partial* $\eta^2 = .20$. RSA was also higher during both the onset and postonset periods than the preonset period, both $F(1, 21)$s > 4.32, ps $< .05$, *partial* η^2s $> .18$ (see Figure 1c).

To attempt to isolate the source of increased RSA during crying, we examined whether slowed breathing (decreased RR) among criers might account for the increased RSA. MANOVA repeated measures analyses were performed on RSA with Time Period as a within-subjects factor and RR as a changing covariate. After including RR in the model, the main effect of Time Period on RSA became nonsignificant, $F(3, 62) = 1.35$, $p = .27$. It is thus possible that increased RSA in crying participants might not be centrally mediated and might be due at least in part to slowed breathing (see Allen, Chambers, & Towers, 2007, for interpretative caveats).

Duration of Crying Effects

To ascertain the full duration of the observed crying effects, we conducted exploratory analyses of minute-by-minute postonset HR, RR, and RSA, using return to baseline as the duration criterion. HR increases over baseline were observed in criers in the preonset and onset periods. HR decreased to baseline levels 1-minute postcrying onset, $F(1, 26) = 2.19$, $p = .15$, *partial* $\eta^2 = .08$, and HR remained at baseline levels for subsequent minutes (Figure 1a). RR was lower than baseline during onset and 1-minute postcrying onset (see above). RR still tended to be lower than baseline during the second and third minutes postcrying onset, $F(1, 23)$s > 3.26, ps $< .10$, *partial* η^2s $> .12$. By the fourth minute postcrying onset, RR had returned to baseline, $F(1, 23) = 1.99$, $p = .17$, *partial* $\eta^2 = .08$ (Figure 1b). Finally, RSA was higher during crying onset than during baseline and tended to remain higher than baseline during the first minute postcrying onset, $F(1, 21) = 3.59$, $p < .10$, *partial* $\eta^2 = .15$. RSA normalized during the second minute postcrying onset, $F(1, 21) = 0.06$, $p = .82$, *partial* $\eta^2 = .00$, and remained at baseline levels for subsequent minutes (Figure 1c). In sum, the duration of crying effects was longest for respiration rate, shortest for heart rate, with respiratory sinus arrhythmia intermediate. All traces of the crying episode had disappeared by the fourth minute post onset.

Discussion

Theorists have offered conflicting views of crying's effects upon the body. The primary goal of this study was to examine whether a reconciliation of the distress-signal and arousal-reduction views of crying might be possible by examining the nature and duration of crying effects on autonomic physiology. In doing so, we attempted a comprehensive assessment of the effects of crying on ANS function, including respiratory sinus arrhythmia and pre-ejection period, to adequately index the parasympathetic and sympathetic influences on cardiovascular functioning, respectively.

Consistent with the first part of the proposed reconciliation—that "arousal" effects are observed early in a crying episode—heart rate acceleration was observed early in the course of crying episodes, in a pre-onset period before participants indicated they were crying. Although findings concerning heart rate acceleration among criers replicated past work suggesting that crying is associated with initial cardiovascular activation (Gross et al., 1994; Kraemer & Hastrup, 1988; Rottenberg et al., 2002, 2003), our other electrodermal and cardiovascular measures adduced surprisingly limited evidence for the distress-signal view of crying, and did not replicate past results showing short-term activation on these measures. Moreover, as the first study to measure pre-ejection period during crying, we did not find evidence of increased sympathetic drive to the heart among criers.

Therefore, before proceeding further, one puzzle to consider is why this study did not find broader evidence of sympathetic activation during crying as past studies have. One possibility concerns major differences in the cry-eliciting stimuli between this and previous studies. Past studies have used sad stimuli, which are suited to examining the special case of crying while

