OPEN

The Relationship of Prolonged Grief Disorder Symptoms With Hemodynamic Response to Grief Recall Among Bereaved Adults

Roman Palitsky, PhD, Da'Mere T. Wilson, MA, Sydney E. Friedman, BA, John M. Ruiz, PhD, Daniel Sullivan, PhD, and Mary-Frances O'Connor, PhD

ABSTRACT

Objective: Bereavement is among the most impactful psychosocial stressors for cardiovascular health, and hypertensive episodes accompanying bereavement-related distress are one putative mechanism for this effect. The present study examined hemodynamic responses to the Grief Recall (GR), a promising method for studying the effects of acute grief on cardiovascular function, and the relationship of grief severity to blood pressure (BP) response.

Methods: N = 59 participants within 1 year of the loss of a close loved one completed the GR, a semistructured interview protocol for eliciting bereavement-related distress (a "grief pang") and cardiovascular response. Systolic (SBP) and diastolic BP (DBP) were measured at two time points: a) an attention-control baseline and (2) after a 10-minute GR interview. Baseline versus post-GR SBP and DBP differences (i.e., BP response) were measured. Grief severity was examined as a predictor of SBP and DBP response, as well as BP recovery **Results:** SBP and DBP increased significantly after GR (SBP, +21.10 mm Hg; DBP, +8.10 mm Hg). Adjusting for variables relevant to cardiovascular function and bereavement (antihypertensive medication use, days since death, gender, age), grief severity predicted the magnitude of increase after GR in SBP but not DBP. No relationship of grief severity and recovery was observed.

Conclusions: The observed association between hemodynamic response and grief severity suggests a mechanistic contribution from hemodynamic effects of acute grief episodes to the cardiovascular impact of grief. This is the first study to show that increased symptoms of prolonged grief disorder are associated with an elevated SBP response. The GR may have further utility for research examining physiological responses to bereavement-related emotions.

Key words: bereavement, grief, blood pressure, prolonged grief disorder.

INTRODUCTION

E pidemiological research and meta-analyses have repeatedly shown that the psychophysiological toll of bereavement includes elevated risk of all-cause mortality (1), including an increase in cardiovascular risk (2,3). In addition to higher rates of cardiovascular events among bereaved individuals (4), biomarkers of cardiovascular risk such as elevated blood pressure (BP) are documented (5,6). What mechanisms might account for the link between bereavement and the cardiovascular risk of the surviving loved one? Bereavement-related emotions have been identified as a likely contributing factor (7). Elevated BP in response to emotional stressors has been well documented (8). Acute grief emotions, which can comprise sadness, anger, anxiety, and yearning, have been shown to induce neurological, hormonal, and sympathetic **BP** = blood pressure, **DBP** = diastolic blood pressure, **GR** = Grief Recall, **SAI** = State Anxiety Inventory, **SBP** = systolic blood pressure

activation (9), as well as elevated BP. Hemodynamic response to acute grief emotions, or "grief pangs," may be an important contributor to the cardiac risk profile of bereaved persons.

Nevertheless, grief researchers have not yet produced compelling evidence for a relationship between a continuous measure of grief severity (as opposed to the categorical variable of bereavement or a duration-based indicator like days since death) and cardiac risk mechanisms in bereavement. The inclusion of prolonged grief disorder in the *International Classification of Diseases, Eleventh Revision (ICD-11)* and *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition, Text Revision) (*DSM-5-TR*) (10) has been accompanied by reliable and valid measures of

ORCID IDs: 0000-0002-0415-6411 (R.P.); 0000-0001-5961-6350 (M.-F.O.).

SDC Supplemental Digital Content

From the Department of Psychiatry and Human Behavior and Spiritual Health (Palitsky), Warren Alpert Medical School of Brown University, Providence, Rhode Island; Department of Spiritual Health, Woodruff Health Sciences Center (Palitsky), Emory University, Atlanta, Georgia; and Department of Psychology (Wilson, Friedman, Ruiz, Sullivan, O'Connor), University of Arizona, Tucson, Arizona.

Address correspondence to Roman Palitsky, PhD, Warren Alpert Medical School of Brown University, 1440 Clifton Rd. Atlanta, GA, 30322. E-mail: roman.palitsky@emory.edu

Received for publication May 24, 2022; revision received February 24, 2023.

