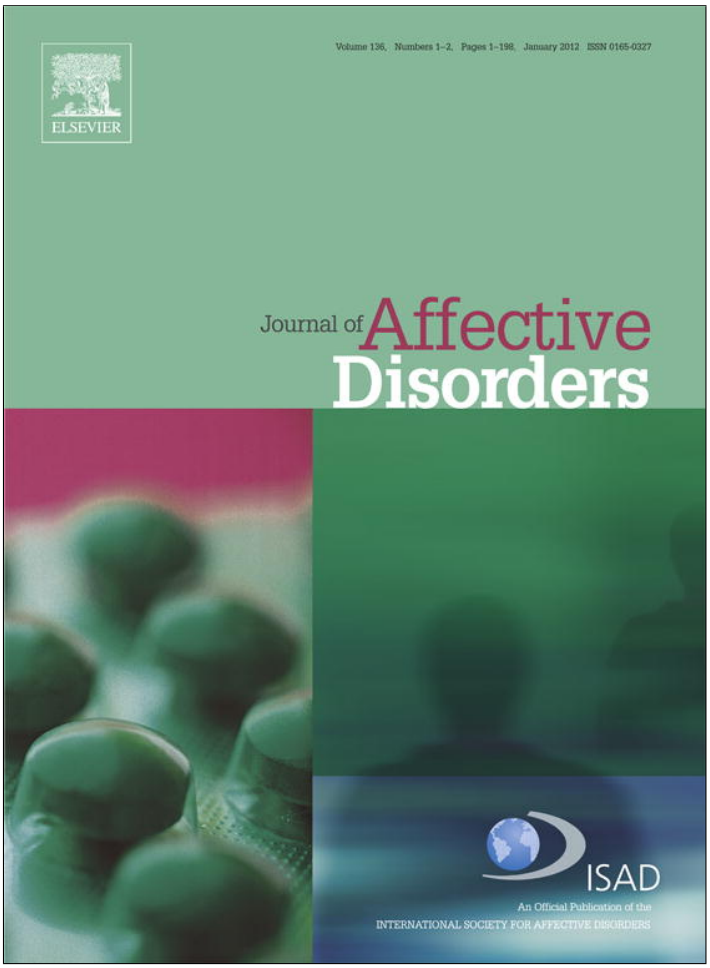

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Research paper

Emotional and physiological reactivity in Complicated Grief

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ABSTRACT

Background: Grief is a psychobiological response to the loss of a loved one. Some grief theorists suggest that this predictable response may arise from withdrawal of psychobiological regulation previously provided by the deceased (e.g. assistance with emotion regulation). Accordingly, recovery from loss may require bereaved individuals to re-establish self-regulatory control to avoid developing Complicated Grief (CG). This model implies that adults with CG may exhibit aberrant emotional responding to environmental stimuli. The present study was designed to test this hypothesis.
Methods: We recruited a sample of 23 bereaved adults with CG and 26 healthy bereaved adults to complete an emotional reactivity paradigm. Participants watched a series of emotional film clips and provided measures of their self-reported emotional response. We also assessed their heart rate, respiratory sinus arrhythmia (RSA), and skin conductance level in response to these clips.
Results: Though emotional and physiological differences between the groups were rare, the CG group exhibited attenuated RSA reactivity to some emotional film clips, suggesting blunted parasympathetic nervous system reactivity in those with the disorder.
Limitations: Limitations include the modest sample size and unequal group sizes.
Conclusions: Individuals with CG do not exhibit pervasive differences in emotional and physiological reactivity compared to healthy bereaved individuals. However, we did observe evidence of blunted parasympathetic nervous system reactivity in individuals with CG, which may mediate emotional inflexibility among those who develop the disorder.

1. Introduction

Grief is a psychobiological response to the death of a loved one characterized by sadness, yearning, waves of emotional pain, and loss of appetite and sleep (Clayton et al., 1968; Lindemann, 1944; Parkes, 1972). Although symptoms usually abate with time (Bonanno et al., 2002), about 6.7% of bereaved individuals experience them for a year or longer (Kersting et al., 2011). The syndrome comprising disabling, chronic grief symptoms is known as Complicated Grief (CG) or Persistent Complex Bereavement Disorder (PCBD; American Psychiatric Association, 2013).

Research on maternal-infant separation has deepened our understanding of grief. When infant mammals are deprived of maternal contact, they display separation distress (e.g. facial expressions of sadness, vocalization, agitation, decreased social interaction and decreased/dysregulated food intake) resembling the human grief response (Hofer, 1984). This separation reaction is triggered by the simultaneous withdrawal of multiple maternal psychobiological regulators (e.g. body warmth, tactile stimulation, nutrition, etc.; Hofer, 1984). Accordingly, Hofer (1984) suggested that termination of hidden regulators provided by a deceased loved one may mediate the human grief response.