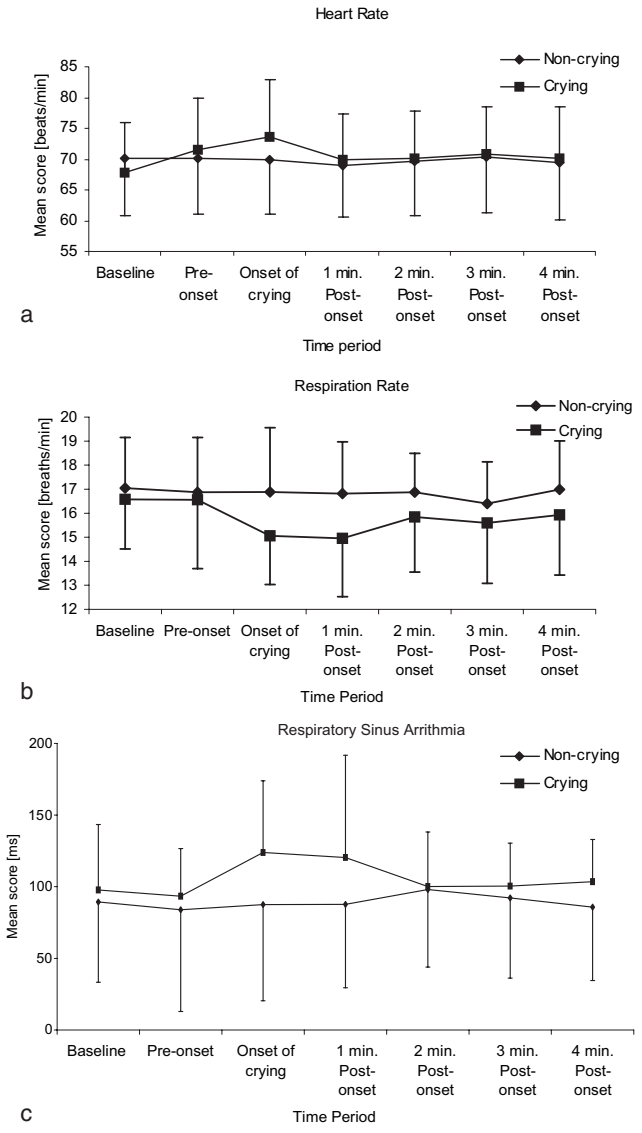

Figure 1. Means (*SD*) of heart rate (a), respiration rate (b), and respiratory sinus arrhythmia (c), as a function of cry status.

sad. However, it is well known that crying in real life is typically accompanied by multiple emotions (Vingerhoets, Boelhouwer, Van Tilburg, & Van Heck, 2001; Vingerhoets et al., 2000). To enhance ecological validity and improve upon the low base rate of experimental crying, we employed a cry-eliciting film that evoked a blend of negative emotions, including anger and disgust, rather than a discrete sad state. The stimulus was longer and more heterogeneous than the sadness stimuli used in prior studies. Consistent with the intended potency of our stimulus, we obtained higher rates of crying than previous studies (~50% vs. 20%). Table 1 reveals that the film elicited substantial increases over baseline across participants on several physiological measures (e.g., blood pressure, skin conductance level). Detection of any differential sympathetic activation specifically associated with crying may depend on the background emotional state of the crier.

The second part of the proposed reconciliation—that arousal reduction is observed as crying episodes wear on—was also partially supported. Crying was accompanied by persistent increases in respiratory sinus arrhythmia (Rottenberg et al., 2003) and decreases in respiration rate (Gross et al., 1994). The robust association of crying with slowed respiration rate supports the idea that crying sets in motion self-soothing behaviors. The brevity of the cardiac acceleration that was observed was also consistent with the idea that parasympathetic activation during crying may counteract initial cardiovascular activation. Finally, duration analyses indicated that crying-related changes were relatively fleeting, lasting less than four minutes. Although these analyses were exploratory in nature, the brevity of crying-related changes may be seen as supportive of the arousal reduction view.

Two caveats are needed concerning these interpretations. First, covariate analyses with respiratory rate suggested that the source of parasympathetic activation was ambiguous. It might have been mediated by respiratory influences, rather than centrally. Second, it should be pointed out that while heart rate decreased rapidly after the onset of crying, it did not decrease to below baseline levels, as would be the case if true parasympathetic dominance prevailed.

Three limitations of our study suggest specific needs for future research efforts. First, in addition to clarifying the source of parasympathetic activation, our study did not isolate the exact temporal sequencing of crying and parasympathetic activation. Does crying induce parasympathetic activation (Rottenberg et al., 2003) or does parasympathetic activation induce crying (Bindra, 1972; Efran & Spangler, 1979; Gross et al., 1994)? Development of heart rate variability metrics that afford valid second-by-second measurements (Allen, Chambers, & Towers, 2007) would be useful for clarifying which changes in parasympathetic activity just precede and which changes just follow the onset of crying. Second, this study relied upon a categorical classification of tearfulness, which may be less sensitive to crying effects than a continuous measure of crying intensity would be. Development of ophthalmologic measures (e.g., noninvasive measure of tear outflow) would be useful for quantifying crying intensity in future work. A third limitation of our study and the literature more generally (Gross et al., 1994; Kraemer & Hastrup, 1988; Rottenberg et al., 2003; Sakuragi et al., 2002) was a failure to ascertain the offset of crying. In our defense, no method is currently available to reliably and precisely measure the crying offset. Consequently, in the present context, it is ambiguous whether effects in postonset crying epochs reflect the effects of continued crying or the aftereffects of crying. Developing methods to measure crying offset will clearly be important for further elucidating the trajectory of crying effects.

Is crying an arousing distress signal or a means to restore psychological balance? Until now, there has been surprisingly little consideration of the possibility that the two major views of crying are complementary, rather than mutually exclusive. The specific form of our proposed reconciliation—that crying may invoke initial physiological activation (distress signal) followed by a rapid decay (arousal reduction)—received modest support in the data. Nevertheless, we believe our study has considerable heuristic value in guiding further research efforts. Future work

is likely to reveal that crying may be both an arousing distress signal and a means to restore psychological and physiological balance, depending on how and when this complex behavior is interrogated.

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