Article Editor: Julian F. Thayer

DOI: 10.1097/PSY.000000000001223

Copyright © 2023 The Author(s). Published by Wolters Kluwer Health, Inc. on behalf of the American Psychosomatic Society. This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

symptoms of grief severityⁱ. The present study examined changes in systolic and diastolic BP (SBP and DBP) in response to the Grief Recall (GR), an emotion elicitation paradigm, among individuals in the first year of bereavement, as well as the association of grief severity with changes in BP. We hypothesized that (H1) SBP and DBP would be elevated after the GR, demonstrating the BP effects of this grief-relevant emotion elicitation. We also hypothesized that (H2) grief severity would be positively associated with the magnitude of increase in SBP and DBP, demonstrating that grief severity in bereavement may be related to cardiovascular risk, and that the GR is a useful procedure for evaluating grief-related physiological response. To disambiguate the influence of biological determinants such as age, gender, and antihypertensive medication, as well as time since death, these variables were included as covariates in analyses. Physiological activity after a task may also constitute risk (11), although we did not have specific hypotheses about the association of recovery after GR with grief severity. To better understand cardiovascular recovery after GR, post-hoc exploratory analyses were conducted to examine BP recovery over time and its association with grief severity.

The GR is an adaptation of the Anger Recall and Separation Recall emotion elicitation paradigms for grief elicitation in bereavement. The Anger Recall is a widely used and effective way to recreate a moment of anger through a laboratory interview (12). Separation Recall, a further innovation on this paradigm, elicits emotion related to a participant's attachment-related memories of separation (13).

The GR protocol is similar in structure to the Anger Recall and Separation Recall, but focuses on bereavement-related emotion and is designed to stimulate "pangs of grief." Participants are asked to recall a time since the death of their loved one when they felt alone or abandoned. A standard set of follow-up questions further elicit grief-related feelings and memories (see Supplementary Document I, http://links.lww.com/PSYMED/A942 for GR script). Previous research using the GR found preliminary evidence that the paradigm elicited an elevation in BP, heart rate, and cortisol in a sample of 10 bereaved individuals (14). The current study examined the association between grief severity and BP response to the GR.

METHODS

Participants

Seventy-eight community participants were recruited for this study, between July 2018 and November 2019. Inclusion criteria were as follows: 18+ years old, recent bereavement (<1 year) of a close relative (spouse, sibling, parent, grandparent, child, or friend who was "like a relative"), and sufficient English fluency to provide informed consent for the study. Participants were recruited through advertisement in a local newspaper, direct contact with those who posted an obituary, outreach to community organizations, paper fliers, and a laboratory repository of recently bereaved persons, according to procedures established in this laboratory (15). Exclusion criteria were medical conditions that could interfere with the assessment of stress-related heart function, and either mental health or situational factors that would interfere with completion of the study. This sample was dictated by constraints on

the study timeline but was deemed adequate (n > 67) to detect a medium effect ($r_p > 0.33$) in linear regression with four covariates.

Procedures

Participants were initially screened for participation in the study over the telephone. Those who met the inclusion criteria completed a laboratory-based study consisting of two sessions spaced 14 to 21 days apart, in which participants were in one room and experimenters were in another room, observable by cameras. During the first session, participants provided informed consent for the study and completed survey questionnaires. In the second session, participants completed the GR and cardiovascular assessment as follows: at the start of this visit, a BP cuff was placed on the left arm. Then, participants completed questionnaires for approximately 20 minutes, allowing acclimation to the cuff. Participants then completed a 10-minute vanilla baseline assessment (16). During the vanilla baseline, participants were presented with 10 pairs of nature photographs on a computer screen, one pair for every minute of the baseline, and asked to select the photograph that they preferred in each pair. This task was intended to ensure that participants were alert, oriented, and exposed to consistent stimuli, without provoking strong emotional responses. After the end of the vanilla baseline, they completed the State Anxiety Inventory (SAI) (17), followed by the 10-minute GR task, followed by the SAI once more, followed by a 10-minute recovery period. BP was collected at 2-minute intervals during the vanilla baseline and after the GR (see BP Measurement for details). After the recovery period, participants completed a final set of questionnaires, followed by a debriefing. Participants were compensated for participating in the study, and all were offered local resources for bereavement support in the community, with an offer of follow-up discussion available for those who experienced any concerns. All procedures were approved by the University of Arizona institutional review board.

