CRYING AND VAGAL RECOVERY DURING BEREAVEMENT

by

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A Thesis Submitted to the Faculty of the

DEPARTMENT OF PSYCHOLOGY

In Partial Fulfillment of the Requirements

For the Degree of

MASTER OF ARTS

In the Graduate College

THE UNIVERSITY OF ARIZONA

2022

THE UNIVERSITY OF ARIZONA
GRADUATE COLLEGE

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Final approval and acceptance of this thesis is contingent upon the candidate's submission of the final copies of the thesis to the Graduate College.

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Abstract:

Background: Crying is a universal behavior seen around birth, around death, and many moments in between. While there is much literature on crying in infants and young children, there is little research investigating the function of crying in adults and specifically during bereavement. The current study aimed to assess if an individual's grief severity moderated the relationship between crying and vagal control. We also sought to replicate findings that depression moderated the relationship between crying and vagal control.

Methods: The current study analyzes data from the Emotion Regulation Diary Study (ERDS). In ERDs, participants underwent a video recorded, grief recall interview during an electrocardiogram. Fifty-nine participants from ERDS qualified for final analyses of crying behavior. Raters coded the grief recall interviews for length of crying (none, 5 minutes, 10 minutes).

Results: There was not significant moderation between crying and vagal control by grief severity nor depression. A significant main effect was found for depression on vagal control following the grief recall interview and accounting for baseline vagal control, however grief severity did not show a significant main effect.

Conclusions: We did not support our hypotheses that grief and depression would moderate the relationships between crying and vagal control. A noteworthy finding emerged showing a main effect of depression on vagal control.

Introduction

Crying is the first behavior humans do upon birth. Prior work on the psychological aspects of crying have considered the evolutionary benefits to crying, the social implications of crying, questions about crying as an attachment behavior (Nelson, 2005; Provine, 2012), and whether crying is physiologically and psychologically beneficial (Gračanin et al., 2014; Rottenberg et al., 2008). Vingerhoets and Bylsma (2016) hypothesized that crying is a form of communication meant to "facilitate social bonding, to elicit sympathy and empathy, to promote cooperative and helpful behavior, and, probably, the inhibition of aggression in assaulters."

Several lines of work have suggested that crying probably had evolutionary advantages, and that it has a function for both attachment and social bonding. "Crying is part of a system of biologically based, inborn attachment behaviors that helps to establish and maintain the parent–child bond" (Nelson, 2005). In humans, crying begins as a means for survival. In infants, and perhaps adults too, crying is a crucial tool in communication. It is a way the infant indicates a need for their caregiver, which in turn provokes physical contact with the caregiver. When a baby cries the mother tends to their survival needs such as food, warmth, and emotional support. During this time in infancy, when the caregiver responds to a baby's cry with nurturing, the baby develops a sense of safety and security and ultimately an attachment.

As cited in Nelson (2005), Bowlby (1960) described the emotional reaction full of loud crying and bodily shaking when a child 6 months of age or older is left by their caregiver as a sign of protest. The child is protesting the separation and working towards a reunion. However, if the caregiver does not return, the child's initial "protest" changed to "despair." Bowlby noticed a change in the characteristics of the child's behavior – from violent crying to an occasional wail. A child might become quieter and more withdrawn, and occasionally become teary. During

bereavement, one might experience protest or despair crying, similar to the separation from an attachment figure during development. In the present study, we aim to characterize the types of crying present during a laboratory based semi-structured interview designed to evoke feelings of grief in bereaved individuals.

Vagal Recovery and Crying

When we cry, we may become red in the face, take more frequent and shallower breaths, and if the crying episode is strong enough, we might perceive an increase in heart rate. These are indicators of activation of the sympathetic nervous system (SNS) (Kraemer & Hastrup, 1988). Activation of the SNS is often referred to the body's "fight or flight" response. Our heart rate and respiration rate increase as our bodies respond to potential danger. While the SNS plays an important role for survival, the parasympathetic nervous system (PNS) is also crucial for autonomic regulation. The PNS is responsible for slowing respiration rate and heart rate and restoring the body to a calm state. The PNS regulates rest, digestion, and growth (Berntson et al., 1997). Part of the PNS is the vagal complex which is comprised of vagal reactivity and vagal recovery (Gregson & Ketring, n.d.)

