

Recreating cardiovascular responses with rumination: The effects of a delay between harassment and its recall

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Abstract

Cardiovascular responses occur not only in the immediate presence of stressors, but also while later thinking about those experiences. Evidence suggests that these delayed responses, such as those produced by ruminating about prior angering experiences, may play an important role in the development of cardiovascular disease. We examine whether physiological consequences of rumination depend on the delay between a stressor and its recall, and whether the magnitude of physiological responses decreases with repetition. Twenty-two participants experienced a three-minute harassment stressor, and later spent 3 min vividly recalling the task. Half the subjects returned for the first time after a week, and half returned after half an hour, and then also after a week. Blood pressure and heart rate were monitored during a baseline period, and during each session's stressor or rumination period. Results indicated that rumination was sufficient to elevate blood pressure (systolic and diastolic) above baseline, that the delay made no difference to the magnitude of the elevation, but that the second rumination seemed to be associated with a smaller response than the first. Response to the stressor was not associated with rumination responses, but the first rumination response was significantly correlated with the second. The effects of stress may be experienced long after the actual stressor is passed, and people who experience large delayed responses may not be the same as those with high initial responses. The "hot" affective portion of rumination may not be diminished by the passage of time, but by prior recreation of the experience.

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Recent work examining the role of cardiovascular responses to stress in CVD morbidity and mortality has started to incorporate a broader characterization of the cardiovascular stress response than previously used. It is becoming increasingly recognized that it is valuable to examine cardiovascular states not only during stressors, but also during recovery from stressors (Christenfeld et al., 2000; Gerin et al., 1994; Gregg et al., 1999; Haynes et al., 1991; Hocking-Schuler and O'Brien, 1997; Linden et al., 1997; Mezzacappa et al., 2001). Post-stress cardiovascular recovery profiles appear to differ depending on family history of hypertension and CHD (Borghi et al., 1986; Gerin and Pickering, 1995; Hocking-Schuler and O'Brien, 1997; Sheffield, Davy Smith, Carroll, Shipley and Marmot, 1997) and also seem to depend on factors such as race (Anderson

et al., 1989; Jackson et al., 1999; Mills and Berry, 1999) and certain personality traits including Type A and anger expression style (Faber and Burns, 1996; Fang and Myers, 2001; Jamieson and Lavoie, 1987; Lai and Linden, 1992).

The concept of recovery from a stressor need not be narrowly defined as the lingering physiological activation immediately following the stressor. Even after a return to baseline cardiovascular levels, the mental recreation of a stressful or anger-provoking event hours, days or even years later can produce a physiological response. Research examining the physiological components of emotion provides some evidence that these later recalls are associated with physiological activation. Many of these studies use the recall of a prior, emotionally-laden experience as an emotion-induction tool to examine the physiological responses associated with particular emotions, and these studies demonstrate that the act of recalling an emotional experience is indeed associated with increases in blood pressure, heart rate and

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galvanic skin response (de Jong-Meyer et al., 1993; Ekman et al., 1983; Schwartz et al., 1981; Stemmler, 1989). Anger recall appears to be a particularly potent elicitor of concomitant increases in blood pressure. Angry ruminations are consistently associated with physiological responses and appear to impede cardiovascular recovery following provocation (Brosschot and Thayer, 1998; Janssen et al., 2001; Lavoie et al., 2001; Neumann et al., 2004; Prkachin et al., 2001; Suchday et al., 2004).

To date, one study using the cardiovascular reactivity paradigm has examined the cardiovascular response to both a stressor and to the later recall of that same stressor. Glynn et al. (2002) exposed participants to either emotional or non-emotional stressors, and then, after a short delay, asked them to recall the experience as vividly as possible. Only the emotional tasks were associated with increases in blood pressure during recall. The size of the response during recall did not depend on whether the initial emotional stressor produced a high or low cardiovascular reactivity response.

The Glynn et al. (2002) study indicates that the emotional nature of the stressor is one important parameter determining whether later recall of a stressful event produces a physiological response. Another parameter that may be important is the delay between the initial stressor and the recall of, or rumination about, that stressor. The relation between the delay and the response at recall is important because it may shed light on the viability of rumination as a pathway for the development of CVD. If the passing of an hour is enough to extinguish the associated physiological response upon reflection, then rumination-associated cardiovascular responses would seem less important in the development of CVD. If, however, even after a considerable delay, it is still possible, simply by thinking about the prior angering event, to produce a significant cardiovascular response, then the role of angry rumination as a risk factor for CVD would seem more plausible.

