The Role of Angry Rumination and Distraction in Blood Pressure Recovery From Emotional Arousal

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Objective: Cardiovascular recovery of prestress baseline blood pressure has been implicated as a possible additional determinant of sustained blood pressure elevation. We hypothesize that angry ruminations may slow the recovery process. **Method:** A within-subjects design was used in which resting baseline blood pressure and heart rate measurements were assessed on 60 subjects, who then took part in two anger-recall tasks. After each task, subjects sat quietly and alone during a 12-minute recovery period randomized to with or without distractions. During baseline, task, and recovery, blood pressure was continuously monitored; during recovery, subjects reported their thoughts at five fixed intervals. **Results:** Fewer angry thoughts were reported in the distraction condition (17%) compared with no distraction (31%; $p = .002$); an interaction showed that this effect was largely the result of the two intervals immediately after the anger-recall task. Trait rumination interacted with distraction condition such that high ruminators in the no-distraction condition evidenced the poorest blood pressure recovery, assessed as area under the curve ($p = .044$ [systolic blood pressure] and $p = .046$ [diastolic pressure]). **Conclusions:** People who have a tendency to ruminate about past anger-provoking events may be at greater risk for target organ damage as a result of sustained blood pressure elevations; the effect is exacerbated when distractions are not available to interrupt the ruminative process. **Key words:** cardiovascular reactivity, blood pressure, anger, hypertension, rumination.

ANOVA = analysis of variance; **AUC** = area under the curve; **BP** = blood pressure; **HTN** = hypertension; **CHD** = coronary heart disease; **CVR** = cardiovascular reactivity; **CVD** = cardiovascular disease; **HR** = heart rate; **DAB-VR** = Destructive Anger Behavior–Verbal Rumination; **DBP** = diastolic BP; **HPA** = hypothalamic–pituitary–adrenal; **SBP** = systolic BP.

INTRODUCTION

Although it is widely accepted that behavioral and physiological responses to stress are implicated in the development of hypertension (HTN) and coronary heart disease (CHD), the mechanisms by which this occurs remain poorly understood (1). One possible reason for this is the predominant focus on physiological responses that occur while the stressful event is actually occurring (i.e., "reactivity") with little attention paid to responses that occur when the stressor is no longer present ("recovery"). However, humans are quite able to reexperience the accompanying effect long after the occurrence of the precipitating event; such memories and affect may cause elevated blood pressure (BP) well beyond the duration of the acute stressor. It is unclear to what extent the BP peaks and troughs that occur during acute stress contribute to vascular damage, but sustained BP elevations are linked with target organ damage (2,3). Thus, it is possible that a 2 mm Hg BP increase lasting several hours would produce a far greater cardiovascular load than a large BP spike lasting only a few minutes. In addition, BP pressure recovery from stress has been hypothesized to be an independent risk factor for HTN and CHD (4–6). In the present study, we focus on the BP recovery from emotional arousal—an anger-recall task (7). We hypothesize that to the extent that a person has a greater trait tendency to ruminate about stressful events, effects on BP will also persist.

Anger and Blood Pressure

A number of studies have demonstrated associations between anger and BP level (8–10). The predominant biopsychosocial model of the relationship between anger and HTN—cardiovascular reactivity (CVR)—proposes that acute BP elevations occurring during the presentation of stressors lead, over time, to elevation of the tonic BP level, HTN, and atherosclerosis (11).

Blood Pressure Recovery May Be a Risk Factor for Hypertension

Several prospective studies have investigated the relationship between delayed blood pressure recovery from psychologic stressors in the laboratory and the development of HTN (13–15). For example, Borghi et al. (14) found that in borderline hypertensives, poor diastolic BP (DBP) recovery after mental stress predicted the development of frank HTN at 5-year follow up; they further found that recovery had greater predictive power than CVR.

Poor BP recovery, and subsequent BP elevation, may be a pathway by which the effects of anger influence the development of HTN. Anger is typically accompanied by sympathetic activation (16), which is the major putative mechanism for explaining its relation to CVD (17). Such activation may persist long after the anger provocation has ended and may both potentiate, as well as be sustained by, anger (18,19). In addition to direct effects on CVD, continued sympathetic activation also maintains BP elevations, and sustained elevated BP has a direct effect on left ventricular and vascular hypertrophy (20,21). Poor CV recovery may also be a marker of chronic sympathetic activation and low parasympathetic tone (16,17,22).

Several studies have shown that CV recovery from anger takes much longer than from other stressors (23,24); e.g., loud noise or physical exertion. Brosschot et al. (22) have noted that poor recovery to prestress levels is required for an adequate model of the relation between anger and the development of CVD; and they note that poor CV recovery is related to persistently low parasympathetic activity (or low vagal tone); in contrast, high vagal tone is associated with lower heart rate, better CV recovery, greater heart rate variability, and has been shown to be cardioprotective (22,25). These considerations have led researchers to suggest that causal explanations of biobehavioral disorders and the design of clinical interventions may be better served by studying psychophysiological recovery than focusing exclusively on CVR (6,26–28). Furthermore, Linden et al. (29) have shown greater generalizability of stress responses to social laboratory stressors than to traditional laboratory stressors, suggesting that social tasks may be more representative of daily life stressors.

Rumination

Sustained blood pressure elevations can occur when someone uses cognitive or emotional processes that sustain arousal. Stress-related thoughts and emotions are, of course, not limited to those occasions when a stressor is physically present. For most people, relatively little time is spent "in the heat of the moment." However, the greater portion of a lifetime can be spent in anticipation of future stressors and recovery from past stressors.

Ruminative responses have been defined as ". . . thoughts and behaviors that focus the individual's attention on the negative mood, and the causes and consequences of the mood, and self-evaluations related to the mood" (30). Rumination, however, may be differentiated from structured problem-solving in that people who ruminate may tend to perseverate about the causes and consequences of their anger or other negative emotions but do not act to change the situation (31). This distinction has been supported by studies that have shown that people who endorse ruminative responses are significantly less likely to engage in active, structured problem-solving (32,33).