GR Procedure

The GR procedure is a one-on-one interview in which the interviewer asks the bereaved person to recall a time since the loss when he or she felt "alone and abandoned, and wished that your loved one had been there for you" (see Supplemental Digital Content 1, http://links.lww.com/PSYMED/A942 for GR script). Once the participants think of a relevant time, they are asked to describe that event, with a standardized set of semistructured interview questions intended to maintain the participants' focus on the specific moment that they named (e.g., "While you were going through that painful situation, do you remember what you were focused on?"). The GR interview lasts 10 minutes; after the GR interview, the participants were asked to sit quietly with their eyes open and their legs not crossed (to reduce influence on recovery BP measures), with as little movement as was comfortable, for the recovery period that followed. Interviewers (R.P. & D.T.W.) were clinical psychology graduate students supervised by a licensed clinical psychologist.

Measures

Participants reported their age, gender, and ethnicity (Asian American, African American, Latino/Hispanic, Native American, White [non-Hispanic], or other [free response]). Gender categories included male, female, nonbinary, and a free response option. Supplemental Digital Content 2, http://links.lww.com/PSYMED/

Prolonged grief disorder can only be diagnosed greater than a year after the death event in the *DSM-5-TR* and 6 months in the *ICD-11*. Thus, symptom severity was used in the present study, reflecting the graded response of grief severity.

Hemodynamic Response to Grief Recall

A943, includes the description of the sample for all gender categories, but the nonbinary and missing data were excluded for analyses, for statistical power considerations. Participants reported the deceased's relationship to the participant (e.g., parent, spouse, child, friend who was "like family"), and the date of the death. Time since the death was calculated as the number of days between the death of the loved one and the date of the first study visit.

Grief severity was assessed with the Prolonged Grief-13 Scale (18). This scale is composed of 13 items that assess the frequency, duration, and extent of symptoms associated with prolonged grief, as well as impairment due to these symptoms. The present study summed the 11 Likert-type responses, with higher scores indicating greater grief severity (1 = not at all to 5 = several times a day, or 1 = not at all to 5 = overwhelmingly), Cronbach α = .89. The remaining two items are dichotomous yes/no questions assessing impairment (reduction in functioning) and frequency (at least daily), and are not used when obtaining a continuous score. A sample item from this scale is as follows: "In the past month, how often have you felt stunned, shocked, or dazed by your loss?"

The SAI was used as a manipulation-check affect measure. This 20-item scale consists of the state portion of the State-Trait Anxiety Inventory and was used as an index of anxious distress. It was given to participants before and after the GR (pre-GR, $\alpha = .68$; post-GR, $\alpha = .55$).

Use of medications with antihypertensive effects was assessed by asking participants to indicate (yes/no) whether they take medication: a) "for a heart condition"; b) "for BP, or blood thinners (anticoagulants)"; and c) "to regulate anxiety." If they responded affirmatively, they were asked what medications they take for each purpose (e.g., "what is the medication you are taking for a heart condition?"). These medications were examined for antihypertensive effects. Antihypertensive medication use was coded dichotomously, with use coded as "1" and nonuse coded as "0." (Medication use was also computed additively as an alternative, with different classes (angiotensin-converting-enzyme inhibitors, β-blockers, and statins) each obtaining a score of 1. These were then summed to create a total score to account for additive effects. However, dichotomous values were used in analyses because alternate analyses with sum scores for medication instead of a dichotomous covariate did not meaningfully change the reported results.

BP Measurement

BP was assessed with GE Dinamap Pro 100 BP Monitors, which provided measures of SBP and DBP used in these analyses. After acclimating to the BP cuff for 20 minutes while answering study questionnaires, participants completed a 10-minute vanilla baseline (16) during which BP was measured every 2 minutes. Once the vanilla baseline ended, they completed the SAI, followed directly by the 10-minute GR task. BP was not measured during GR to not distract participants. BP was measured again directly after the end of GR, whereas participants completed the SAI a second time. For 10 minutes after the end of GR, BP was measured once every 2 minutes. During this time, participants were asked to sit still with their eyes open and legs not crossed.