Foster and Webster (2001) examined the relation between physiological responses to an emotion-recall task, and the age of the emotional memory recalled. Specifically, participants were instructed to identify an incident that had made them angry in the past (the average age of the memory was 2.1 years) and their heart rate and galvanic skin responses were recorded during the directed recall of this memory. The results indicated that older memories were associated with larger galvanic skin responses, but not heart rate responses. The authors suggested that the physiological response to the recall of an emotional memory increases with time. However, due to the correlational nature of the study, it is possible that the older memories were of more traumatic events, or that people who chose older events were people who generally show greater responses. This work does show, however, that memories from quite far back are still capable of eliciting some physiological response, although the precise effects of delay, and the cardiovascular concomitants of rumination, are not fully elucidated.

The purpose of the present study is to explore, experimentally, the relation between the delay to recall of an anger-provoking stressor and the cardiovascular response to that recall. Participants were brought into the laboratory and exposed to a harassment stressor. For the second visit, half of the subjects

returned 30 min later, and half 1 week later for the recall task. In addition, those that recalled the task at 30 min also recalled a second time at 1 week. This design allows the assessment of the effect of a delay on the physiological response at recall. It also allows us to explore the relation between the size of the initial cardiovascular response to the stressor and the size of the response at recall, as well as the effect of repeated recall on cardiovascular responses.

1. Method

1.1. Overview

On the first visit to the laboratory, all participants completed a mental arithmetic task with harassment while their blood pressure and heart rate were monitored. On the second visit to the laboratory, participants were asked to recall as vividly as possible the arithmetic task they had experienced on the previous visit. Half of the subjects (those in the Immediate condition) returned to the laboratory for the recall task 30 min after the first session. The other half (Delayed condition) returned exactly 1 week later. Those in the Immediate condition also returned 1 week later, and performed the recall task a second time.

1.2. Subjects

Participants included 9 males and 13 females at a large university. The mean age was 20.1 years and none of the participants reported having conditions or taking medications that might affect the dependent variables of interest. Participants received course credit in exchange for participation.

1.3. Physiological monitoring

Systolic blood pressure, diastolic blood pressure, and heart rate were collected using an Ohmeda Finapres 2300 blood pressure monitor. This instrument takes beat-to-beat pressures in a non-invasive manner, using the Penaz method. The participant wears an inflatable finger cuff on the third finger of the non-dominant hand. The Finapres has been demonstrated to be a useful alternative to intra-arterial blood pressure measurement in laboratory testing and clinical practice (Gorback et al., 1999; Imholz et al., 1990; Wieling et al., 1991). In addition, it has been shown to track intra-arterial readings extremely well, even during sudden changes of blood pressure, making it a good candidate for use during reactivity testing (Parati et al., 1989).

1.4. Procedure for the each study session

At each visit participants were told they would perform a simple task while their blood pressure and heart rate were monitored. The cuff of the Finapres monitor was attached to the middle finger of the non-dominant hand and a six-minute baseline was taken. Participants were instructed to sit quietly and relax during this period, and the experimenter left the room. Following the baseline period, the participants were given instructions for one of the two tasks, either the mental arithmetic

with harassment, or the directed recall, depending on whether it was the first or a later visit (see below).

1.5. Stressor task: mental arithmetic with harassment

On the initial visit to the laboratory, participants were asked to count backwards as quickly and accurately as possible by thirteen starting from the number 2036. While the participants counted backwards, the experimenter repeatedly harassed and interrupted the participant. The timing and content of these interruptions were standardized and independent of the participants' performances. These included comments such as "OK, you are going to have to start again and this time I will let you count by sevens so it is easier for you" and "I am going to start you one more time and if you do not speed up, I am not going to be able to use your data". Negative verbal feedback or harassment of this sort has been consistently been shown to elicit feelings of anger and to produce an acute cardiovascular response (Janssen et al., 2001; Lavoie et al., 2001, Glynn et al., 2002).