Parasympathetic functioning is monitored via an efferent branch of the vagus nerve that regulates the chronotropic control of the heart via nucleus ambiguus efferent projections to the sino-atrial node (Rottenberg et al., 2003). One way to measure vagal response is by measuring respiratory sinus arrhythmia (RSA), which is the wavelike rise and fall in heart period that corresponds with respiration. During phases of inspiration, mechanisms in the brainstem decrease the vagal efferent action on the heart. During phases of expiration, the vagal efferent stimulus to the heart is restored.

Rottenberg and colleagues (Rottenberg et al., 2003) have investigated parasympathetic responses associated with crying. Rottenberg (2003) looked at the effects of crying on vagal recovery in depressed and non-depressed individuals. They monitored RSA for 60 seconds during a baseline period, 170 seconds as participants watched a sad film, and 90 seconds after the film during which time participants were asked to sit comfortably. Twenty-six percent of the nondepressed group cried and 48% of the depressed group cried. Non-depressed criers had a sizeable increase in parasympathetic activity during the post-film period. Non-depressed non-criers showed no significant change in their parasympathetic activity in the post-film compared to their baseline status. Rottenberg and colleagues found that all depressed individuals (criers and non-criers) showed the same pattern as non-depressed non-criers. These findings suggest that the normal functioning of crying might be self-regulatory and depression appears to decrease parasympathetic activity following crying.

Vagal Recovery, Depression, and Grief

Individuals suffering from depression, or major depressive disorder (MDD) typically have difficulty in most aspects of their lives. They might have poor sleep, struggle with unplanned weight fluctuations, be uninterested in things they used to enjoy, have difficulty keeping up with various activities of daily living such as basic hygiene, and appear with a flat affect (American Psychiatric Association, 2013). Individuals with MDD have also been noted to have lower RSA compared to healthy controls (Koch et al, 2019) and to have blunted reactivity to laboratory stressors (Schiweck et al., 2019) compared to health controls.

Depression is often conflated with grief, and while they have many similarities, there are some differences (Shear et al., 2011; Prigerson et al., 2021). One of the main differences between

grief and depression is that individuals who are grieving experience a yearning for their lost loved one, while those who have depression do not. Another key symptomatic difference is that grief comes in waves, while depression is more global. Bereaved individuals "get a break" from their pangs of grief, while depressed individuals do not often have periods of euthymia.

On a cardiovascular level, bereaved individuals have been shown to have lower baseline RSA compared to non-bereaved individuals (Fagundes et al., 2018; Buckley et al., 2012; O'Connor et al., 2002). In addition, a study done by LeBlanc and colleagues (2016) found evidence demonstrating blunted vagal reactivity to emotional film clips in those with complicated grief compared to healthy bereaved individuals. Notably, research has not investigated whether bereaved individuals grief and/or depression severity differ in their cardiovascular profile.

Specific Aims & Hypotheses

The current study seeks to answer questions about the observable behaviors and physiology of crying within the context of bereavement. The purpose of the present study is a preliminary analysis of parasympathetic activity and crying, including an attempt to replicate the finding that there is a general parasympathetic response to crying, and that depression may moderate that response. Importantly, the present study goes beyond the replication by investigating whether there is an emotional self-regulatory system associated with crying in bereavement specifically, and whether this is moderated by grief severity. Prior work leads us to consider three hypotheses in the context of a bereaved population:

1) Criers will have higher vagal cardiac control during recovery following grief recall compared to non-criers, accounting for baseline vagal cardiac control.

2) Grief severity will moderate the relationship between crying and vagal cardiac control during recovery, such that the relationship between crying and vagal recovery will be stronger when grief severity is lower.
3) Depression will moderate the relationship between crying and vagal cardiac control during recovery, such that the relationship between crying and vagal recovery will be stronger when depression is lower.