Rumination May Maintain Sustained Blood Pressure Elevation

Stressful or anger-provoking events often lead to subsequent cognitions (ruminations) about the event, and these may be accompanied by negative affect (34,35). For example, in the days after the terrorist attack on the World Trade Center, 30% of Americans reported being "quite a bit" or "extremely" upset when reminded of the attack, and 16% reported being "quite a bit" or "extremely" bothered by repeated, disturbing memories, thoughts, or dreams about the attack (35). In a demonstration of the effects that such cognitions may have on blood pressure, we have recently found that at four sites in the United States (New York, Washington, DC, Mississippi, and Chicago), self-monitored blood pressure was significantly

higher in the months after the attacks than in the months before (36).

In a laboratory study of rumination, Glynn et al. (23) harassed subjects during a mental arithmetic task; after the conclusion of the session, subjects returned to the laboratory after either 20 minutes or 1 week. On return, subjects were asked to recall the stressful task, imagining it as vividly as possible. Both groups showed substantial blood pressure elevations with no significant effect of time elapsed. These results suggest that recall of an emotional stressor can recreate blood pressure elevations and that the magnitude of activation provoked by recall may be sustained over significant periods of time. As an example of the potency of recalled stressors, we note that the recall of an anger-provoking event has been demonstrated to increase the difficulty of terminating ventricular tachycardia in arrhythmia-prone patients (37) and to acutely decrease left ventricular ejection fraction in subjects with CVD (7).

Cognition, Affect, and Physiological Activation (a Rumination–Arousal Model)

At the broadest level, we hypothesize that engaging in rumination after anger provocation sets in motion an intertwined set of processes: the cognition leads to anger (and possibly other negative emotions, e.g., anxiety), and the emotion produces elevated autonomic activation (causing blood pressure elevation). Brosschot and Thayer (22) have suggested that rumination ". . . may act to convert the immediate psychological and physiological concomitants of life events and daily stressors into prolonged physiological activation, which in turn is necessary for the development of a chronic pathogenic state." Figure 1 shows a proposed "rumination–arousal" model based on this idea.

A difficulty in untangling the causality among cognition, affect, and physiological response is that they may be reciprocally determined: increased autonomic arousal may prolong anger and vice versa (this was well-described in Schachter's two-factor theory of emotions [18]); and the prolonged anger may promote ruminative *thoughts* and vice versa. We suggest
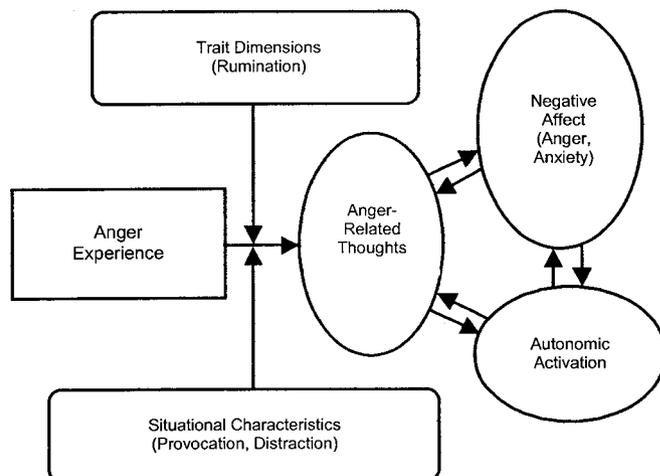

Figure 1.   The rumination–arousal model.

that these constructs operate as a "feed-forward" process and will continue to precipitate the process until a distraction occurs to displace the angry thoughts.

The Significance of 'Distraction' as a Moderator of Angry Thoughts, Negative Affect, and Blood Pressure

The use of distraction involves the availability of a compelling substitute to occupy one's thoughts. To the extent that one's thoughts are taken up with, say, an engrossing novel or an absorbing discussion about vacation plans, one will not be thinking about an argument that occurred earlier that day. Rusting and Nolen-Hoeksema found that distraction reduced angry mood after a personally relevant anger-induction task, whereas rumination increased feelings of anger (38).

If, indeed, ruminating about previous events is linked to sustained blood pressure elevation, then the use of distraction provides a useful means of studying the role of cognition in physiological activation. In a recent paper, distraction was related to faster cardiac recovery (assessed by the hemodynamic pattern), and to greater high-frequency power and less low-frequency power, assessed by spectral analysis of heart rate variability, which suggests diminished beta-adrenergic activation and enhanced vagal tone (39). In a study conducted in our laboratory, we have found that emotional stressors, which lend themselves to rumination in a way that purely physical stressors do not, were associated with poorer blood pressure recovery (23). Further supporting the role of rumination in this delayed recovery, we found that providing a distraction immediately after the stressor greatly sped recovery. Finally, when the distractor was removed, we observed a spontaneous "reelevation" of the blood pressure response, albeit not to the original level that was observed during the stressor (23).

In the present study, the absence or availability of distracting stimuli was manipulated as a means of evaluating the role of angry cognition in the sustained elevation of blood pressure. Distraction was operationalized as any stimulus that diverts the subject from one train of thought to another; in the present study, we provided colorful posters and toys as a means of accomplishing this. We used an anger-recall task to induce angry mood, and examined the additive and interactive effects of trait rumination and distraction on angry thoughts and sustained blood pressure elevations.

Study Hypotheses

1. The distraction manipulation will have a main effect on a) angry thoughts and b) blood pressure recovery such that when in the no-distraction condition, subjects will tend to have more angry thoughts and poorer recovery in the postanger recall rest period compared with when in the distraction condition.
2. The interaction between the distraction manipulation and trait rumination will affect a) angry thoughts and b) blood pressure recovery such that persons who score high on trait rumination, when in the no-distraction condition, will tend to have more angry thoughts and

poorer recovery than low trait ruminators in either of the distraction conditions.

The focus of the study is on sustained blood pressure elevation after anger recall; however, heart rate data are also reported.

METHODS
Subjects

Sixty subjects (30 males, 30 females; mean age 37.9 ± 13.8 years) participated in the study. Table 1 shows the breakdown by ethnicity and race. Subjects were excluded if they had a history of HTN or CHD. Subjects were recruited by advertisement at Cornell University Medical Center, were asked to refrain from smoking or drinking caffeinated beverages for 4 hours before the experimental sessions, and were paid $25 for participating. Informed consent, approved by the Cornell Institutional Review Board, was given by all subjects.