Baseline values for SBP and DPB were calculated by averaging the final two baseline measurements (taken at minutes 8 and 10 for all participants except for two, for whom, because of error readings in the BP monitor, the last two baseline readings that were available for each of these two participants were used, which included BP measurements taken before minute 8 of the baseline) per field recommendations (19,20). The BP measure taken directly after GR was used in the calculation of change scores between baseline versus post-GR BP. This was the first available taskdependent BP measure, because BP was not assessed during GR to avoid distracting participants. As this was the most proximate measure to the task, this was the only measure used to calculate post-GR BP reactivity. Some participants continued speaking in response to the GR task after the end of 10 minutes. For these participants, the first BP reading after they finished speaking was used as the first post-GR BP measure (i.e., the reading 2 minutes after the end of GR instead of directly at the end of GR). Raw change scores were computed by subtracting baseline values from post-GR values; residualized change scores were taken by regressing post-GR BP measures onto baseline BP measures and saving the unstandardized residuals. BP recovery was operationalized as the difference between baseline and recovery measures of BP at 2, 4, 6, 8, and 10 minutes post-GR.

Analyses

Analyses were conducted in SPSS 27 (IBM). To examine BP response to the GR, repeated-measures analyses of variance compared BP scores at baseline and at 0, 2, 4, 6, 8, and 10 minutes post-GR. As a manipulation check, scores on the SAI were also compared using paired *t* tests. To assess for selective attrition, participants whose data were not available for analyses were compared with those included in the study on age, days since loss, and grief severity via independent-samples *t* tests, and compared on gender via a χ^2 test.

To examine the associations between grief severity and pre-post GR BP change, the associations between grief severity and change in SBP and DBP were examined in separate linear regression analyses with percentile bootstrapping (5000 iterations), using residualized change scores (Δ SBP_{resid} and Δ DBP_{resid}) to adjust for baseline BP values (analyses with raw change scores were also conducted and are available in Supplemental Digital Content 2, http://links.lww.com/PSYMED/A943). Both analyses adjusted for age, gender, time since death, and antihypertensive medication use as covariates. Because the sample only included five Hispanic/Latino/a participants (all others identified as non-Hispanic White), race/ethnicity was not included as a covariate.

Post-hoc exploratory analyses evaluated BP recovery after GR, as well as its association with grief severity, using repeatedmeasures analyses of covariance for SBP and DBP values at minutes 2, 4, 6, 8, and 10 of the recovery period. Because task-related increase in BP is often an independent predictor of recovery, consistent with common practices in the BP recovery measurement (21,22), analyses adjusted for baseline BP and raw BP reactivity, in addition to the covariates used in primary reactivity analyses. Grief severity was included as a covariate to evaluate its effect on recovery over time. Study data are available from corresponding author on request.

RESULTS

Seventy-eight participants were recruited for this study. Seventyfive participants consented to participate in the study. After enrollment, one participant withdrew and one participant was excluded after enrollment because he or she reported a loss type that did not meet the study criteria. Among the remaining participants, 73 participated in session 1 of the study and 64 returned for session 2, which included the GR interview. Among session 2 participants, two found the BP cuff to be painful and completed GR without it, 2 deviated from the GR procedure (e.g., taking a telephone call in the middle), and for one participant, the BP measurement device reported an error and did not provide data. Ultimately, complete BP data from 59 participants were available for the present analyses. Comparison between participants whose data were versus were not available for analyses did not reveal differences in gender ($\chi^2 = 0.03, p = .864$), age (t(71) = 1.18, p = .255), days since loss (t(71) = 0.25, p = .805), or grief severity (t(71) = -0.61, p = .546). See Supplemental Digital Content 2, http://links.lww.com/PSYMED/A943, for participant demographics.