Method

Dataset

The Emotion Regulation Diary Study (ERDS) is a University of Arizona IRB approved study of 75 bereaved individuals from the southern Arizona area. Participants were recruited through recruitment letters sent to individuals who posted obituaries online; hospice organizations in Tucson that facilitated referrals and distributed recruitment letters; newspaper advertisements; flyers distributed across Tucson; and by word of mouth through other studies. Participants gave informed consent to participate in the study and were compensated $35.00 for their participation.

Recruitment focused on recently bereaved adults who experienced the death of a first-degree relative (spouse, sibling, parent, child) or a person close “like family”. All participants underwent a brief medical screening prior to enrollment of the study. Individuals who had medical conditions that might have complicated the assessment of stress-related heart function (e.g., implanted pacemaker, etc.) as well as individuals who had situational factors that might have made it difficult for them to complete the study were excluded.

Procedure

Figure 1 details the ERDS procedures is taken from Palitsky (2020). Participants went through an initial visit where they provided informed consent to participate in the ERDS study and provided self-report data. After a 14-day period, participants returned to the lab for their second visit. During the second visit, participants underwent an emotion elicitation protocol using a Grief Recall (GR) interview (Ehrenthal et al., 2011; Palitsky et al., under review).

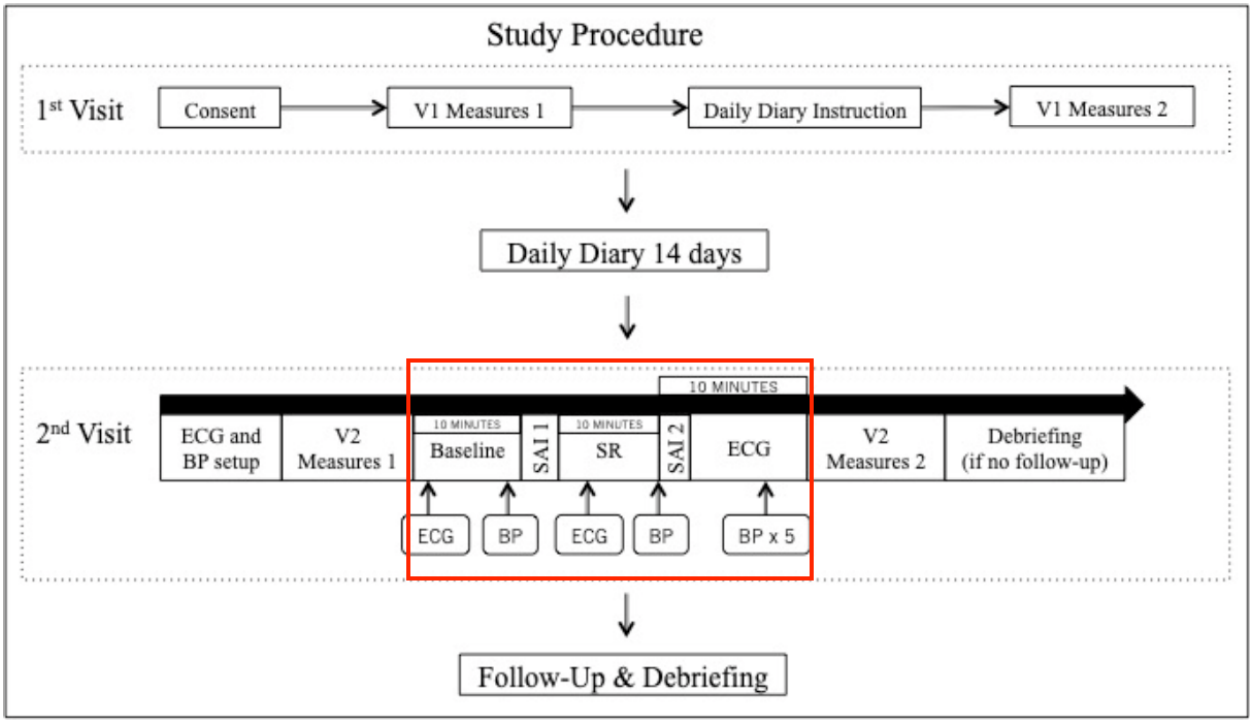
Prior to the GR interview, participants were connected to a cardiac measurement system, which included four leads for impedance and two leads for ECG. ECG was recorded continuously across baseline for 10 minutes (T1), the GR interview, and the recovery period (T2), where participants were asked to sit quietly for 10 minutes. Participants were videotaped for the duration of their time in the laboratory.