1.6. Recall task

The return visits to the laboratory (at 30 min and 1 week for the Immediate group and 1 week for the Delayed group) took place in the same room and with the same experimenter as the original task. The subjects who were to return after 30 min were told that they could do whatever they liked outside the lab – homework, socializing and the like – but should be back in half an hour. The Delayed subjects were reminded that they had an appointment to return to the laboratory after 1 week before they were dismissed. The Immediate subjects were similarly reminded that they were due back in 1 week after they finished their first recall session. On the return visits to the laboratory, participants were told: "What I would like you to do for the next three minutes is try to recreate the previous task in your head the best that you can. Try to remember all of the details as vividly as possible. It is very important that you focus on exactly how you felt for the whole three minutes. Pretend you are going through the entire experience in your mind. Remember this is going to take place entirely in your mind, so do not move around at all". The experimenter remained in the room during this rumination period and busied herself with paperwork. This was done to control for the effects of the experimenter's presence on blood pressure levels during recall, since she had been in the room during the original stressor task. Following the completion of each recall task, participants rated how detailed the mental recreation of the event had been on a seven-point Likert-type scale with the end points "not at all" and "extremely."

1.7. Data reduction and analysis procedures

Three cardiovascular variables were examined: systolic blood pressure, diastolic blood pressure, and heart rate. Analyses of change scores were based on the difference between the mean of the period of interest and the mean of the baseline period for that session. Raw, rather than residualized,

change scores were used, as recommended by Llabre, Spitzer, Saab, Ironson and Schneiderman (1991). All blood pressure means were computed using the pulse-based technique (Glynn et al., 1997).

2. Results

2.1. Visit 1: mental arithmetic stressor

The average systolic blood pressure during baseline was 106.2 mm Hg, the diastolic blood pressure was 65.9 mm Hg and the average heart rate was 77.5 bpm. There were no significant differences in baseline levels of blood pressure or heart rate between the groups (all $t(20)$'s < .36; p 's > .73).

The mental arithmetic stressor task produced sizeable blood pressure and heart rate responses. The increases from baseline for the groups are shown in Fig. 1. The mean increase for the Immediate group was 28.5 mm Hg for systolic blood pressure, 16.1 mm Hg for diastolic blood pressure and 18.7 bpm for heart rate. The average increase for the Delayed group was 26.8 mm

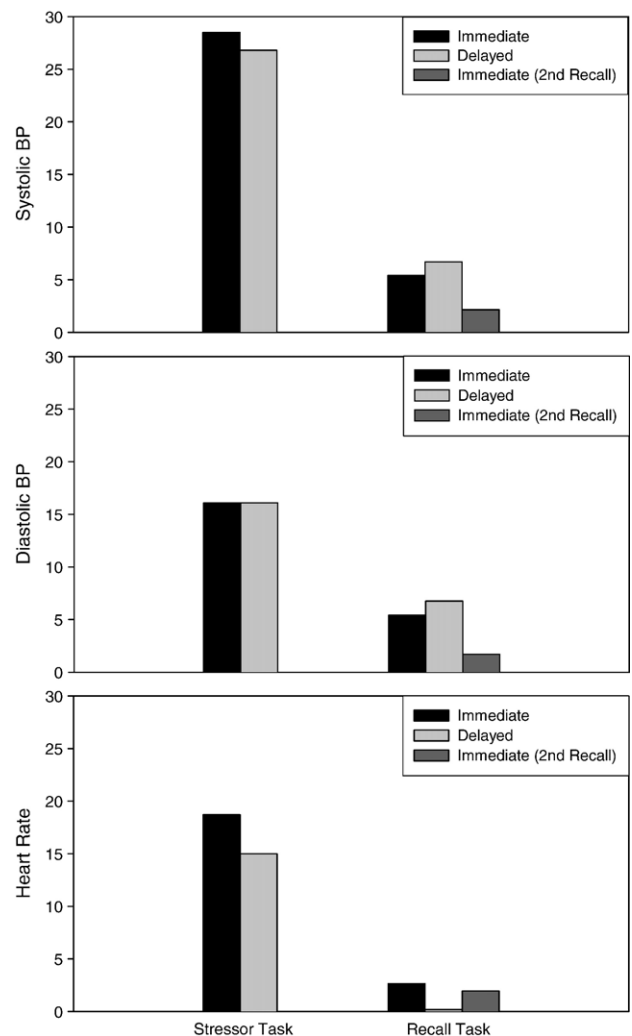


Fig. 1. Mean increase (from baseline) in systolic blood pressure, diastolic blood pressure and heart rate during the stressor and recall tasks for the two study groups.