Overview of Procedures

All subjects participated in both the distraction and no-distraction conditions, in counterbalanced order, with a 1-week interval between sessions. Before the experimental sessions, subjects came to the psychophysiology laboratory where the experimenter explained the study and obtained informed consent.

During both experimental sessions, continuous blood pressure and heart rate (HR) measurements were taken during a 10-minute baseline period, a 5-minute task period (anger recall [7]), and a 12-minute posttask recovery period.

Anger Incident Measure

During the initial session, subjects were asked to list three situations that had occurred during the prior year in which they became upset and angry—situations that were poorly resolved and that still upset them to think about. After completing the anger incident measure, subjects completed Likert-type seven-point scales assessing how angry they felt at the time that each incident occurred. The two incidents associated with the highest anger ratings were randomly assigned for use in the two experimental sessions.

Anger-Recall Task

Following the protocol developed by Ironson and colleagues (7), the experimenter asked the subjects to describe in detail what had occurred during the incident and to describe how they felt during the incident. Although subjects were asked to speak for a specified interval (5 minutes), they were allowed to talk for as long as they liked; thus, the amount of time spent describing the anger-provoking situation was allowed to vary.

During the task, the experimenter nodded and made eye contact to encourage the subject to continue speaking, but did not indicate agreement or disagreement with the statements.

Distraction Manipulation

The manipulation involved the availability of distraction during posttask recovery. In both conditions, a free-standing screen (1.5 m wide × 2 m high)

TABLE 1. Breakdown of Sample by Ethnicity and Race (*n* = 60)

	Women	Men	Total
Ethnic category			
Hispanic or Latino (%)	2 (3.3)	5 (8.3)	7 (11.7)
Not Hispanic or Latino (%)	28 (46.7)	25 (41.7)	53 (88.3)
Racial category			
Asian/Pacific Islander (%)	0 (0.0)	3 (5.0)	3 (5.0)
Black or African American (%)	10 (16.7)	4 (6.7)	14 (23.3)
White (%)	17 (28.3)	22 (36.7)	39 (65.0)
Other declined to state (%)	3 (5.0)	1 (1.7)	4 (6.7)

was placed in the subject's view, approximately 1.5 m away. On one side of the screen, the panels were blank; on the other side, more than 30 brightly colored cards and posters were placed. In the no-distraction condition, the blank side of the screen was turned to face the subject, and all other incidental distractions were eliminated in the stark, windowless, room. In the distraction condition, the visually interesting side of the screen was turned to the subject. In addition, magazines and small toys (for example, small puzzles that required manipulating a tiny ball bearing to fall into a hole) were placed in easy reach. The experimenter was blind to the condition until the beginning of the recovery period during both sessions.

Assessment of Trait Rumination

The Destructive Anger Behavior–Verbal Rumination (DAB-VR) assesses the tendency to engage in angry ruminative thoughts. The DAB-VR is a five-item scale with good internal reliability (Cronbach's alpha = 0.69) and adequate test–retest reliability (0.63 over a 12-week period) (40). Sample items include, "After discussing my anger, I continue to dwell on it" and "After discussing my anger, I feel even more agitated." The measure is moderately and significantly correlated with several measures of anger expression, including the Multidimensional Anger Inventory, the State-Trait Anger Scale, the Cook-Medley Hostility Scale, and the Anger Expression Inventory.

Sampling of Self-Reported Thoughts

During the recovery period, we assessed the content of a selected sample of the subjects' thoughts. The method of thought sampling used was constrained by a crucial consideration: The major outcomes were the blood pressure and HR assessed during recovery as a function of the thoughts that the subject might have during this period. However, the *reporting* of such thoughts might interfere with the observed association between the thoughts and their physiological concomitants. To minimize this, subjects were told that they would hear a tone at fixed intervals during the recovery period. When the subjects heard the tone, they were to write down just one or two words concerning whatever they had been thinking about at that precise moment. Subjects were told that when the recovery period was over, the experimenter would ask the subject to expound on these brief notations. Thought sampling occurred five times during the recovery period (although subjects did not know how many there would be in advance) occurring 0.5, 1.5, 4.0, 8.0, and 11.0 minutes into the period.

The thoughts reported during the recovery phase were coded as "anger-recall-related," "distraction-related" (i.e., were triggered by one of the distraction stimuli; relevant only for the distraction condition), or "other." The coding was not subjective on the experimenter's part; subjects indicated which of these categories each reported thought fell into. The only purpose for the "distraction-related" category was to provide a manipulation check, i.e., how many people in the distraction condition were actually reporting distraction-related thoughts. The main outcome, concerning self-reported thoughts, however, was between anger-recall-related thoughts and nonanger-recall-related thoughts.

Recording of Cardiovascular Activity

Blood pressure and HR were collected using an Ohmeda Finapres 2300 Blood Pressure Monitor (Datex-Ohmeda, Englewood, CO), which takes and records beat-to-beat pressures in a noninvasive manner using the Peñaz method (41). The Finapres was attached through the serial line to a computer for this purpose. Systolic and diastolic pressures, as well as HR, were assessed. This method uses a finger cuff, which was worn on the third finger of the nondominant hand. The method of operation of the Finapres monitor has been described in detail by several authors (42–45). It has been shown to track intraarterial readings extremely well, even during sudden changes in blood pressure (46), making it quite useful for reactivity and recovery testing. We have also shown that the resulting summary measures of blood pressure and HR are extremely reliable because of the large number of measurements that are available to be averaged (47). However, Finapres BP change scores are generally larger than those obtained from auscultatory or oscillometric BP measurements (48).

Procedure

The subject was fitted with a finger blood pressure cuff, and then was asked to sit quietly during a 10-minute baseline rest period with the experimenter out of the room.

The experimenter then gave the instructions for the anger-recall task and explained that immediately after the task, the subject would relax during a second rest period (i.e., recovery), this one lasting 12 minutes, and explained the "thought-sampling" task at this time (so that the recovery period could begin immediately at the end of the anger-recall period).