To examine differences between BP measurements at baseline, immediately after GR, and at minutes 2, 4, 6, 8, and 10 of recovery, repeated-measures analyses of variance were used. Mauchly tests of sphericity were significant for SBP and DBP (p values < .001), and Huynh-Feldt corrections were applied. Within-subject tests were significant for SBP ($F(4.50,238.32) = 37.63, p < .001, \eta_p^2 =$ 0.42) and DBP ($F(4.46,236.23) = 17.20, p < .001, \eta_p^2 = 0.25$). Lending support for H1, significant increases were observed in SBP from baseline (mean [standard error], or M [SE] = 124.32 [15.01] mm Hg) to immediately post-GR (mean [standard deviation], M [SD] = 145.43 [25.17], p < .001, 95% confidence [CI] = 16.68–25.52). DBP also increased from baseline (M [SD] = 69.05 [8.47]) to immediately post-GR (M [SD] = 77.15 [10.67], p < .001, 95%CI = 5.87-10.34). This corresponded with a mean increases in SBP of 21.10 mm Hg and DBP of 8.10 mm Hg. Pairwise comparisons also revealed that compared with baseline, elevated SBP and DBP were sustained at minutes 2, 4, 6, 8, and 10 of recovery (p values < .001). Nevertheless recovery did take place, such that all SBP and DBP recovery values were lower than the measure taken immediately after GR (p values < .01; see Supplemental Digital Content 2, http://links.lww.com/PSYMED/A943 for details).

Paired *t* tests also revealed increases in SAI scores from baseline (M = 30.95) to post-GR (M = 40.00, t(58) = 6.53, p < .001, 95% CI = 6.34–11.80, d = 0.85), indicating that the GR task contributed to an increase in anxious distress.

Regression analyses revealed significant associations between baseline grief severity and ΔSBP_{resid} (B = 0.447, SE = 0.215, p = .042, 95% CI = 0.024 to 0.871). No significant association was observed between grief severity and ΔDBP_{resid} (B = 0.074, SE = 0.127, p = .55, 95% CI = -0.180 to 0.319; Table 1), lending support for H2 for SBP but not DBP. Analyses with raw (nonresidualized) change scores revealed the same pattern of results, available in Supplemental Digital Content 2, http://links.lww. com/PSYMED/A943. Figure 1 represents results of regression analyses using raw change scores for ease of interpretation.

Recovery

Post-hoc repeated-measures analyses of covariance were used to examine the association of grief severity with BP recovery at minutes 2, 4, 6, 8, and 10, with baseline and reactivity BP, time since death, gender, antihypertensive medication, and grief severity included as covariates. Mauchly test revealed that sphericity had been violated for SBP and DBP (*p* values < .05) and Huynh-Feldt corrections were applied. No significant within-subject effects of time on recovery of SBP (*F*(4,176) = 1.345, *p* = .255, $\eta_p^2 = 0.03$) or DBP were observed (*F*(4,176) = 0.87, *p* = .47,

TABLE 1.	Regression	Analyses	for	Predictors	of	ΔSBP_{resid}
and ΔDBP_{resid}						

				95% CI		Model
Variable Name	В	SE	р	LL	UL	R^2
Predictors of ΔSBP_{resid}						0.18
Grief severity	0.447	0.215	0.042	0.024	0.871	
Age	0.277	0.123	0.025	0.040	0.525	
Gender	0.863	4.904	0.87	-9.050	9.901	
BP medication	5.095	4.963	0.31	-5.230	14.431	
Days since death	0.008	0.025	0.74	-0.044	0.057	
Predictors of ΔDBP_{resid}					0.12	
Grief severity	0.074	0.127	0.55	-0.180	0.319	
Age	0.083	0.061	0.17	-0.042	0.196	
Gender	4.436	2.524	0.085	-0.400	9.532	
BP medication	2.537	2.744	0.36	-3.065	7.716	
Days since death	0.006	0.013	0.63	-0.022	0.030	

 $\Delta SBP_{resid} = residualized difference of baseline systolic blood pressure versus post-Grief Recall systolic blood pressure, such that higher scores indicate greater increase from baseline to post-GR values; <math>\Delta DBP_{resid}$ = residualized difference of baseline versus post-GR diastolic blood pressure, computed analogously with ΔSBP ; B = unstandardized regression coefficient; SE = standard error; CI = confidence interval, with LL and UL indicating lower and upper confidence interval limits, respectively; Grief severity = Prolonged Grief-13 Scale sum score; Gender = dichotomous variable for gender with male as reference category; BP medication = use of anthypertensive medication; Days since Death = days elapsed since the death of the participant's loved one.