Hg for systolic and 16.1 mm Hg for diastolic blood pressure, and 15.0 for heart rate. Repeated measures ANOVAs with period (baseline vs task) as the within-subjects factor, group (Immediate vs Delayed) as the between-subjects factor and blood pressure or heart rate levels as the dependent variables were conducted. These analyses revealed main effects of period (all $F(1,20)$'s > 68.73 ; p 's $< .01$) but no main effects of group (all $F(1,20)$'s < 0.32 ; p 's $> .58$) and no significant interaction terms (all $F(1,20)$'s $< .84$; p 's $> .37$) for systolic and diastolic blood pressures and heart rate.

2.2. Visit 2: recall task

There were no differences in baseline physiological measures for the groups upon return for the second session (all $t(20)$'s < 1.43 and p 's $> .17$). The mean systolic blood pressure at the second visit was 112.6 mm Hg, the mean for diastolic was 68.6 mm Hg, and the mean was 77.8 bpm for heart rate.

Fig. 1 shows the mean increases from baseline for the recall task for those who returned 30 min after the mental arithmetic task and those who returned 1 week later. Both groups showed increases in systolic and diastolic blood pressures during recall. Repeated measures ANOVAs with group (Immediate vs Delayed) as the between-subjects factor and period (baseline vs recall task) as the within-subjects factor revealed main effects of period for both systolic and diastolic blood pressure levels (both $F(1,20)$'s > 5.79 , p 's $< .05$) confirming that the rumination task produced significant increases in blood pressure. There were no main effects of experimental group on blood pressure levels (both $F(1,20)$'s < 1.74 , p 's $> .20$) and no significant interactions (both $F(1,20)$'s $< .53$, p 's $> .48$). Heart rate levels did not appear to be affected by the rumination task. There was no effect of period for this dependent measure ($F(1,20) = .16$, $p > .69$).

While the rumination task did elevate blood pressure levels, they clearly remained below those observed during the original task. Repeated measures ANOVA's with period as the within factor and group as the between factor confirmed these mean differences. Blood pressure and heart rate responses were higher during the initial stressor than during the recall task (all $F(1,20)$'s > 15.91 , p 's $< .01$).

There was no difference between the Immediate and Delayed conditions in self-reports of how detailed the mental recreation of the stressor task was ($t(20) = 0.85$, $p = .4$).

2.3. Visit 3: repeat recall task

For subjects who returned for a second recall session 1 week after their initial recall (Immediate group), the mean systolic blood pressure during the rumination task was 2.2 mm Hg above baseline. This increase in systolic blood pressure was not reliably different from baseline ($t(9) = .80$, $p = .45$). The means during the repeat-rumination task for diastolic blood pressure (1.7 mm Hg) and for heart rate (2.0 bpm) also did not differ from baseline (both $t(9)$'s < 1.14 , p 's $> .20$).

For 9 out of 10 participants, the systolic blood pressure response during the second recall task was smaller than it had been at the first recall task. This pattern was confirmed statistically

with a significant binomial test for systolic blood pressure ($p < .05$).

Participants' self-reports of how detailed the recall was did not differ between the first and second recalls ($t(9) = .71$, $p = .5$).

2.4. Relations between stressor task reactivity and physiological responses during recall

Blood pressure and heart rate responses during the stressor task were not at all predictive of responses during the recall task ($r = .09, .08, .17$ for systolic blood pressure, diastolic blood pressure, and heart rate respectively, all p 's $> .4$). While on average, blood pressure levels of the Immediate group were not reliably elevated above baseline during their second recall task, individual blood pressure scores were, of course, mixed, with some showing elevations, and some not. This variability during the second recall was significantly associated with the magnitude of the response during the first recall, with a correlation coefficient of .63 ($p = .05$) for systolic blood pressure. A similar, though non-significant, effect emerged for diastolic blood pressure, with a correlation between the two rumination responses of .40 ($p = .26$). The participants who showed the highest reactivity when thinking about the prior event after 30 min were the ones who showed some elevations above baseline when they also recalled it 1 week later.