After the anger-recall task, the experimenter determined the experimental condition (distraction or no distraction) using a randomization table and arranged the setting accordingly (turning the screen, which had been hidden until this time, to the appropriate side, and providing magazines and toys in the distraction condition). The experimenter then left the room.

After 15 minutes, the experimenter returned and asked the subject to elaborate on each notation. For example, if the subject had written "brother," the subject might explain that she was referring to the argument with her brother that she had described during the anger-recall task. Alternatively, if the subject had written "Paris," the subject might explain that he had been looking at the postcard of the Eiffel Tower on the screen.

The second session was identical to the first, except that subjects participated in the other distraction condition. To keep the experimenter blind to condition until the beginning of each session's recovery period, a different experimenter conducted the second experimental session.

Data Analysis

We have previously described a method for the analysis of blood pressure recovery using a curve-fitting procedure ([49] see Figure 2). Initially, each individual's time series of BP and HR data are smoothed, taking 15-point moving averages within each phase of the protocol (baseline, anger-recall task, and recovery). The primary outcome is the relative time-integrated area under the curve (AUC), which represents cardiovascular load and is implicitly
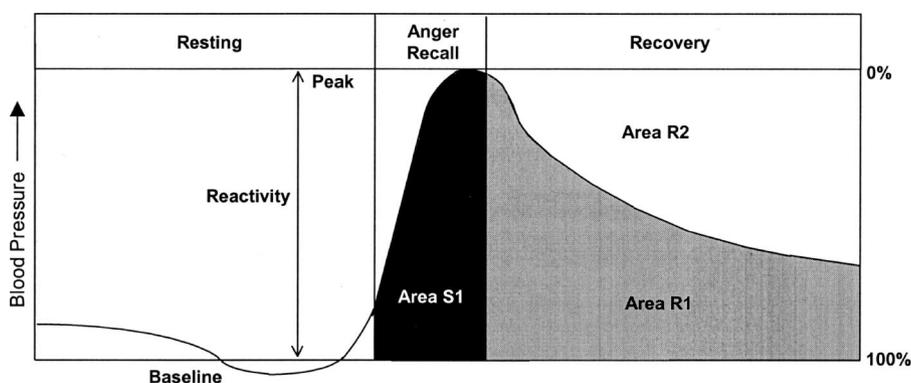

Figure 2.   Curve-fitting procedure for the assessment of blood pressure recovery.

adjusted for the magnitude of the stress response. Baseline values were computed as the mean of the individual blood pressure and HR measurements taken during the final 2 minutes of the initial rest period (allowing a 15-second interval between baseline and task period). We have shown previously that this is sufficient to provide a stable baseline when using beat-to-beat measures (50).

Paired t tests were used to assess the effect of the anger-recall task on the reactivity of the cardiovascular; a repeated-measures analysis of variance (ANOVA) was used to examine the proportion of anger-recall task-related thoughts reported during the recovery period (Mauchly's test was used to test the assumption of sphericity), and the Tukey post hoc test for within-subject differences was used to test the differences between individual means. Repeated-measures ANOVAs were used to estimate the main effects of distraction condition and trait rumination (DAB-VR), as well as their interaction, on the cardiovascular recovery measures.

RESULTS
Baseline Cardiovascular Measures

Because sessions 1 and 2 were identical up until the recovery period (at which point the distraction manipulation was implemented), the first set of analyses examine the effects of session order, independent of distraction condition, and the effects of distraction condition, independent of session order. Table 2 shows the baseline cardiovascular values. As the table shows, session order had little effect on blood pressure or HR. A subsequent analysis, using order of distraction condition and trait rumination as factors, and blood pressure and heart rate load, represented as area under the curve, as the outcome measures, showed that the order of the sessions was not significant (lowest $p = .113$).

Baseline values were also computed for the distraction and no-distraction conditions (independent of order), and these are also shown in Table 2. Again, there was little difference between conditions (all p's > .35).

Duration of Anger Recall Across Conditions

Although the duration of the task was meant to be 5 minutes, some subjects did not talk for that long, and others talked longer (we did not want to cut the subject off); thus, there was variability in the duration. However, this did not vary by condition; the average durations were 4:34 (standard deviation [SD] = 2.29) and 4:29 (SD = 2.39) in the distraction and no-distraction conditions, respectively. The correlation between the conditions for this measure was 0.64 ($p < .001$). Thus, duration of speaking did not appear to represent confounding to the internal validity of the study.

Effects of Anger-Recall on Blood Pressure and Heart Rate

Table 2 shows the blood pressure and HR change values between baseline level and the level observed during the anger-recall tasks. Anger-recall had a substantial impact on blood pressure and a lesser impact on heart rate. Paired t tests showed that the anger-recall task significantly elevated blood pressure and heart rate (smallest $t = 7.2$; all $p < .0005$).

Table 2 also shows the reactivity values observed during sessions 1 and 2, as well as for the two distraction conditions, independent of session order. The blood pressure and HR changes tended to be higher during the first session, and the differences were significant for systolic blood pressure (t [59] = 2.23, $p < .05$) and for HR ($t = 2.29$, $p < .05$).

The reactivity scores also tended to be higher during the no-distraction condition, although it is important to note that the reactivity changes occurred before the manipulation, and thus differences were likely the result of chance. The differences for the systolic and diastolic blood pressure were marginally significant, with $t = 1.75$ ($p = .085$, systolic) and $t = 1.74$ ($p = .088$, diastolic). The difference for HR was significant ($t = 2.11$, $p < .05$).

Although it is possible that the differences in reactivity scores between conditions were the result of random error, we further explored these data to see if condition interacted with session order. In fact, we found significant interactions for systolic blood pressure (F [1, 58] = 4.14, $p = .046$) and for heart rate (F [1, 58] = 4.27, $p = .043$). No significant effect was observed for diastolic pressure ($p = .17$). As Table 2 shows, the lower values tended to occur during session 2 (this may be the result of habituation) and in the no-distraction condition. To control for possible session effects, subsequent analyses were conducted with and without inclusion of the corresponding reactivity measure as a covariate. Inclusion of reactivity values did not affect the results; therefore, we present results of the analyses without reactivity.