Consistent with his hypothesis, research has shown close relationships serve a co-regulatory function for both partners. Perhaps the best evidence for co-regulation between close relationship partners comes from the literature on stress-buffering. Specifically, numerous studies have demonstrated that both children and adults in the presence of a close relationship partner have reduced emotional and physiological reactivity to stressors (Carter et al., 1995; Coan et al., 2006; Feldman et al., 2010). Furthermore, even when the partner is absent, adults in a state of romantic love experience reduced physiological reactivity to negative emotional stimuli (Schneiderman et al., 2011). This outcome is likely mediated in part by the anti-stress effects of the neuropeptide oxytocin (Uvnas-Moberg, 1998). In addition, comforting thoughts and memories of an individual's close relationship partner may support emotion regulation through the instantiation of non-threatening appraisals (Mikulincer and Shaver, 2008).

Hence, the death of a loved one may dysregulate psychobiological systems that were previously under co-regulatory control (e.g. emotional regulation; Sbarra and Hazan, 2008). Recovery

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from loss is then predicated on the bereaved person's ability to re-establish self-regulation over these systems. An individual unable to do this may be at risk for CG (Sbarra and Hazan, 2008).

Theoretical work notwithstanding, there are scant data on emotional or physiological reactivity to emotional stimuli in people with CG. Individuals with CG do report experiencing intense negative emotions and physiological reactions in response to reminders of the deceased and the death (Horowitz et al., 1997; Shear et al., 2011). However, it remains unknown how individuals with CG respond to negative cues unrelated to their loss. Research on the stress-buffering effects of close relationships suggests that those with CG likely experience heightened emotional and physiological reactivity to negative emotional stimuli in daily life. However this hypothesis remains untested.

Individuals with CG also report symptoms of emotional numbing, loss of interest in relationships and activities, and difficulty experiencing positive memories of the deceased (APA, 2013; Horowitz et al., 1997; Prigerson et al., 2009; Shear et al., 2011). These symptom reports suggest that emotional dysregulation in those with CG might also include blunted emotional and physiological reactivity to positive environmental stimuli. These observations lead to a second untested hypothesis that individuals with CG will demonstrate blunted emotional and physiological reactivity to positive emotional stimuli in daily life.

To test these hypotheses, we had bereaved participants with and without CG watch a series of emotional film clips (sad, scary, funny, and neutral) while we measured their self-reported emotional and autonomic reactivity. Autonomic outcome measures included heart rate (HR), skin conductance level (SCL), and respiratory sinus arrhythmia (RSA, i.e. high frequency heart rate variability). HR is affected by both the sympathetic and parasympathetic branches of the autonomic nervous system (Andreassi, 2007; Stern et al., 2001), thus increases in HR reflect participants' general physiological reactivity. In contrast, SCL is a relatively pure measure of sympathetic nervous system (SNS) activity (Andreassi, 2007) and thus increases in SCL reflect participants' SNS reactivity. Finally, RSA reflects parasympathetic nervous system (PNS) inhibitory control of HR via the vagus nerve, which is withdrawn in response to stress (Porges, 2011). Decreases in RSA therefore provided a measure of participants' PNS reactivity.

2. Methods

2.1. Participants

We recruited bereaved adults via advertisement and referral. To meet inclusion criteria, all participants must have experienced the death of a close relationship partner (i.e. a parent, child, sibling, or romantic partner) more than 12 months prior to the study. Loss of a biological grandparent was also allowed ($n=2$) if the grandparent was the participant's primary parental figure. Exclusion criteria were lifetime psychosis, lifetime manic episode, lifetime hypomanic episode, past 12-month substance dependence, or past 12-month alcohol dependence, as assessed by the MINI International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). Individuals were also excluded if they reported characteristics that could affect cardiac measures of physiological reactivity (HR and RSA), including a physician-diagnosed heart murmur, a pacemaker, body mass index (BMI) greater than 33, or if they were taking medications whose side effects affect the speed or pattern of the heartbeat.

Eligible participants were assigned to the bereaved with CG or the healthy bereaved group based on the following criteria. Participants who scored >4 on the Brief Grief Questionnaire (BGQ; Shear et al., 2006) at phone screen and also scored >25 on the Inventory of Complicated Grief (ICG; Prigerson et al., 1995) during the lab visit were assigned to the CG group. Participants who scored ≤ 4 on the BGQ at phone screen and ≤ 25 on the ICG during the lab visit were assigned to the healthy bereaved group. Participants with discrepant group placement based on their BGQ and ICG scores were excluded. We also administered the DSM-5 PCBD criteria (APA, 2013) during the lab visit. However, as these criteria have not been empirically validated, we did not limit participation based on whether participants met these criteria.[1]

Fifty-five participants qualified for the study. Data for six were excluded from analyses. Two refused to complete the emotional reactivity paradigm, two were excluded for equipment difficulties, one was excluded because of an inability to wear the respiration band, and one was excluded for excessive movement which rendered his data unusable. Hence, we analyzed data for 23 participants with CG and 26 healthy bereaved participants.

