Vagal Rebound and Recovery From Psychological Stress

ELIZABETH SIBOLBORO MEZZACAPPA, PHD, ROBERT M. KELSEY, PHD, EDWARD S. KATKIN, PHD, AND RICHARD P. SLOAN, PHD

Objective: To characterize cardiovascular recovery and examine the possible relationship of vagal activity and reflexes to risk for heart disease. **Methods:** Subjects performed cold pressor and mental arithmetic tasks. Heart rate, heart period variability, and pre-ejection period were obtained for 1 minute before, during, and after each task (Experiment 1). In the second experiment, subjects performed a Stroop color-word task and a mental arithmetic task. Heart rate, heart period variability, blood pressure, and baroreflex sensitivity were obtained during the 5-minute baseline, task, and recovery periods (Experiment 2). **Results:** In Experiment 1, heart rate during recovery was lower than baseline despite continued pre-ejection period shortening, whereas recovery heart period variability was higher than baseline. In Experiment 2, blood pressure increased throughout the session. However, recovery heart rate after mental arithmetic was lower than baseline heart rate, and heart period variability was higher during both recovery periods than during baseline. Vagal rebound, a sharp increase in variability in the first minute of recovery, was reduced in men in Experiment 1 and in individuals with a family history of cardiovascular disease in Experiment 2 and was associated with degree of change in baroreflex sensitivity between task and rest. **Conclusions:** Cardiovascular recovery from stress is associated with increased vagal modulation despite residual sympathetic activation. Vagal rebound may be involved in mechanisms resetting the baroreflex sensitivity at the onset and offset of stress. Diminished vagal rebound during recovery from stress is associated with standard risk factors for cardiovascular disease. The results support an association between attenuated vagal reflexes and risk for cardiovascular disease. **Key words:** Cardiac recovery, psychological stressors, cardiovascular disease, vagal reflexes, sympathetic nervous system, parasympathetic nervous system.

ATRAMI = Autonomic Tone and Reflexes After Myocardial Infarction; BP = blood pressure; BRS = baroreflex sensitivity; CP = cold pressor; DBP = diastolic blood pressure; ECG = electrocardiogram; HR = heart rate; HPV = heart period variability; HRV = heart rate variability; ICG = impedance cardiographic; MA = mental arithmetic; MI = myocardial infarction; PNS = parasympathetic nervous system; PEP = pre-ejection period; rMSSD = root mean squared successive difference; SBP = systolic blood pressure; SNS = sympathetic nervous system;

INTRODUCTION

Low levels of tonic vagal cardiac control are well established as a risk factor for cardiovascular events and illness (1–3). Additionally, reduced vagal reflex activity has been associated with greater risk of cardiac mortality after MI (4). The autonomic tone and reflexes after myocardial infarction (ATRAMI) investigation of 1284 patients found that cardiac mortality in the months after MI was related to low levels of HRV and reduced BRS, independent of left ventricular ejection fraction and frequency of ventricular arrhythmias. Moreover, HRV and BRS were independent predictors, with BRS adding significantly to the predictive value of HRV alone, leading the authors to conclude that augmented reflex vagal activity contributes to survival after MI (4).

Cole et al. (5) assessed 2428 heart disease patients who underwent routine exercise testing. They found that heart rate recovery, defined as the decrease in HR from peak exercise to one minute after cessation of exercise, was inversely related to death 6 years later even after controlling for the HR increase during exercise, exercise workload, cardiac risk and demographic factors. Cole et al. (5) proposed that the delayed fall in HR during the first minute after exercise was a function of decreased reflex vagal activity (6). Thus, it seems that an attenuated vagal reflexive response to the termination of exercise stress is a powerful predictor of overall mortality.

Recovery from psychological stress also has been linked with cardiovascular disease risk (7–16). However, these studies generally focus on HR or BP only, and not on the underlying components of HR or BP. This article examines the recovery phenomena observed in two independent studies in two different laboratories. Our goal was to evaluate cardiovascular responses and underlying SNS and PNS reflexes after the termination of psychological stress. We were particularly interested in vagal reflex activity during recovery in view of its apparent relationship to cardiovascular disease (4, 5).