Effects of Distraction on Self-Reported Thoughts During Recovery

The thoughts reported during the recovery phase were coded as anger-recall task-related and (during the distraction condition only) as distraction-related (these were thoughts that

TABLE 2. Mean (SD) Baseline Cardiovascular Values and Cardiovascular Changes During Anger-Recall for Sessions 1 and 2, and by Distraction Condition

	Session 1	Session 2	Distraction	No Distraction
Baseline SBP	120.0 (22.9)	118.5 (17.3)	118.1 (19.7)	120.5 (21.0)
Baseline DBP	70.0 (13.5)	70.6 (12.7)	70.5 (14.3)	70.1 (11.8)
Baseline HR	73.7 (13.0)	73.8 (11.9)	74.1 (12.9)	73.4 (12.0)
SBP change (mm Hg)	30.7 (16.8)**	26.1 (16.5)	26.5 (14.7)*	30.2 (18.5)
DBP change (mm Hg)	18.2 (8.7)	16.3 (10.0)	16.2 (8.0)*	18.3 (10.6)
HR change (beats/min)	10.4 (8.1)**	8.0 (6.1)	8.1 (5.1)**	10.4 (8.8)

*$p < .10$; **$p < .05$.
SD = standard deviation; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate.

referred specifically to one or more of the distracting materials that were available in that condition), or, in both conditions, as "other." The percentage of anger recall and distraction-related thoughts were then computed for each subject.

In the distraction condition, 50% of all reported thoughts referred to a provided distraction, and 83% of subjects had at least one distraction-related thought. An average of 17% of the thoughts was anger-recall-related in the distraction condition compared with an average of 31% in the no-distraction condition. This difference was significant, with $t = 3.2$ ($p = .002$). Thus, it would appear that distraction did have a systematic effect on angry rumination.

Figure 3 shows the percentages of anger-recall task-related thoughts across persons for each of the five individual reports. It can be seen that the differences tended to be greater in the earlier intervals, i.e., those most closely after the task, and that the difference tended to decrease in the later intervals. A repeated-measures ANOVA indicated that both main effects were significant, with $F (1, 59) = 10.96$ ($p = .002$) (effect of distraction condition) and $F (4, 236) = 8.68$ ($p < .0005$) (effect of time, i.e., the five sampling intervals). Thus, part of hypothesis 1, which predicted a main effect of the distraction manipulation on angry thoughts during recovery, was supported. The interaction between the two factors also was significant, with $F (4, 236) = 4.05$ ($p = .003$). Mauchly's test indicated that the sphericity assumption was not violated in these analyses. Tukey's post hoc test for repeated measures indicated that the differences at intervals 1 and 2 were significant between conditions ($p < .01$); the differences at the remaining three intervals were not significant.

Effects of Distraction by Trait Rumination Interaction on Self-Reported Thoughts

We examined the correlation between trait rumination (DAB-VR) and the percentage of anger-recall task-related thoughts collapsed across distraction–no-distraction conditions. The correlation was small and nonsignificant ($r = 0.15$, $p = .26$). When examined within each distraction condition, however, the correlations were $r = 0.08$ ($p = .54$) (distraction) compared with $r = 0.39$ ($p = .002$) (no-distraction condition).
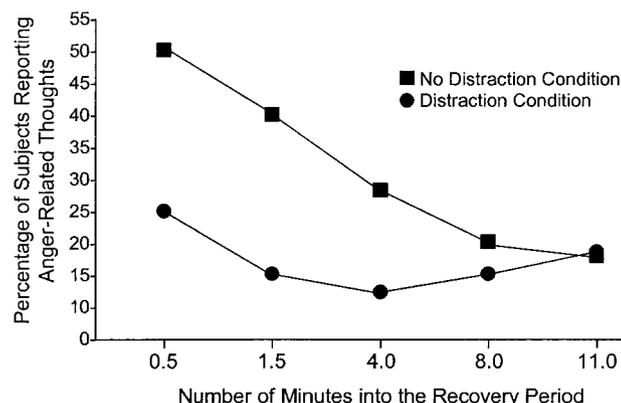

Figure 3. Percentage of anger-recall task-related thoughts across persons at each of the five sampled intervals during the recovery period.

A repeated-measures ANOVA indicated that the interaction between DAB-VR and distraction condition also was not significant, with $F (1, 58) = 0.90$ ($p = .35$). Thus, the part of hypothesis, which predicted a significant effect of the interaction on self-reported angry thoughts during the recovery period, was not supported.

Effects of Trait Rumination on Blood Pressure and Heart Rate During Recovery

Pearson correlations were computed between DAB-VR and the AUC for blood pressure and HR across conditions. In the distraction condition, the correlations were negative for both blood pressure measures and for HR, but not significant (range, -0.08 to -0.022); in the no-distraction condition, in contrast, the correlations were all positive, but only one of the correlations was significant (for diastolic pressure; $r = 0.25$, $p < .05$). The next set of analyses more fully address the effects of DAB-VR and distraction condition, and their interaction, on cardiovascular recovery.

Effects of Distraction and Trait Rumination on Blood Pressure and Heart Rate During Recovery

A small number of subjects did not react to the anger recall task in terms of their blood pressure or HR. Because there can be no sensible measure of recovery if no response has occurred, we omitted subjects from analyses involving recovery who exhibited less than 0 mm Hg reactivity in BP or zero beats per minute in HR. The number of subjects excluded on this were: one from each distraction condition for systolic blood pressure analyses, none for the diastolic blood pressure analyses, and three and two from the distraction and no distractions, respectively, for HR.

The outcome measure in these analyses were the AUCs, representing the cardiovascular load borne under each condition. The results are shown in Table 3, and Figure 4 shows the interactions for systolic blood pressure (the pattern for diastolic pressure is similar).

As Tables 3 and 4 show, the data partly supported hypothesis 1, which predicted a main effect of the distraction manipulation on AUC; the effect was significant for diastolic blood pressure and marginal for systolic blood pressure. Hypothesis 2, which predicted an interaction between the distraction manipulation and trait rumination, also was supported, with significant effects observed for both systolic and diastolic blood pressure. No main effect for trait rumination was observed.