2.2. Procedure

The Harvard University Committee on the Use of Human Subjects approved the protocol. Participants were prescreened by phone and those potentially eligible completed a 2.5-h lab session. After obtaining written informed consent, we administered a clinical interview to all participants that included the ICG (Prigerson et al., 1995), the DSM-5 PCBD criteria (APA, 2013), and the MINI (Sheehan et al., 1998).

After the clinical interview, participants completed the emotional reactivity paradigm in a testing room where they sat opposite a desktop computer. We attached electrocardiogram electrodes (BIOPAC Systems Inc. EL503 general-purpose disposable electrodes) to participants' left and right wrists as well as electrodermal activity electrodes (BIOPAC Systems Inc. EL507 electrodermal activity electrodes) to the medial volar phalanges of their left middle and left ring fingers. We also attached a respiration band (BIOPAC Systems Inc. SS5LB respiratory effort transducer) around participants' chests at the point of maximum respiratory expansion. These electrodes and respiration band measured participants' physiological reactivity during the paradigm via a BIOPAC MP100A system (BIOPAC Systems Inc., Goleta, CA).

The task instructions and emotional film clips were presented on a desktop computer via Superlab 4.5 stimulus presentation software (Cedrus Corporation, San Pedro, CA). Participants were instructed to sit quietly for five minutes to allow for baseline data collection. Participants then began the emotional film clips task, which involved watching four film clips and rating their emotional responses to these film clips. We selected four film clips based on recommendations by Gross and Levenson (1995). The sad clip was a 2 min and 44 s segment from the movie *The Champ*, in which a boy watches his father die (Zeffirelli, 1979). The scary clip was a 1 min and 23 s segment from the movie *The Shining*, in which a boy has a paranormal experience while playing in a hallway (Kubrick, 1980). The funny clip was a 2 min and 35 s segment from *When Harry Met Sally*, in which a woman simulates an orgasm in a restaurant (Reiner, 1989). The neutral clip was a 2 min and 49 s segment of waves crashing on a beach (Amiri, 2013).

The order of the film clips was randomly assigned per participant (order counter-balanced between groups). Before each film clip, participants had 2 min to rate their pre-clip emotions. Specifically participants rated their subjective experience of the target emotion for each film clip (sad film – sad, scary film – afraid, funny film – amused, and neutral film – calm) on Visual Analogue Mood Scales (VAMS; Bond and Lader, 1974). Participants were then given

[1] Among CG participants, 10 met current DSM-5 PCBD criteria, whereas 13 did not; 3 missed Criterion B, 9 missed Criterion C, and 1 denied Criterion D.

a 20 s warning before the start of each film clip to ensure that they were attending to the computer monitor. After watching each film clip, participants had 2 min to rate their post-clip level of the target emotion for each clip (sad film – sad, scary film – afraid, funny film – amused, and neutral film – calm) on the VAMS. We monitored data collection from behind a one-way mirror.

After the emotional film tasks, participants completed the Trier Social Stress Test (Kirschbaum et al., 1993). Unfortunately, too few participants completed the task to enable interpretation of these data. Participants either refused to do this task or were excluded because of hypertension. However, as participants were aware that they would complete the Trier following the emotional film clips task, we note the Trier here in order to fully characterize the study methods. Finally, participants completed a battery of questionnaires via the Internet survey tool Qualtrics and were debriefed and compensated $50.

2.3. Measures

2.3.1. Brief Grief Questionnaire (BGQ; Shear et al., 2006)

The BGQ is a 5-item, researcher-administered questionnaire of CG symptom severity. Higher scores indicate more severe CG. The BGQ has good internal consistency (α=.75; Ito et al., 2012). A score of >4 on the BGQ is considered a positive screen for CG (Shear et al., 2006).

2.3.2. Inventory of Complicated Grief (ICG; Prigerson et al., 1995)

The ICG is a 19-item, self-report instrument of CG symptom severity. Higher scores indicate more severe CG. The ICG has excellent internal consistency (α=.94; Prigerson et al., 1995). A score of >25 on the ICG is considered a positive screen for CG (Prigerson et al., 1995).

2.3.3. Diagnostic assessments

We administered the DSM-5 PCBD criteria (APA, 2013) and the MINI (Sheehan et al., 1998) to all participants during the clinical interview. The MINI is a structured diagnostic interview that assesses for current and some lifetime DSM-IV disorders (Sheehan et al., 1998).

2.3.4. Visual Analogue Mood Scales (VAMS; Bond and Lader, 1974)

We used VAMS to assess participants' subjective emotional experiences immediately before and after watching each of the emotional film clips. To complete the VAMS, participants responded to the question, “How ____ do you feel right now?” A different emotional adjective occupied the blank space for each film clip (sad clip – sad; scary clip – scared; funny clip – amused; neutral clip – calm). Participants rated their current emotional state by selecting a number along a line anchored at 0 (“not at all”) and 100 (“extremely”).