 $\eta_p^2 = 0.02$), suggesting that time was not a significant predictor of recovery values when adjusting for other covariates. No effect of time by grief severity was observed for SBP (*F*(4,176) = 1.65, p = 1.63, $\eta_p^2 = 0.04$) or DBP (*F*(4,176) = 0.37, p = .810, $\eta_p^2 = 0.01$), failing to provide evidence that grief severity was not associated with recovery over time.

DISCUSSION

Our findings contribute evidence that SBP and DBP increase after the GR in bereaved participants, consistent with prior findings (14), accompanied by an increase in state anxiety. Furthermore, this is the first study, to our knowledge, to demonstrate an association between greater baseline grief severity and an elevated SBP response to a standardized laboratory-based interview, although no associations with recovery were observed in post-hoc analyses. GR thus shows promise as an emotion elicitation protocol for studying the impact of grief severity on cardiovascular functioning.

Previous work has shown an association between bereavement (without assessing grief severity) and elevated SBP as assessed by 24-hour ambulatory BP monitoring (5). The ambulatory monitoring study did not observe bereaved/nonbereaved differences in DBP, and although the present study observed post-GR differences in SBP and DBP, it also did not observe an association between grief severity and DBP. SBP response may be more sensitive to emotions that elicit acute sympathetic activation than DBP response, possibly because of greater dependence of SBP on stroke volume, and of DBP on peripheral resistance (23).

Prior literature examining SBP response to psychological stressors suggests, on one hand, that magnitude of response may predict subsequent problems such as hypertension (24), but on

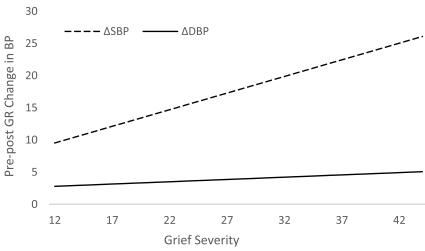


FIGURE 1. Association of grief severity with change in BP after Grief Recall. This figure represents predicted values of change in raw SBP and DBP from pre- to post-GR across observed grief severity scores. Grief severity = Prolonged Grief-13 sum scores; observed values are represented on the *x* axis. For Δ SBP and Δ DBP, positive values indicate an increase in blood pressure. Reference group for this figure include male participants who are not taking antihypertensive medication at observed sample averages of days since death (151.37) and age (65.80). BP = blood pressure; GR = Grief Recall task. Δ SBP = pre-post GR raw change in systolic blood pressure. Δ DBP = pre-post GR change in diastolic blood pressure.

the other hand, a case may be made that a robust SBP response can be adaptive. In patients with heart failure, *lower* BP reactivity was associated with a greater risk of adverse CVD events and mortality (25,26). Wright et al. (27) found that *increased* systolic reactivity in response to an acute stressor was associated with greater subjective well-being. Our findings suggest the opposite in the case of bereavement, with grief severity associated with greater SBP response. The nature of the context and stress of bereavement are likely relevant for understanding the pattern of SBP response. Namely, Kupper et al. (25), Sherwood et al. (26), and Wright et al. (27) used standardized stressors, to which a robust response may indicate health and flexibility. In bereavement, the difference in the nature of the stressor (i.e., loss is constantly present and difficult to resolve) may explain the association of greater physiological response with greater grief severity.

In addition to reactivity, it may be important to investigate BP recovery after GR further (11). Our study observed that SBP and DBP recovery occurred (i.e., SBP and DBP values taken at 2 or more minutes after GR were diminished, compared with values immediately after GR), but that recovery over time was not associated with grief severity. However, this study was not able to examine features of recovery that may be important in bereavement, such as time to recovery from baseline. Because duration of heightened BP may be associated with the a) ambulatory BP (22), b) style of emotional response (28,29), and c) psychological and health outcomes (11,30), BP recovery after GR would benefit from further study in paradigms further tailored for evaluating this outcome.