The GR interview began with asking participants to think of a time when they felt alone and abandoned and wished that their loved one had been there for them. After participants indicated that they thought of a time when they felt this way, they were asked to rate how stressful the situation was on a scale from one to ten, where ten was the most stressful. The interviewer asked the participant to expand on the situation. When the interviewer decided that this was an appropriate situation for the recall task, they said, “tell me about it,” and the ECG was marked as the start of GR. Upon preliminary investigation of the data, this was the point when many participants began to cry.

The current study consists of secondary analyses from the parent study, ERDS. In the current study, further exclusion was necessary to only include participants whose video was able to be behaviorally coded. This study originally looked at video data from 60 individuals who participated in ERDS, but due to a missing ECG value, 59 participants made up the final total study pool.

The current study investigated the physiological (ECG) and emotional events that took place before and after the GR interview. Time 1 (T1) corresponds to the average vagal control (RSA) over the baseline period, while Time 2 (T2) corresponds to the average vagal control (RSA) in the recovery period. The portion of the data used for the current study is outlined in red in Figure 1.

Figure 1. ERDS Study Procedures



Measures

Grief Severity

Prolonged Grief-13 Scale (PG-13) (Prigerson et al., 2009) is a 13-item assessment that addresses the frequency, duration, and magnitude of symptoms associated with prolonged grief as well as any impairment due to these symptoms. For the current study, this scale was used to

assess grief severity. All items on the PG-13 require Likert-style responses, except for two items which are dichotomous yes/no questions (and are not used for the continuous measure). Possible scores on the PG-13 range from 11 to 55 with higher scores indicating more grief symptom severity.

Depression

The Center for Epidemiologic Studies Depression Scale (CES-D) (Radloff, 1977) assesses for depression symptoms. The CES-D is a self-report questionnaire comprised of 20 Likert-style questions. These questions investigate the frequency a participant experiences emotional, cognitive, motivational, and somatic symptoms of depression. Scores on the CES-D range from 0 to 60 with higher scores indicating greater depressive symptom severity.

Vagal Cardiac Control during Baseline and Recovery

Participants' ECG data was collected by means of a dual impedance cardiograph system (Mindware Technologies: ECG sampling rate >100 Hz, signal gain = 500) using a six-lead configuration. The leads were placed on the right clavicle, suprasternal notch, xyphoid process, lower left (false) rib, and two leads placed on the back, 1.5 inches above the suprasternal notch and below the xyphoid process respectively. Leads were connected to adhesive electrodes which were applied using 7% chloride gel. Respiratory sinus arrhythmia (RSA) was calculated in the frequency domain as a measure of heart rate variability, by means of calculating the variance in the time series related to respiration (.12-.042 Hz) in Mindware HRV Analysis 3.1.3 software (Palitsky, 2020). The processing of data to mitigate R-spikes in the ECG waveforms was conducted visually with the use of BioLab v. 3.1.2 (Mindware Technologies LTD) and Mindware HRV Analysis Program v. 3.1.3 (Palitsky, 2020). The author assisted in conducting

the ERDS study by connecting the participants to the six ECG leads and reduced the HRV data to account for ectopic or aberrant beats using Mindware.

Crying

Palitsky (2020) did not investigate crying in his ERDS study; therefore, new analyses explored the potential relationship between crying and vagal response. New coding of perceived emotional behavior included global coding of crying behavior, but also additional descriptive components and associated behaviors of crying such as whether the interviewee faced the interviewer or not. This additional descriptive coding provided a deeper understanding of the range of crying behavior, previously not researched in a bereaved sample, although more detailed coding was not used for predictive analysis in the current study.