2.3.5. Demographics questionnaire

Participants completed a brief questionnaire at the end of the lab visit, which assessed demographic characteristics (e.g. age, sex) as well as loss-related characteristics (e.g. relationship to the deceased, date of the death).

2.4. Psychophysiological data analysis

Psychophysiological data files were saved in Acq*Knowledge* and analyzed with MindWare 3.0 (MindWare Technologies LTD, Gahanna, OH). We extracted HR and RSA data from electrocardiography and respiration data with the MindWare HRV 3.014 module. The electrocardiography and respiration data for the baseline as well as the four emotional film clips were analyzed in 30-s epochs (epochs <27 s were not analyzed resulting in 10 usable epochs for baseline, 5 for the sad clip, 2 for the scary clip, 5 for the neutral clip, and 5 for the funny clip). We visually examined data for each epoch to assess for the presence of artifacts (e.g. movement artifacts, irregular beats), and edited artifacts with the *Edit R's* tool in MindWare HRV. We excluded epochs that required edits to ≥10% of the data segment (2.3% of epochs excluded). RSA calculation for each epoch was then performed via spectral analysis using the Fast Fourier Transformation (FFT) technique. These methods are consistent with recommendations made by Berntson et al. (1997). The HRV module also provided a HR value for each epoch in beats per minute. We calculated mean HR and RSA for baseline as well as the four emotional film clips by averaging across the relevant epochs. The first author performed the HR and RSA analyses for all study participants. In addition, the second author performed HR and RSA analyses for a randomly selected 25% of participants. Inter-rater reliability was excellent for both HR (r=1.00, p<.001) and RSA (r=0.998, p<.001).

We used the MindWare EDA 3.012 module to extract SCL level from the electrodermal activity data. One SCL value was extracted for the baseline segment as well as for each of the four emotional film clips. This value represents participants' mean SCL in microsiemens for the entire baseline period or the entire emotional film clip. Mean SCL data were positively skewed. We therefore used a log transformation to normalize the SCL data and conducted subsequent analyses on the logSCL data.

3. Results

3.1. Sample characteristics

Sample demographic and clinical characteristics are presented in Table 1. CG and healthy bereaved participants did not differ with regard to age, sex, or ethnicity. Race varied significantly by group, which appeared to reflect a larger proportion of Black participants in the CG group and a larger proportion of Asian participants in the healthy bereaved group.

Time since the loss, relationship to the deceased, and cause of death also differed between the groups. Individuals with CG reported a shorter time since the loss, were more likely to have lost a sibling or romantic partner (as opposed to a parent), and were more likely to have lost their loved ones to a sudden illness, accident, or suicide versus a prolonged illness. These findings reflect established risk factors for CG (Fujisawa et al., 2010; Kersting et al., 2011).

3.2. Baseline psychophysiology

At baseline, the groups did not differ in mean HR, $t(46)$=0.47, p=.64, d=0.14, or mean RSA, $t(46)$=1.41, p=.17, d=0.41. However, those with CG had lower logSCL than healthy bereaved participants, $t(47)$=2.49, p=.02, d=0.72. See Table 2.

3.3. Reactivity to the sad stimulus

3.3.1. Self-report data

We submitted VAMS sad scores for the sad clip to a mixed, time (pre-clip vs. post-clip) X group (CG vs. healthy bereaved) ANOVA. Participants reported more sadness post-clip than pre-clip, $F(1, 47)$=84.43, p<.001, η_p^2=.64, and CG participants reported greater overall sadness than did healthy bereaved participants, $F(1, 47)$=5.93, p=.02, η_p^2=.11. However, the interaction between time

Table 1
Sample demographic and clinical characteristics.