Antihypertensive medications are currently being explored as preventative interventions for cardiovascular risk in bereavement (14,31). Our results suggest that cardiovascular response in bereavement is varied and that variability in BP response to acute emotional stressors may be assessed by means of the GR. Such procedures may be used to better understand which patients might most benefit from preventative pharmacological interventions. Furthermore, because grief severity is implicated in the magnitude of SBP response, potential psychological treatments such as grief therapy should be investigated as ways to address the emotional antecedents of cardiovascular risk in bereavement by nonpharmacological means.

Several limitations constrain the interpretation of this study. The racial and ethnic makeup, as well as the size, of the sample limit the generalization of these findings. Research suggests that cultural differences correspond with variation in the experience and expression of grief (32,33); cultural differences in response to the GR should be investigated in subsequent research. In addition, this study recruited participants who experienced the loss of a close loved one, constituting different types of loss. Subsequent research focusing on specific types of loss (e.g., widowhood) or recruiting a large enough sample to enable comparisons across loss types would be valuable for understanding unique effects of the GR for different loss types.

It remains to be seen whether greater SBP response to GR predicts worse cardiovascular outcomes. Prospective research on cardiovascular risk that uses the GR with bereaved participants would help to establish whether, as with heart failure patients, reactivity may predict better outcomes or, as with healthy populations, magnitude of response would predict greater risk. These findings also suggest that the frequency with which bereaved individuals think of the deceased such that they feel alone and abandoned may be associated with cardiovascular impacts of bereavement. The effect of this daily-life variable should be explored in further studies of cardiovascular risk in bereavement. The GR is a feasible laboratorybased protocol for conducting such research, and because elevations in SBP are associated with grief severity, it may be especially appropriate for assessing bereavement-related distress.

Source of Funding and Conflicts of Interest: This work was supported by the Association for Assessment and Research in Counseling and the Society for the Scientific Study of Religion. The authors declare no conflicts of interest.

REFERENCES

- Shor E, Roelfs DJ, Curreli M, Clemow L, Burg MM, Schwartz JE. Widowhood and mortality: a meta-analysis and meta-regression. Demography 2012;49:575–606.
- Buckley T, McKinley S, Tofler G, Bartrop R. Cardiovascular risk in early bereavement: a literature review and proposed mechanisms. Int J Nurs Stud 2010;47:229–38.
- Carey IM, Shah SM, DeWilde S, Harris T, Victor CR, Cook DG. Increased risk of acute cardiovascular events after partner bereavement: a matched cohort study. JAMA Intern Med 2014;174:598–605.
- Mostofsky E, Maclure M, Sherwood JB, Tofler GH, Muller JE, Mittleman MA. Risk of acute myocardial infarction after the death of a significant person in one's life: the Determinants of Myocardial Infarction Onset Study. Circulation 2012; 125:491–6.
- Buckley T, Mihailidou AS, Bartrop R, McKinley S, Ward C, Morel-Kopp MC, et al. Haemodynamic changes during early bereavement: potential contribution to increased cardiovascular risk. Heart Lung Circ 2011;20:91–8.
- Prigerson HG, Bierhals AJ, Kasl SV, Reynolds CF 3rd, Shear MK, Day N, et al. Traumatic grief as a risk factor for mental and physical morbidity. Am J Psychiatry 1997;154:616–23.
- Schwartz BG, French WJ, Mayeda GS, Burstein S, Economides C, Bhandari AK, et al. Emotional stressors trigger cardiovascular events. Int J Clin Pract 2012;66: 631–9.
- Chida Y, Steptoe A. Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: a meta-analysis of prospective evidence. Hypertension 2010;55:1026–32.
- Norcliffe-Kaufmann L, Kaufmann H, Martinez J, Katz SD, Tully L, Reynolds HR. Autonomic findings in Takotsubo cardiomyopathy. Am J Cardiol 2016;117:206–13.
- Prigerson HG, Kakarala S, Gang J, Maciejewski PK. History and status of prolonged grief disorder as a psychiatric diagnosis. Annu Rev Clin Psychol 2021; 17:109–26.
- Panaite V, Salomon K, Jin A, Rottenberg J. Cardiovascular recovery from psychological and physiological challenge and risk for adverse cardiovascular outcomes and all-cause mortality. Psychosom Med 2015;77:215–26.
- Ironson G, Taylor CB, Boltwood M, Bartzokis T, Dennis C, Chesney M, et al. Effects of anger on left ventricular ejection fraction in coronary artery disease. Am J Cardiol 1992;70:281–5.
- Ehrenthal JC, Friederich HC, Schauenburg H. Separation recall: psychophysiological response-patterns in an attachment-related short-term stressor. Stress Health 2011;27:251–5.
- Karl S, Fallon M, Palitsky R, Martinez JA, Gündel H, O'Connor MF. Low-dose aspirin for prevention of cardiovascular risk in bereavement: results from a feasibility study. Psychother Psychosom 2018;87:112–3.
- Stelzer EM, Knowles LM, Wilson DT, O'Connor MF. Recruitment and retention in clinical and experimental bereavement research: lessons learned from creating a research registry. Death Stud 2020;44:771–7.