DISCUSSION

The study hypotheses were largely supported by the data: the distraction manipulation had a main effect on both angry thoughts and cardiovascular recovery. In addition, the distraction condition interacted, as predicted, with the DAB-VR, such that high trait ruminators had the poorest cardiovascular recovery when no distractions were present.

The data suggest that people who tend to ruminate will do so after an anger-provoking event (or, in this case, the recall of

TABLE 3. Mean (and SD) Area Under the Curve by DAB-VR Median Split (Low Versus High) for the Distraction and No-Distraction Conditions

	Distraction Condition		No Distraction Condition	
	Low DAB-VR (*n* = 28)	High DAB-VR (*n* = 32)	Low DAB-VR (*n* = 28)	High DAB-VR (*n* = 32)
Systolic blood pressure	0.16 (0.26)	0.17 (0.32)	0.12 (0.24)	0.25 (0.24)
Diastolic blood pressure	0.19 (0.30)	0.12 (0.38)	0.14 (0.21)	0.22 (.022)
Heart rate	0.09 (0.53)	−0.08 (0.37)	−0.07 (0.34)	−0.03 (0.37)

SD = standard deviation; DAB-VR = Destructive Anger Behavior–Verbal Rumination.

such an event) unless and until they are distracted from their angry thoughts. These data provide preliminary support for the rumination–arousal model depicted in Figure 1.

These findings are partly consistent with those of a recent study that also examined effects of distraction on recovery after anger-recall (39). Neumann et al. found that distraction decreased angry mood and rumination and predicted faster HR recovery; however, they did not find effects on BP recovery. They also did not find a significant interaction between distraction and a dispositional factor, hostility, on recovery. A number of methodological differences between the two studies may explain the inconsistencies. First, the two studies used different distraction manipulations: subjects in the Neumann et al. study were instructed to read a neutral article, whereas in our study, subjects were provided with multiple opportunities for distraction but were not specifically directed to engage in any of the available distractions. Another important difference is that Neuman et al. assessed hostility, which is correlated with but distinct from trait rumination. It is possible that the processes disrupted by distraction are not as closely related to hostility as they are to rumination. Other differences include the fact that Neuman et al. studied only women; they measured rumination retrospectively, whereas we used a thought-sampling procedure to measure angry thoughts during the recovery period; and although they used the same anger-recall task, the duration was shorter, as was the duration of the recovery period.
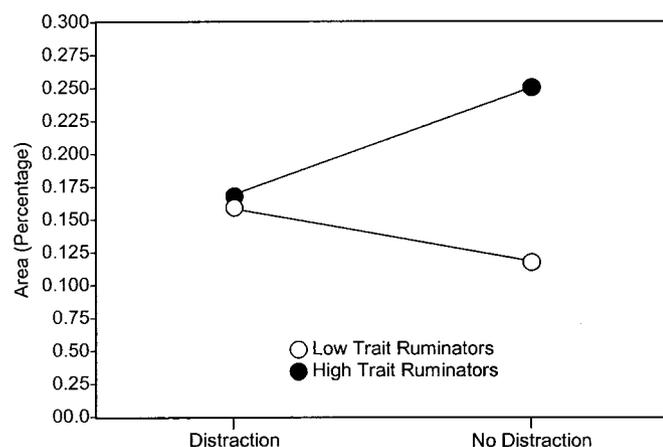
The primary limitation of the current study is the use of the laboratory setting. However, there is evidence suggesting that responses to psychosocial stressors in the laboratory predict ambulatory blood pressure (29). Still, it is important to test these hypotheses outside of the laboratory, and this is the focus of our current work. Another limitation of this study is the complexity of the distraction manipulation used. Although the fact that we did not provide a single distraction or specifically instruct subjects to engage in distraction may result in more generalizable findings, it also makes these findings difficult to interpret, because we do not know which distractions subjects engaged in, if any, and how engaged they were.

An important question that remains unanswered concerns the generalizability of the effects of trait rumination and distraction on ruminative thoughts. It seems clear that rumination itself is not limited to the laboratory. The question is whether the ruminative process has physiological effects that are clinically meaningful, and that remains to be tested. We speculate that the ambulatory blood pressure level, which is determined by a multitude of influences, including posture,

TABLE 4. ANOVA Results Table for Analysis of Effect of DAB-VR and Distraction Condition on Cardiovascular Recovery Measures

	Area Under the Curve	
	F	Significance
Systolic blood pressure (df = 1,56)		
Distraction condition	3.74	.058
DAB-VR	0.21	.649
Distraction X DAB-VR	4.24	.044
Diastolic blood pressure (df = 1,58)		
Distraction condition	4.16	.046
DAB-VR	1.51	.224
Distraction X DAB-VR	4.15	.046
Heart rate (df = 1,53)		
Distraction condition	0.60	.442
DAB-VR	0.81	.373
Distraction X DAB-VR	0.24	.636

Note: To evaluate the effect of the interaction between condition and session order, the analyses were conducted both with and without reactivity as a covariate; inclusion of the covariate made little difference, and no significant values became nonsignificant as a result of the covariate analysis. The statistical analyses shown in this table therefore do not control for reactivity. ANOVA = analysis of variance; DAB-VR = Destructive Anger Behavior–Verbal Rumination.

Figure 4. Interactions in cardiovascular recovery between distraction conditions and trait rumination.

physical activity level, sex, race, and many psychosocial factors, is also influenced by the reciprocally determined combination of angry thoughts, the accompanying affect, and the physiological arousal, as suggested by the rumination–arousal model described in Figure 1. In a current study, we are monitoring ambulatory blood pressure immediately after the laboratory session as a means of testing the extent to which angry thoughts, as recorded in an electronic diary, are associated with elevated blood pressure in both low and high trait ruminators.

The question of how cardiovascular recovery should be assessed in the field has long been debated. We suggest that "recovery" represents a special case of a more general phenomenon. Recovery can only occur immediately after a specific stimulus event. However, the evidence suggests that persons ruminate even after such events have long ended (30,31) and that such rumination can elevate blood pressure (as observed in the present study). It seems possible that the cognitive and affective effects of an anger-producing event, especially in persons who have a greater tendency to ruminate, may produce a far greater cardiovascular load than occurs during and immediately after the event. Thus, the reactivity and immediate recovery may provide just a small window into a broader, and more chronic, stress–response pattern.