	Healthy bereaved	CG	Test for group difference
Age; M(SD)	38.73 (17.27)	45.30 (14.00)	$t(46.67)=1.47$, $p=0.15$
Sex			$X^2(1)=0.07$, $p=0.79$
Female; % (*n*)	73.08% (19)	69.57% (16)	
Male; % (*n*)	26.92% (7)	30.43% (7)	
Race			$X^2(3)=7.85$, $p=0.05$
White; % (*n*)	73.08% (19)	56.52% (13)	
Black; % (*n*)	7.69% (2)	26.09% (6)	
Asian; % (*n*)	15.38% (4)	0.00% (0)	
Multiracial; % (*n*)	3.85% (1)	13.04% (3)	
Not reported; % (*n*)	0.00% (0)	4.35% (1)	
Ethnicity			$X^2(1)=1.21$, $p=0.27$
Hispanic; % (*n*)	0.00% (0)	4.35% (1)	
Non-Hispanic; % (*n*)	100.00% (26)	91.30% (21)	
Not reported; % (*n*)	0.00% (0)	4.35% (1)	
Months since loss; M (SD)	114.62 (82.34)	64.61 (83.86)	$t(47)=2.10$, $p=0.04$
Relationship to deceased			$X^2(3)=17.79$, $p<0.001$
Mother; % (*n*)	38.46% (10)	8.70% (2)	
Father; % (*n*)	57.69% (15)	34.78% (8)	
Sibling; % (*n*)	3.85% (1)	34.78% (8)	
Romantic partner; % (*n*)	0.00% (0)	21.74% (5)	
Cause of death			$X^2(4)=10.50$, $p=0.03$
Sudden illness; % (*n*)	11.54% (3)	34.78% (8)	
Prolonged illness; % (*n*)	76.92% (20)	39.13% (9)	
Accident; % (*n*)	0.00% (0)	13.04% (3)	
Suicide; % (*n*)	0.00% (0)	4.34% (1)	
Other; % (*n*)	11.54% (3)	8.70% (2)	
ICG score; M(SD)	13.81 (5.29)	42.26 (12.37)	$t(29.03)=10.23$, $p<0.001$
DSM-5 PCBD; % (*n*)	0.00% (0)	43.48% (10)	$X^2(1)=14.20$, $p<0.001$
Current MDD; %(*n*)	0.00% (0)	26.09% (6)	$X^2(1)=7.73$, $p=0.01$

CG=Complicated Grief, ICG=Inventory of Complicated Grief, DSM-5=Diagnostic and Statistical Manual of Mental Disorder, PCBD=Persistent Complex Bereavement Disorder, MDD=Major Depressive Disorder.

and group was nonsignificant, $F(1, 47)=.54$, $p=.46$, $\eta_p^2=.01$. These results suggest no diagnostic group difference with regard to participants' emotional reactivity to the sad clip.

3.3.2. Psychophysiological data

Psychophysiological reactivity to the sad clip was examined with a series of mixed, stimulus (neutral clip vs. sad clip) X group (CG vs. healthy bereaved) ANOVAs. A significant interaction between stimulus and group would suggest group differences in physiological reactivity to the sad clip. Overall, the sad clip evoked lower HR than the neutral clip, $F(1, 47)=8.70$, $p=.01$, $\eta_p^2=.16$, but there were no significant effects of group, $F(1, 47)=0.32$, $p=.58$, $\eta_p^2=.01$, or film by group interaction, $F(1, 47)=1.04$, $p=.31$, $\eta_p^2=.02$, for HR.

With regard to RSA, participants demonstrated lower mean RSA in response to the sad clip compared to the neutral clip, $F(1, 47)=6.65$, $p=.01$, $\eta_p^2=.12$. The effect of group was nonsignificant $F(1, 47)=1.12$, $p=.30$, $\eta_p^2=.02$, and the film by group interaction for RSA was nonsignificant, $F(1, 47)=2.57$, $p=.12$, $\eta_p^2=.05$. Finally, for logSCL, participants demonstrated higher logSCL in response to the sad clip compared to the neutral clip, $F(1, 47)=7.70$, $p=.01$, $\eta_p^2=.14$, and healthy bereaved participants demonstrated higher logSCL than CG participants, $F(1, 47)=5.70$, $p=.02$, $\eta_p^2=.11$, However the film by group interaction for logSCL was nonsignificant, $F(1, 47)=.002$, $p=.96$, $\eta_p^2<.001$. Overall, results suggested no diagnostic group differences in participants' physiological reactivity to the sad clip. See Table 2.

3.4. Reactivity to the fear stimulus

3.4.1. Self-report data

We submitted VAMS fear scores for the scary film clip to a mixed, time (pre-clip vs. post-clip) X group (CG vs. healthy bereaved) ANOVA. Participants reported more fear post-clip than pre-clip, $F(1, 47)=44.60$, $p<.001$, $\eta_p^2=.49$. The main effect of group was nonsignificant, $F(1, 47)=1.58$, $p=.21$, $\eta_p^2=.03$, and the interaction between time and group was nonsignificant, $F(1, 47)=0.002$, $p=.97$, $\eta_p^2<.001$. Overall, results suggested no diagnostic group difference in participants' self-reported fear reactivity.

3.4.2. Psychophysiological data

Psychophysiological reactivity to the scary clip was examined with a series of mixed, stimulus (neutral clip vs. scary clip) X group (CG vs. healthy bereaved) ANOVAs. With regard to HR, analyses revealed a nonsignificant effect of film clip, $F(1, 47)=0.41$, $p=.53$, $\eta_p^2=.01$; a nonsignificant effect of group $F(1, 47)=.20$, $p=.66$, $\eta_p^2=.004$; and a nonsignificant film by group interaction, $F(1, 47)<0.001$, $p=.996$, $\eta_p^2<.001$. For RSA, there was a nonsignificant effect of film clip, $F(1, 47)=.57$, $p=.45$, $\eta_p^2=.01$; a nonsignificant effect of group $F(1, 47)=2.15$, $p=.15$, $\eta_p^2=.04$; and a nonsignificant film by group interaction, $F(1, 47)=.02$, $p=.88$, $\eta_p^2<.001$. Finally, for logSCL, we observed a trend towards higher

Table 2
Emotional and physiological reactivity group Ms(SDs).