First, we coded the 10-minute interview in five-minute segments. The videos will be coded for whether the participant cried or not in each five-minute segment (not crying = 0, one segment crying = 1, both segments crying = 2). Then, additional coding in each segment was used to differentiate between which components of crying the participant demonstrated. Vingerhoets and Bylsma (2016) classify three different components of crying: (a) production of tears, (b) changes in breathing, and (c) distressed vocalization. The video quality was not clear enough to always see tears on the faces of the participants, but there were other indicators that tears were produced such as reaching for Kleenex. For this reason, use of multiple coders ensured agreement of the perceived behaviors. The three components of crying were dichotomous. For example, *did the participant engage in component (a)? (no = 0, yes = 1).*

Video data was also coded for behaviors exhibiting disengagement or avoidance. These behaviors included whether the participant had their arms crossed or not, whether the participant was looking up towards the ceiling or away from the interviewer or not, or if the participant gave

short answers (one word) (Dael et al., 2012). These behaviors could describe the social communication functions associated with crying but are not in the analyses for the current study.

In addition to behaviors exhibiting disengagement or avoidance, video data was coded for evidence of agitation, nervousness, and anger. Evidence of agitation or nervousness included restless legs, laughter, or fidgeting. Evidence of anger included verbal indication that the participant is angry, interrupting the interviewer, or an increase in their vocal volume. These behaviors were coded to indicate whether the crying could be considered "protest" or "despair".

In the case of missing data or disagreement between coders, the first author determined the cry status or other investigated behavior in the video segments.

Hypotheses

Hypothesis 1 (H1): Criers will have higher vagal control at T2 compared to non-criers at T2, accounting for T1.

Hypothesis 2 (H2): Grief severity will moderate the relationship between crying and vagal recovery, such that the relationship between crying and vagal recovery will be strongest when grief severity is lower.

Hypothesis 3 (H3): Depression will moderate the relationship between crying and vagal recovery, such that the relationship between crying and vagal recovery will be strongest when depression is lower.

Power Analysis

Although little work assessing the association between crying and vagal reactivity currently exists, a power analysis using estimates from Rottenberg and colleagues (2003) was performed.

Given a mean difference of 0.3 in vagal reactivity between criers (M = 6.4, SD = 1) versus non-criers (M = 6.1, SD = 1), and a 0.9 within-person correlation between pre and post vagal reactivity, the power to detect a significant difference between groups was approximately 0.67 for unequal group sizes with a crier group of n = 26 (35%), and approximately 0.83 assuming equal group sizes (n = 37 for criers and non-criers).

Table 1

Demographics Characteristics

Sample Characteristics	n	%	*M*	*SD*
Gender				
Female	41	68.33		
Male	17	28.33		
Non-Binary	1	1.67		
Age			65.67	17.66
Days since death			139.7	76.7
Loss type				
Spouse/ romantic partner	34	57.63		
Child > 21 years	4	6.78		
Sibling	6	10.17		
Parent	10	16.95		
Grandparent	2	3.39		
Friend	5	8.47		
Ethnicity				
White	55	91.67		
Latinx	4	6.67		

Note. N = 59

Data Analysis

Analysis of the data began with an interrater reliability check between coders. Following evidence of reasonable interrater reliability, all data was checked for normality, and skewness, and adjusted (e.g., transformations, missing data replacement) to meet statistical assumptions.

For H1, a multiple linear regression was conducted to determine if crying predicts RSA at T2 in criers compared to non-criers, accounting for T1. By accounting for T1, we investigated cardiac reactivity of vagal recovery to crying during the grief recall task. Age and gender were used as additional predictors to improve the final model fit.

$$Y(vagal\ recovery_{T2}) = \beta_0 + \beta_1(crying) + \beta_2\ (vagal\ control_{T1})$$

Both H2 and H3 are also multiple linear regression hypotheses. H2 and H3 have interaction terms, so depending on whether the interaction term is significant, I will a) conduct a simple slopes analysis or b) examine the main effects model. The models for H2 and H3 are listed respectively below. Again, age and gender will be used as additional predictors if they improve the final model fit.