There is an additional implication embodied in the notion that the ambulatory blood pressure may be more relevant than acute laboratory measures for the study of psychosocial influences on the development of cardiovascular disease. The implicit assumption underlying the cardiovascular reactivity model, which focuses solely on the brief and immediate blood pressure and HR response to stress, is that it is the *intensity* of the cardiovascular response that is important for disease development. The model may be extended to incorporate the blood pressure recovery of the prestress baseline. However, data suggest that it is the *duration* of the response, even at low-intensity, that matters (2,3). Thus, even a small increase in blood pressure resulting from angry rumination that occurs over an extended period of time may have a greater effect on target organ damage than that attributable to the sustained elevation that may occur immediately after the actual anger-producing event.

Our focus has been on blood pressure, rather than HR, responses. Sustained blood pressure elevations have been implicated in the development of damage to the target organs, including the vessels, kidneys, brain, and heart (2,3). It is worth noting that HR recovery did not follow the pattern of blood pressure. In fact, as shown in Table 3, the postanger HR load, assessed by AUC, is extremely small compared with that of blood pressure. In a current study, we are evaluating the effects of rumination on HR variability, which provides a measure of autonomic dysregulation, and this may shed light on the precise nature of the mechanism by which rumination affects blood pressure recovery.

A great deal of effort has been expended on the study of the physiological consequences of acute stressors, although their pathogenic influences in the development of HTN and CHD

remain controversial (1). Exposure to stress must be considered in a causal model of stressor effects; however, little evidence indicates that physiological responses to acute stress generalize beyond the experimental laboratory. This suggests that we must focus on naturally occurring chronic stressors if we are to understand the mechanisms by which stress is linked to poorer cardiovascular outcomes. The present study is an attempt to shift the focus from acute stress to its after-effects. It also shifts the focus from stressful events to their interaction with coping styles such as rumination. This shift will aid in an effort to identify the factors that lead to sustained blood pressure elevations. It may be that the most important effects of a stressor occur long after the stressor itself has ended.

REFERENCES

1. Schwartz AR, Gerin W, Davidson KD, Pickering TG, Brosschot JF, Thayer JF, Christenfeld N, Linden W. In search of a coherent model of stressor effects on short-term cardiovascular adjustments and the development of cardiovascular disease. Psychosom Med 2003;65:22–35.
2. Perloff D, Sokolow M, Cowan RM. The prognostic value of ambulatory blood pressures. JAMA 1983;249:2792–8.
3. Liu JE, Roman MJ, Pini R, Schwartz JE, Pickering TG, Devereux RB. Cardiac and arterial target organ damage in adults with elevated ambulatory and normal office blood pressure. Ann Intern Med 1999;131:564–72.
4. Hocking Schuler JL, O'Brien WH. Cardiovascular recovery from stress and hypertension risk factors: a meta-analytic review. Psychophysiology 1997;34:649–59.
5. Haynes SN, Gannon LR, Orimoto L, O'Brien WH, Brandt M. Psychophysiological assessment of poststress recovery. Psychol Assess 1991;3:356–65.
6. Gerin W, Pickering TG. Association between delayed recovery of blood pressure following acute mental stress and parental history of hypertension. J Hypertens 1995;13:603–10.
7. Ironson G, Taylor CB, Boltwood M, Bartzokis T, Dennis C, Chesney M, Spitzer S, Segall GM. Effects of anger on left ventricular ejection fraction in coronary artery disease. Am J Cardiol 1992;70:281–5.
8. Suls J, Wan CK, Costa PT Jr. Relationship of trait anger to resting blood pressure: a meta-analysis. Health Psychol 1995;14:444–56.
9. Everson SA, Goldberg DE, Kaplan GA, Julkunen J, Salonen JT. Anger expression and incident hypertension. Psychosom Med 1998;60:730–5.
10. Markovitz JH, Matthews KA, Wing RR, Kuller LH, Meilahn EN. Psychological, biological and health behavior predictors of blood pressure changes in middle-aged women. J Hypertens 1991;9:399–406.
11. Manuck SB, Kasprowicz AS, Muldoon MF. Behaviorally-evoked cardiovascular reactivity and hypertension: conceptual issues and potential associations. Ann Behav Med 1990;12:17–29.
12. Pickering TG, Gerin W. Cardiovascular reactivity in the laboratory and the role of behavioral factors in hypertension: a critical review. Ann Behav Med 1990;12:3–16.
13. Treiber FA, Musante L, Kapuku G, Davis C, Litaker M, Davis H. Cardiovascular (CV) responsivity and recovery to acute stress and future CV functioning in youth with family histories of CV disease: a 4-year longitudinal study. Int J Psychophysiol 2001;41:65–74.
14. Borghi C, Costa F, Boschi S, Mussi A, Ambrosioni E. Predictors of stable hypertension in young borderline subjects: a five-year follow-up study. J Cardiovasc Pharmacol 1986;8(suppl 5):S138–41.
15. Stewart JC, France CR. Cardiovascular recovery from stress predicts longitudinal changes in blood pressure. Biol Psychol 2001;58:105–20.
16. Julius S. Sympathetic hyperactivity and coronary risk in hypertension. Hypertension 1993;21:886–93.
17. Amerena J, Julius S. The role of the autonomic nervous system in hypertension. Hypertens Res 1995;18:99–110.
18. Schachter S, Singer J. Cognitive, social and physiological determinants of emotion. Psych Rev 1962;69:379–99.
19. McEwen BS. Seminars in medicine of the Beth Israel Deaconess Medical Center: protective and damaging effects of stress mediators. N Engl J Med 1998;338:171–9.
20. Devereux RB, Pickering TG, Harshfield GA, Kleinert HD, Denby L,

Clark L, Pregibon D, Jason M, Kleiner B, Borer JS, Laragh JH. Left ventricular hypertrophy in patients with hypertension: Importance of blood pressure response to regularly recurring stress. Circulation 1983; 68:470–6.

21. Verdecchia P, Schillaci G, Borgioni C, Ciucci A, Gattobigio R, Zampi I, Santucci A, Santucci C, Reboldi G, Porcellati C. Prognostic value of left ventricular mass and geometry in systemic hypertension with left ventricular hypertrophy. Am J Cardiol 1996;78:197–202.