	Baseline		Neutral clip		Sad clip		Scary clip		Funny clip	
	HB	CG	HB	CG	HB	CG	HB	CG	HB	CG
Pre-VAMS	N/A	N/A	75.19 (18.59)	70.91 (31.63)	5.58 (9.12)	21.39 (29.36)	3.23 (10.10)	11.30 (24.13)	8.31 (19.49)	14.78 (19.64)
Post-VAMS	N/A	N/A	76.85 (20.42)	81.30 (28.53)	54.50 (25.61)	63.04 (29.49)	29.88 (25.00)	37.61 (38.29)	51.35 (27.77)	77.74 (24.92)
HR	72.56 (11.52)	70.95 (12.18)	71.74 (11.05)	70.27 (11.71)	71.03 (11.29)	68.80 (12.42)	71.39 (11.89)	69.92 (11.79)	71.38 (11.20)	69.49 (12.28)
RSA	5.75 (1.40)	5.15 (1.53)	5.82 (1.34)	5.25 (1.48)	5.41 (1.47)	5.15 (1.39)	5.73 (1.52)	5.19 (1.15)	5.44 (1.30)	5.09 (1.32)
SCL	2.57 (2.05)	1.49 (1.75)	3.43 (2.38)	2.13 (2.44)	3.73 (2.43)	2.32 (2.72)	3.86 (2.57)	2.25 (2.90)	3.98 (2.71)	2.47 (3.23)
logSCL	0.27 (0.38)	−0.01 (0.40)	0.40 (0.39)	0.13 (0.42)	0.45 (0.39)	0.18 (0.41)	0.46 (0.39)	0.15 (0.41)	0.46 (0.40)	0.18 (0.42)

HB=Healthy bereaved, CG=Complicated Grief, VAMS=Visual Analogue Mood Scale; HR=heart rate in beats per minute; RSA=respiratory sinus arrhythmia; SCL=skin conductance level in μohms; logSCL=log transformation of SCL in μohms.

logSCL in response to the scary compared to the neutral clip, $F(1, 47)=3.99$, $p=.052$, $\eta_p^2=.08$, and healthy bereaved participants exhibited overall higher logSCL than CG participants, $F(1, 47)=6.49$, $p=.01$, $\eta_p^2=.12$. However, the film by group interaction for logSCL was nonsignificant, $F(1, 47)=1.21$, $p=.28$, $\eta_p^2=.03$. Taken together, these results suggest no group differences in participants' physiological reactivity to the scary stimulus. See Table 2.

3.5. Reactivity to the amusing stimulus

3.5.1. Self-report data

We submitted VAMS amused scores for the funny film clip to a mixed, time (pre-clip vs. post-clip) X group (CG vs. healthy bereaved) ANOVA. Participants were significantly more amused post-clip than pre-clip, $F(1, 47)=159.02$, $p<.001$, $\eta_p^2=.77$. Furthermore, participants with CG reported greater overall amusement than did healthy bereaved participants, $F(1, 47)=10.11$, $p=.003$, $\eta_p^2=.18$. Finally, the group by time interaction was significant, $F(1, 47)=5.62$, $p=.02$, $\eta_p^2=.11$, which reflected greater self-reported amusement for CG participants compared to healthy bereaved participants at post-clip, $t(47)=3.48$, $p=.001$, $d=1.00$, but not at pre-clip, $t(47)=1.16$, $p=.25$, $d=.33$. Thus, contrary to our hypothesis, participants with CG demonstrated greater self-reported emotional reactivity to the funny clip than did healthy bereaved participants.

3.5.2. Psychophysiological data

Psychophysiological reactivity to the funny clip was examined with a series of mixed, stimulus (neutral clip vs. funny clip) X group (CG vs. healthy bereaved) ANOVAs. With regard to HR, the effect of film clip was nonsignificant, $F(1, 46)=1.50$, $p=.23$, $\eta_p^2=.03$; the effect of group was nonsignificant, $F(1, 46)=0.30$, $p=.59$, $\eta_p^2=.01$; and the film by group interaction was nonsignificant, $F(1, 46)=0.04$, $p=.84$, $\eta_p^2=.001$. For RSA, participants had lower RSA in response to the funny clip compared to the neutral clip, $F(1, 46)=5.69$, $p=.02$, $\eta_p^2=.11$. The effect of group was nonsignificant, $F(1, 46)=1.90$, $p=.17$, $\eta_p^2=.04$, and the film by group interaction was nonsignificant, $F(1, 46)=3.63$, $p=.06$, $\eta_p^2=.07$. Finally, for logSCL, participants exhibited greater logSCL in response to the funny film clip compared to the neutral clip, $F(1, 47)=8.24$, $p=.01$, $\eta_p^2=.15$. Furthermore, healthy bereaved participants had higher logSCL than CG participants did, $F(1, 47)=5.89$, $p=.02$, $\eta_p^2=.11$. The film by group interaction for logSCL was nonsignificant, $F(1, 47)=0.16$, $p=.69$, $\eta_p^2=.003$. Overall, results suggested an absence of diagnostic group differences in participants' physiological reactivity to the funny clip. See Table 2.