3. Discussion

The results shed light on two aspects of the relation between a harassment stressor and the later physiological response to the recall of that stressor. First, the data suggest that recall of even a relatively minor stressor such as the one we employed in this study is associated with increased blood pressure at recall both 30 min later and 1 week later. Second, our results suggest that the size of this blood pressure response does not diminish after a week's delay. Participants who were asked to recall this stressor 1 week later exhibited responses that were as large as those exhibited by participants who recalled the task only 30 min later. It may be that with longer delays the ability to recreate some of the cardiovascular response would be lost. However, even after a week, there is no sign of any diminution of the effect.

The results also provide some preliminary insight into the effects of repeated rumination. While directed recall of the stressor task for the first time produced reliable blood pressure increases above baseline levels, the same directed recall for the second time did not. In addition, for nine out of the ten participants who performed the recall task twice, the systolic blood pressure response during the first task was greater than the response during the second. This finding may be related to Pennebaker's work on the therapeutic benefits on disclosure, in which writing or talking about a past emotional experience is tied to positive health outcomes (Pennebaker, 1997). Here we find that just thinking about a prior stressor, without any disclosure, appears to alter later physiological responses. Compared with real-world stressors, ours is relatively minor. It may be that the presence or magnitude of the reduction in response associated with repeated rumination varies according

to the severity of the stressor. It is also possible that effects of repeated rumination differ depending on the type of rumination, with some sorts being more effective in reducing the concomitant physiological response than others.

Although physiological responses during the initial stressor task were not associated with responses during the recall task, systolic blood pressure responses during the first recall task were strongly positively associated with systolic blood pressure responses during the second recall task. Individuals who were the most activated during the first recall task also were most activated during the second. This finding tentatively suggests that identifying just high stress reactors as those at risk of later cardiovascular disease may importantly miss people who tend to show high responses to later rumination about anger or stress. The ability, or tendency, to recreate anger by ruminating, may be an important individual difference variable (Melamed, 1986, 1993). In particular, trait measures of hostility and anger expression style may identify those that have a specific propensity to keep anger alive (Brosschot and Thayer, 1998; Chambers and Davidson, 2000; Llabre et al., 2004; Smith et al., 2004).

Heart rate, although elevated during the mental arithmetic task, was not responsive to the recall task. Recalling the stressor task at 30 min or at 1 week did not produce any reliable increases in heart rate. This pattern of physiological responses is consistent with some earlier findings that elevations during rumination and recovery appear to be vascular in nature rather than myocardial (Chafin et al., 2004; Glynn et al., 2002; Gregg et al., 1999).

Two aspects of our findings in particular add to the growing body of evidence that argues for a broader definition of the reactivity hypothesis. First, ruminating on a past (and ended) stressor is associated with increased blood pressure. Our laboratory stressor is a fairly minor one, and it may be that more relevant, real-life stressors will be associated with even larger physiological responses at recall. This later recreation of a physiological response to stress does suggest a way that brief or intermittent stressors, such as interactions with an annoying supervisor, can have recurring or chronic effects. While many stressors are brief, the stress response need not be. Second, there appears to be some temporal stability to the blood pressure response produced during recall of a stressor. It will be worth examining in future work whether this stability is present because it was the same task that people were recalling on the two occasions, or because there are some people who tend to show high blood pressure responses whenever they ruminate. That is, one could examine whether there would be high stability in blood pressure responses between rumination sessions when the topic of the rumination changes between sessions. Furthermore, it may be useful to examine whether the general tendency to recreate stressors through rumination is associated with delayed cardiovascular recovery following a stressor. The people who are able to recreate the blood pressure response through rumination long after the task may be the same people who delay recovery immediately after the task by spontaneously ruminating.

If one includes the cardiovascular response to stress outside the presence of the stressor in the disease process, then it is important to begin to understand what factors might serve to reduce such responses. Our evidence suggests that even a delay of a week is not

enough to prevent rumination from recreating at least some of the physiological response. If, as the reactivity hypothesis suggests, cardiovascular responses, especially those without accompanying physical exertion, are damaging to the cardiovascular system, then stressful events have the potential to continue to do harm long after they are ended. This potential seems to require that the original stressor has an emotional component (Glynn et al., 2002), but not that the stressor has been recent. In addition, our data suggest that this potential may be reduced, or possibly eliminated, if the person has previously ruminated about the event. The second rumination, while still cognitively vivid, did not bring the “hot” physiological response. It may be that the advice to count to ten before responding to an angering event is misplaced. It may be, instead, that the opportunity to recall the event a few times before acting will be effective in reducing the anger.

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