22. Brosschot JF, Thayer JF, Gerin W. The perseverative cognition hypothesis: a review of worry, prolonged stress-related physiological activation and health. J Psychosom Res. In press.

23. Glynn LM, Christenfeld N, Gerin W. The role of rumination in recovery from reactivity: cardiovascular consequences of emotional states. Psychosom Med 2002;64:714–26.

24. Vitaliano PP, Russo J, Paulsen VM, Bailey SL. Cardiovascular recovery from laboratory stress: biopsychosocial concomitants in older adults. J Psychosom Res 1995;39:361–77.

25. Sloan RP, Shapiro PA, Bagiella E, Myers MM, Gorman JM. Cardiac autonomic control buffers blood pressure variability responses to challenge: a psychophysiologic model of coronary artery disease. Psychosom Med 1999;61:58–68.

26. Jamieson JL, Lavoie NF. Type A behavior, aerobic power, and cardiovascular recovery from a psychosocial stressor. Health Psychol 1987;6: 361–71.

27. Christenfeld N, Glynn LM, Gerin W. On the reliable assessment of cardiovascular recovery: an application of curve-fitting techniques. Psychophysiology 2000;37:543–50.

28. Linden W, Earle T, Gerin W, Christenfeld N. Physiological stress reactivity and recovery: conceptual siblings separated at birth? J Psychosom Res 1997;42:117–35.

29. Linden W, Rutledge T, Con A. A case for the usefulness of laboratory social stressors. Ann Behav Med 1998;20:310–6.

30. Nolen-Hoeksema S, Morrow J. A prospective study of depression and posttraumatic stress symptoms after a natural disaster: the 1989 Loma Prieta earthquake. J Pers Soc Psychol 1991;61:105–21.

31. Lyubomirsky S, Nolen-Hoeksema S. Effects of self-focused rumination on negative thinking and interpersonal problem solving. J Pers Soc Psychol 1995;69:176–90.

32. Lang PJ. Cognition and emotions: concept and action. In: Izard C, Kagan J, Zajonc RB, eds. Emotions, Cognition, and Behavior. New York: Cambridge University Press; 1984.

33. Larsen RJ, Diener E. Promises and problems with circumplex model of emotion. Rev Pers Soc Psychol 1992;13:25–9.

34. Nolen-Hoeksema S, Parker L, Larson J. Ruminative coping with depressed mood following loss. J Pers Soc Psychol 1994;67:92–104.

35. Schuster MA, Stein BD, Jaycox LH, Collins RL, Marshall GN, Elliott MN, Zhou AJ, Kanouse DE, Morrison JL, Berry SH. A national survey of stress reactions after the September 11, 2001, terrorist attacks. N Engl J Med 2001;345:1507–12.

36. Gerin W, Chaplin W, Schwartz JE, Holland J, Alter R, Wheeler R, Duong D, Pickering TG. Sustained blood pressure elevation following an acute stressor: the effects of the September 11, 2001 attack on the New York City World Trade Center. J Hypertens 2005;279–84.

37. Lampert R, Jain D, Burg MM, Batsford WP, McPherson CA. Destabilizing effects of mental stress on ventricular arrhythmias in patients with implantable cardioverter-defibrillators. Circulation 2000;101:158–64.

38. Rusting CL, Nolen-Hoeksema S. Regulating responses to anger: effects of rumination and distraction on angry mood. J Pers Soc Psychol 1998; 74:790–803.

39. Neumann SA, Waldstein SF, Sollers JJ, Thayer JF, Sorkin JD. Hostility and distraction have differential influences on cardiovascular recovery from anger recall in women. Health Psychol 2004;23:631–40.

40. Chambers L, Davidson K. Destructive anger rumination predicts resting blood pressure in older but not younger participants [Abstract]. Ann Behav Med 2000;22:S204.

41. Wesseling KH. Finapres, continuous noninvasive finger arterial pressure based on the method of Peñaz. In: Meyer-Sabellek W, Anlauf M, Gotzen R, Steinfeld L, eds. Blood Pressure Measurement. Darmstadt, Germany: Steinkopff Verlag; 1990.

42. Kurki T, Smith NT, Head N, Dec-Silver H, Quinn A. Noninvasive continuous blood pressure measurement from the finger: optimal measurement conditions and factors affecting reliability. J Clin Monit Comput 1987;3:6–13.

43. Smith NT, Wesseling KW, De Witt B. Evaluation of two prototype devices producing noninvasive pulsatile calibrated blood pressure from a finger. J Clin Monit Comput 1985;1:17–29.

44. Wesseling KH, Settels JJ, van der Hoeven GM, Nijboer JA, Butijn MW, Dorlas JC. Effects of peripheral vaso-construction on the measurement of blood pressure in a finger. Cardiovasc Res 1985;19:139–45.

45. Imholz BP, van Montfrans GA, Settels JJ, van der Hoeven GM, Karemaker JM, Wieling W. Continuous noninvasive blood pressure monitoring: reliability of Finapres device during the Valsalva manoeuvre. Cardiovasc Res 1988;22:390–7.

46. Parati G, Casadei R, Di Renzo M, Mancia G. Comparison of finger and intra-arterial blood pressure monitoring at rest and during laboratory testing. Hypertension 1989;13:647–55.

47. Gerin W, Pieper C, Marchese L, Pickering TG. The reliability of cardiovascular change scores: a comparison of intermittent vs continuous methods. J Psychom Res 1993;37:1–9.

48. Christenfeld N, Gerin W, Glynn LM. The assessment of cardiovascular recovery from stress using continuous blood pressure measurement. Psychophysiology 1995;32(suppl):S9.

49. Kario K, Schwartz JE, Gerin W, Robayo N, Maceo E, Pickering TG. Psychological and physical stress-induced cardiovascular reactivity and diurnal blood pressure variation in women with different work shifts. Hypertens Res 2002;25:543–51.

50. Gerin W, Pieper C, Pickering TG. Anticipatory and residual effects of an active coping task on pre and post-stress baselines. J Psychosom Res 1994;38:139–49.