H2 main effects model:

$$Y(vagal\ control_{T2}) = \beta_0 + \beta_1(crying) + \beta_2(grief\ severity) + \beta_3\ (vagal\ control_{T1})$$

H3 interaction model:

$$\begin{aligned} &Y(vagal\ control_{T2}) \\ &\quad = \beta_0 + \beta_1(crying) + \beta_2(grief\ severity) \\ &\quad + (\beta_3(crying \times grief\ severity)\ +\ \beta_4\ (vagal\ control_{T1}) \end{aligned}$$

For H3, the equations will be the same, replacing depression for grief severity. Each model fit will be checked for significance, and the simplest model that explains the most variance will be chosen. Age and gender will be used as additional predictors.

Results

An interrater reliability check was conducted using a Fleiss kappa. The Fleiss kappa is used for inter-rater reliability on dichotomous variables with more than two raters. There were

three coders per video and a Fleiss kappa was conducted for each video segment. A kappa of .75 for was found for both video segments. It was concluded that 31% of participants did not cry in either segment, 37% cried in one segment, and 32% cried in both segments of the interview.

For all three hypotheses, and as seen in Table 2, we examined correlations between all variables used in each model. Specifically, after examining the relationship between T1 RSA and cry length, we found that the two variables are not associated. This means that regardless of how long an individual cried their RSA at T1 did not differ from one another. Thus, we included T1 RSA as a predictor in the models to evaluate vagal recovery. Additionally, neither grief severity nor depressive severity were associated with how long an individual cried.

A regression analysis was performed in R (v4.0.5; R Core Team, 2021) to test H1, assessing the amount of crying as a predictor of RSA. Amount of crying, or cry length, was determined by if a participant did not cry at all, cried in one segment, or cried in both segments (No Cry = 0 segments, Cry 1 = 1 segment, Cry 2 = 2 segments). There was a significant effect of T1 RSA on T2 RSA, $b = .92$, $SE = .07$, $p < .001$, but crying was not a significant predictor of T2 RSA, $b = .03$, $SE = .15$, $p = .82$. The addition of gender and age did not improve the model fit ($R^2 = 0.76$) over the original ($R^2 = .77$).

To test H2, regression was used to assess the moderating role of grief severity in the association between how long a participant cried and their T2 RSA. Grief severity was grand-mean centered. An interaction term was created by multiplying cry length by grief severity. Regression analyses did not reveal a significant interaction, $b = -.02$, $SE = .02$, $p = .31$. The model was tested further using age and gender as additional predictors ($R^2 = 0.77$), but additional covariates did not improve the model fit over the original ($R^2 = 0.78$). Further moderation analyses were not conducted; however, a main effects analysis was conducted. The effect of

crying on T2 RSA was not significant $b = .02$, $SE = .15$, $p = .88$. T1 vagal control showed a significant main effect on T2 vagal control, $b = .93$, $SE = .07$, $p < .001$. Grief severity did not show a significant effect on T2 vagal control, $b = .02$, $SE = .12$, $p = .13$. Figure 2 illustrates the change in RSA (T2 – T1).

To test H3, regression was used to assess the moderating role of depression in the association between whether how long a participant cried and their T2 RSA. Cry length was used in the same was as H2 and depression was grand-mean centered. An interaction term was created by multiplying cry length and depression. Regression analyses did not reveal a significant interaction, $b = .00$, $SE = .01$, $p = .86$. The model was tested further using age and gender as additional predictors to improve the model fit ($R^2 = 0.77$); however, additional predictors did not improve the model fit over the original ($R^2 = 0.78$). Further moderation analyses were not conducted; however, a main effects analysis was conducted. The effect of crying on T2 RSA was not significant $b = -.02$, $SE = .15$, $p = .91$. Vagal control at T1 showed a main effect on T2 vagal control, $b = .94$, $SE = .07$, $p < .001$. Depression also showed a main effect on T2 vagal control, $b = .02$, $SE = .01$, $p = .05$. Figure 3 illustrates a change in RSA (T2 – T1) by depressive severity.