- Jennings JR, Kamarck T, Stewart C, Eddy M, Johnson P. Alternate cardiovascular baseline assessment techniques: vanilla or resting baseline. Psychophysiology 1992;29:742–50.
- Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press. 1983.
- Prigerson HG, Horowitz MJ, Jacobs SC, Parkes CM, Aslan M, Goodkin K, et al. Prolonged grief disorder: psychometric validation of criteria proposed for DSM-V and ICD-11. PLoS Med 2009;6:e1000121.
- Shapiro D, Jamner LD, Lane JD, Light KC, Myrtek M, Sawada Y, et al. Blood pressure publication guidelines. Psychophysiology 1996;33:1–12.
- TRUE Consortium. Recommended standards for assessing blood pressure in human research where blood pressure or hypertension is a major focus. Kidney Int Rep 2017;2:733–8.
- Linden W, Earle TL, Gerin W, Christenfeld N. Physiological stress reactivity and recovery: conceptual siblings separated at birth? J Psychosom Res 1997;42:117–35.
- Trivedi R, Sherwood A, Strauman TJ, Blumenthal JA. Laboratory-based blood pressure recovery is a predictor of ambulatory blood pressure. Biol Psychol 2008; 77:317–23.
- Rüddel H, Langewitz W, Schächinger H, Schmieder R, Schulte W. Hemodynamic response patterns to mental stress: diagnostic and therapeutic implications. Am Heart J 1988;116(2 Pt 2):617–27.
- Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, Zhu S, et al. Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. Circulation 2004;110:74–8.
- Kupper N, Denollet J, Widdershoven J, Kop WJ. Cardiovascular reactivity to mental stress and mortality in patients with heart failure. JACC Heart Fail 2015;3:373–82.
- Sherwood A, Hill LK, Blumenthal JA, Adams KF Jr., Paine NJ, Koch GG, et al. Blood pressure reactivity to psychological stress is associated with clinical outcomes in patients with heart failure. Am Heart J 2017;191:82–90.
- Wright BJ, O'Brien S, Hazi A, Kent S. Increased systolic blood pressure reactivity to acute stress is related with better self-reported health. Sci Rep 2014;4:6882.
- Fredrickson BL, Maynard KE, Helms MJ, Haney TL, Siegler IC, Barefoot JC. Hostility predicts magnitude and duration of blood pressure response to anger. J Behav Med 2000;23:229–43.
- Gerin W, Davidson KW, Christenfeld NJ, Goyal T, Schwartz JE. The role of angry rumination and distraction in blood pressure recovery from emotional arousal. Psychosom Med 2006;68:64–72.
- Steptoe A, Marmot M. Psychosocial, hemostatic, and inflammatory correlates of delayed poststress blood pressure recovery. Psychosom Med 2006;68:531–7.
- Tofler GH, Morel-Kopp MC, Spinaze M, Dent J, Ward C, McKinley S, et al. The effect of metoprolol and aspirin on cardiovascular risk in bereavement: a randomized controlled trial. Am Heart J 2020;220:264–72.
- Stelzer EM, Zhou N, Maercker A, O'Connor MF, Killikelly C. Prolonged grief disorder and the cultural crisis. Front Psychol 2019;10:2982.
- Wilson DT, O'Connor MF. From grief to grievance: combined axes of personal and collective grief among Black Americans. Front Psychiatry 2022;13:850994.