3.6. Exploratory analyses of RSA reactivity

We observed trends toward film by group interactions for RSA reactivity to the sad ($p=.12$) and funny ($p=.06$) clips. To explore these further, we examined group differences in RSA reactivity to each emotional film clip by conducting separate repeated (neutral vs. emotional clip) ANOVAs for each diagnostic group. For participants with CG, analyses revealed a nonsignificant decrease in RSA from the neutral to the sad clip, $F(1, 22)=0.45$, $p=.51$, $\eta_p^2=.02$; and from the neutral to the funny clip, $F(1, 22)=.09$, $p=.77$, $\eta_p^2=.004$. In contrast, healthy bereaved participants had significant decreases in RSA from the neutral to the sad clip, $F(1, 25)=9.30$, $p=.01$, $\eta_p^2=.27$; and from the neutral to the funny clip, $F(1, 25)=12.42$, $p=.002$, $\eta_p^2=.33$. Neither group demonstrated a significant change in RSA from the neutral to the fear clip, CG: $F(1, 22)=.15$, $p=.70$, $\eta_p^2=.01$; and healthy bereaved: $F(1, 25)=.49$, $p=.49$, $\eta_p^2=.02$. Overall, these results suggest attenuated RSA reactivity to some emotional stimuli among those with CG when compared to healthy bereaved participants.

4. Discussion

Our study is the first to examine emotional and physiological responding to generic emotional cues in bereaved adults with and without CG. The primary findings from this investigation can be summarized as follows. (1) The groups did not differ at baseline on tonic HR or RSA, but the CG group demonstrated lower baseline SCL than the healthy bereaved group. (2) Contrary to our hypotheses, the groups did not differ in their self-reported emotional or physiological responses to either the scary or sad film clips. (3) Surprisingly, the CG group exhibited *heightened* self-reported emotional reactivity to the funny clip, yet the groups did not differ physiologically in their response to this clip. (4) Exploratory analyses suggested that individuals with CG have blunted RSA reactivity to some emotional stimuli.

4.1. Baseline autonomic arousal

CG is a risk factor for physical health problems, including cardiac problems (Prigerson et al., 1997); however, relatively little is known about tonic autonomic arousal in individuals with the disorder. We therefore explored baseline differences in HR, RSA, and SCL between bereaved adults with and without CG. For HR and RSA, baseline means did not differ between the groups. These findings were surprising in light of the cardio-protective effects of oxytocin (Norman et al., 2012; Uvnas-Moberg, 1998) and good quality close relationships (e.g. Diamond and Hicks, 2005; Donoho et al., 2015). We therefore anticipated evidence of elevated autonomic arousal among individuals who had not recovered from a significant loss. Instead we found no association between tonic HR or RSA and CG diagnosis.

We did observe lower baseline SCL among those with CG relative to the healthy bereaved individuals. This finding is consistent with data documenting a negative association between depression symptoms and electrodermal activity (Schwedtfeger and Rosenkaimer, 2011). However, it remains unclear what might account for blunted electrodermal activity in individuals with emotional disorders, such as depression and CG.

4.2. Emotional and physiological reactivity in CG

Our primary aim was to examine self-reported emotional and physiological reactivity to emotional stimuli among bereaved individuals with and without CG. Based on the established association between close relationships and attenuated reactivity to stress, we hypothesized that individuals who had lost a close relationship partner and had subsequently developed CG would show hyper-reactivity to negative laboratory stimuli. Instead, we found no evidence to suggest that individuals with CG are more emotionally or physiologically reactive to a sad or scary film clip. We also hypothesized that those with CG would show blunted reactivity to positive stimuli. Instead, we observed *greater* self-reported amusement among those with CG in response to the funny clip, and an absence of significant group differences in physiological reactivity to the funny clip. Both sets of results were especially surprising in light of the fact that individuals with CG report intense emotional and physiological reactivity to reminders of the deceased as well as emotional numbing symptoms (APA, 2013; Horowitz et al., 1997; Prigerson et al., 2009; Shear et al., 2011).