Figure 2. Grief severity by change in RSA

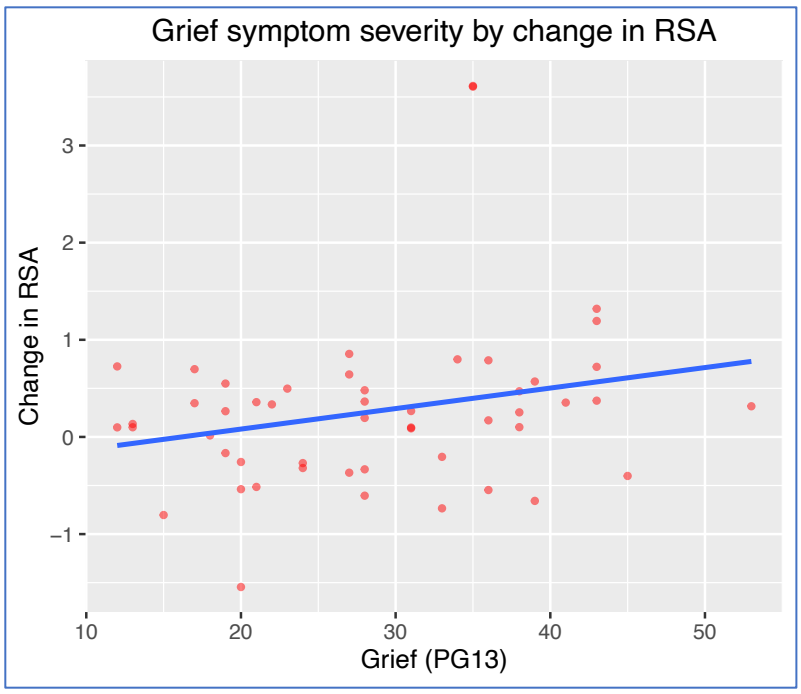


Figure 3. Depressive severity by change in RSA.