Perhaps attachment-mediated differences in reactivity to emotional stimuli are more strongly tied to acute, rather than prolonged, grief states. Though the modal response to loss is resilience, approximately 25% of bereaved individuals experience intense symptom of acute grief immediately following loss (Bonanno et al., 2002). Many of these individuals gradually recover,

whereas a subset does not (Bonanno et al., 2002). It is likely that different processes contribute to the development vs. maintenance of grief. For example, the withdrawal of co-regulation provided by the deceased partner may produce short-term emotional dysregulation for some bereaved individuals, expressed as acute grief. Indeed, acute grief is characterized by both panic (Parkes, 1972) and anhedonia (Lindemann, 1944) symptoms. Individuals suffering from acute grief may gradually reestablish emotional self-regulation in the months following loss; however, subsequent maladaptive responses such as catastrophic interpretations of grief reactions or grief-related avoidance (see Boelen et al., 2006) may prolong other grief symptoms and, for some, result in CG. Future studies could explore this hypothesis by comparing emotional reactivity between bereaved individuals who are and are not experiencing acute grief reactions.

Another possibility is that individuals with CG do experience aberrant emotional reactivity, but only in response to ideographic grief-related stimuli. For example, Bonanno et al. (2007) obtained HR measures from bereaved adults as they discussed their relationship with the deceased. After controlling for participants' depression and PTSD symptoms, the authors found that CG symptoms predicted *decreases* in participants' HR when talking about the deceased (Bonanno et al., 2007). In another study, Diminich and Bonanno (2014) asked bereaved adults to discuss moments of conflict and intimacy that they had experienced with the deceased as well as with a living attachment figure, while they obtained measures of participants' self-reported affect, emotion word use, and facial displays of emotion. CG participants reported more affect when discussing conflict with a living attachment figure than when discussing conflict with the deceased. Compared to healthy bereaved participants, CG participants also demonstrated greater negative emotion word use across conditions, fewer facial expressions of sadness when discussing conflict, and fewer facial expressions of happiness across conditions. Taken together, these studies suggest that individuals with CG do respond differently to deceased-related and attachment-related stimuli. However it is unclear whether individuals with CG are best characterized as hyper- or hypo-reactive to these stimuli, as the direction of the effect appears to shift depending on response channel. In addition, to our knowledge, no study has yet compared emotional or psychophysiological reactivity to ideographic death-related stimuli between bereaved individuals with and without CG.

4.3. Exploratory RSA analyses

Exposure to the sad and funny film clips reduced RSA relative to the neutral film clip among healthy bereaved participants, but not among those with CG. Phasic reductions in RSA reflect withdrawal of inhibitory vagal control of heart rate, resulting in increased physiological arousal. PNS withdrawal of inhibitory control is a relatively faster and more flexible mechanism for affecting cardiac arousal than is SNS excitatory influences (order of milliseconds vs. order of seconds; Thayer and Lane, 2000). Thus, phasic RSA suppression in response to emotional stimuli apparently reflects adaptive and flexible attention modulation in response to environmental demands (Thayer and Lane, 2000). This adaptive PNS response appears compromised in individuals with CG.

These results are consistent with research on aberrant PNS functioning in individuals with depression (Rottenberg et al., 2007; Yaroslavsky et al., 2013). And although there are no studies on PNS reactivity in CG, people with the disorder are less facially expressive (Diminich and Bonanno, 2014) and have more difficulty up- and down-regulating their facial expressions on command (Gupta and Bonanno, 2014) than do people without CG. Notably, the vagus nerve also has a branch that innervates the muscles of the face, thereby mediating facial expressions of emotion (Porges, 2011). Taken together, these results suggest that PNS-governed flexible emotional responding is impaired in those with CG.

4.4. Limitations

Our study has limitations. First, our study groups are modest in size. Our *a priori* power analysis indicated that 26 participants per group would enable detection of large effects in physiological reactivity between groups. However, recruitment challenges resulted in our CG group falling short of this target by three participants. Although our study may have been underpowered, the few previous studies that report on psychophysiological responding in bereaved adults have had even smaller diagnostic groups (e.g. Bonanno et al., 2007; O'Connor et al., 2002). In any event, we provided effect size estimates to aid interpretation of our results.

Second, unequal group sizes compromised full counterbalancing of the emotional film clip orders between two groups. Third, the emotional film clips may have been insufficiently potent for probing PNS reactivity (see Rottenberg et al., 2007).

4.5. Conclusions

These limitations notwithstanding, our study provides an important first look at patterns of emotional and physiological reactivity to generic emotional stimuli in individuals with CG. Overall, the results of this study suggest more similarities than differences in emotional and physiological responding between bereaved adults with and without the disorder. One exception may be attenuated PNS reactivity among those with CG – though our analyses of RSA reactivity were exploratory and require replication. Future studies exploring the emotional lives of individuals with CG are needed to deepen our understanding of the etiology and maintenance of the disorder.

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