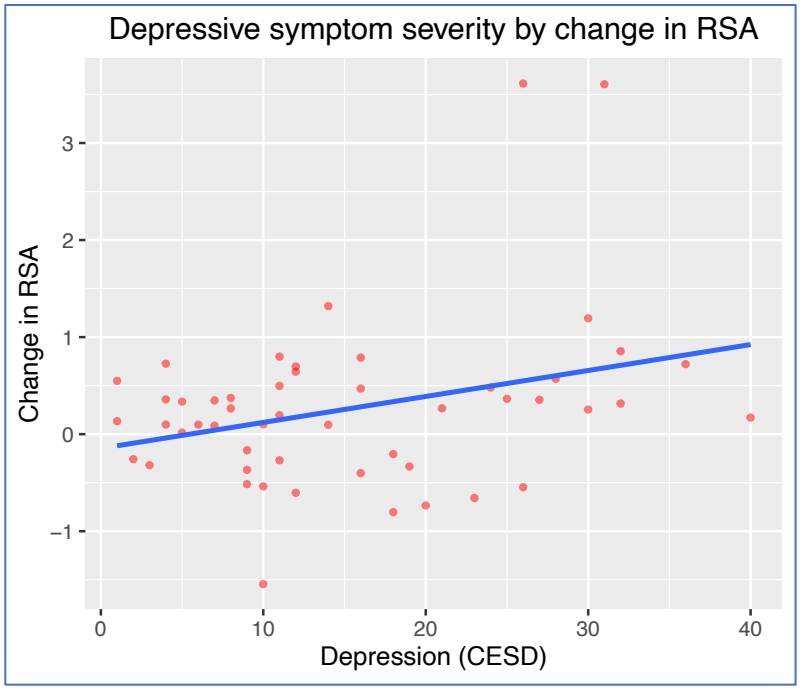


Table 2

Means, standard deviations, and correlations with confidence intervals

Variable	*M*	*SD*	1	2	3	4	5	6
1. Gender	1.73	0.49						
2. Age	65.83	17.7	0.09 [-.17, .34]					
3. Grief severity (PG13)	27.83	9.82	-0.09 [-.34, .17]	-.27* [-.50, -.02]				
4. Depressive severity (CESD)	14.51	9.86	-0.18 [-.42, .08]	-0.19 [-.43, .07]	.69** [.53, .81]			
5. T1 RSA	4.84	1.75	0.03 [-.24, .30]	-0.03 [-.29, .25]	-0.14 [-.39, .14]	-0.22 [-.46, .05]		
6. T2 RSA	5.14	1.81	0.05 [-.22, .31]	-0.02 [-.28, .25]	0 [-.26, .27]	-0.04 [-.30, .23]	.88** [.80, .93]	
7. Cry length	1.02	0.8	0.15 [-.11, .39]	0.15 [-.11, .39]	0.04 [-.22, .29]	0.16 [-.10, .40]	-0.13 [-.39, .14]	-0.07 [-.33, .20]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$.

Discussion

In the present study, we proposed three hypotheses. Hypothesis 1 (H1) predicted that criers would have higher vagal control at T2 compared to non-criers at T2, accounting for T1. The results of the present study did not support H1. Since the study had several limitations, it is difficult to draw a strong conclusion from this null result, but it decreases confidence in previous theories that crying has a self-regulatory role through vagal control (Gracanin et al., 2014; Rottenberg et al., 2003).

Hypothesis 2 (H2) predicted that grief severity would serve a moderating role between crying and vagal recovery, and similarly, hypothesis 3 (H3) predicted depression would serve a moderating role between crying and vagal recovery. Overall, the results showed that neither grief severity nor depression moderated the relationship between crying during the grief recall task and vagal recovery after the grief recall task.

Ultimately, we did not successfully replicate the results found by Rottenberg and colleagues (2003). While our models were underpowered to test for moderation, it still leaves the question of how to interpret our results. Our correlation analysis found that grief and depression symptoms were associated with each other, but neither of them were associated with RSA at T1 or T2. Future research should compare vagal control in individuals with depression (as done by Rottenberg and colleagues) compared to bereaved individuals. More specifically, our study was not a direct replication as it included only bereaved individuals, some with increased depressive severity and some with increased grief severity. Finally, neither grief severity nor depressive severity were associated with how long an individual cried. These findings can be interpreted as suggesting that grief and depression might not differ at the level of vagal control as much as we might have thought.

However, an interesting and noteworthy finding emerged comparing the results of the moderation analyses for H2 and H3. The H2 statistical model included grief severity, measured using the PG13 scale, and the H3 statistical model included depressive symptom severity, measured using the CES-D. Depressive symptom severity significantly predicted **higher** RSA at T2 (albeit at a $p = .05$ level), while grief symptom severity did not. Individuals with depression compared to healthy controls have reduced RSA at baseline and during recovery (Koch et al., 2019; Schiweck et al., 2018). Our finding that depressive severity predicts increased RSA at T2

does not replicate this literature. Also in the literature, bereaved individuals compared to non-bereaved controls have lowered RSA at baseline and in recovery (Fagundes et al., 2018; Buckley et al., 2012; O'Connor et al., 2002; LeBlanc et al., 2016), and we also did not see the parallel extension of this when looking at grief severity.

The current study faced several limitations. First, this study had a small number of participants, most of which were women. Considering Thayer and colleagues' (1998) discussion of gender differences in RSA among depressed participants, it is important to consider a more representative sample with an increased gender diversity. Additionally, the current study had a large range of participant ages (18 – 90 years old). As outlined by Shaffer and Ginsberg (2017) age can vastly effect RSA. Older individuals appear to have largely different heart rate variability than younger individuals (Shaffer & Ginsberg, 2017), although age was not associated with RSA in this study. Finally, this study is secondary to the parent study, ERDS, which was not originally designed to investigate crying. Since the original study was not intended to investigate crying, video and audio quality decreased the accuracy of assessing crying status.

Although we failed to replicate previous findings (Rottenberg et al., 2003) and we failed to find a moderating role of grief severity between crying and vagal recovery, there is a pattern in the literature, on a cardiovascular level, that bereavement and depression appear to act similarly compared to healthy controls. Our study does not support the conclusion that grief severity is associated with baseline RSA or vagal control during recovery.

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