From the Behavioral Medicine Program (E.S.M., R.P.S.), Columbia University, New York; Department of Psychology (E.S.M., E.S.K.), State University of New York at Stony Brook, New York; and Department of Pediatrics (R.M.K.), University of Tennessee Health Science Center, Memphis, Tennessee.

Address reprint requests to: Elizabeth Sibolboro Mezzacappa, Behavioral Medicine Program, Box 427, New York Presbyterian Hospital, 622 W. 168th Street, New York, NY 10032-3784. Email: esm@columbia.edu

Received for publication May 26, 2000; revision received November 1, 2000.

0033-3174/01/6304-0650

EXPERIMENT 1

Methods

The specific aim of the first study was to examine HR and underlying SNS and PNS contributions to cardiac function before, during, and after standard laboratory stressors. Sympathetic cardiac reactions were indexed by PEP, the time between the onset of depolarization of the ventricle and the opening of the aortic valve. The PEP is inversely related to the degree of sympathetic activation—the shorter the PEP, the greater the SNS arousal (17–19). Parasympathetic activity was indexed by the rMSSD of the RR intervals. The rMSSD is a measure of high frequency HPV that is positively correlated with the degree of vagal cardiac control (1–3).

Subjects. Data were obtained from 12 men and 15 women who participated as controls in a larger study on the effects of breastfeeding on reactivity to stress. They were recruited from undergraduate introductory psychology classes at SUNY at Stony Brook and by flyers posted around campus and the nearby university hospital. The subjects were between 18 and 45 years old and free of any medical condition affecting the cardiovascular system. Except for birth control, or antibiotic and topical treatments, subjects were not taking any medications. Their mean age was 25.4 years (SD = 5.6); 44.4% were white, 14.8% were African American, 29.6% were Asian, and 7.4% were of mixed ethnicity. Timing of evaluations of women was not related to the menstrual cycle. Subjects received $20 or class credit as compensation for the 2-hour study.

Apparatus. Thoracic impedance was assessed with a Model 304B Minnesota Impedance Cardiograph (Instrumentation for Medicine, Greenwich, CT) using tetrapolar aluminum-Mylar band electrodes (Instrumentation for Medicine). Voltage recording bands were placed around the base of the neck and around the thorax at the level of the xiphisternal junction. Bands for impressing the AC current were placed 3 cm above the base of the neck and 3 cm below the xiphisternal junction (19).

Tasks. Two tasks involving different patterns of autonomic activation were used—spoken MA and CP (20). The mental arithmetic task, which evokes vagal withdrawal and a predominantly beta-adrenergic response, required subjects to subtract serially by 7s from a 4-digit number for 1 minute. Subjects were instructed to work as quickly and accurately as possible. The cold pressor task, which evokes vagal withdrawal and a predominantly alpha-adrenergic response, required immersion of the dominant hand up to the wrist in 1 liter of an ice/water mix at 4°C for 1 minute. The order of task presentation was counterbalanced.

Procedure. After consent was obtained and a brief explanation of the experiment was given, subjects completed questionnaires for the larger study and electrodes were applied for impedance cardiographic measures. After further instructions about the tasks, subjects completed additional questionnaires for the larger study.

After a 10-minute resting baseline, of which the last 5 minutes were recorded, subjects completed the 1-minute mental arithmetic and cold pressor tasks in random order. Subjects received task-specific instructions immediately before each task period. One minute of recovery data were recorded immediately after the termination of each task, and an additional 5-minute baseline rest period separated the two tasks. After the last recovery minute, the instruments were removed, and the subjects were debriefed and released.

Signal processing. The ECG and dZ/dt signal (the first derivative of the change in thoracic impedance) from the impedance cardiograph were digitized at 500 Hz, whereas the Z_0 signal (nasal thoracic impedance) from the impedance cardiograph was digitized at 250 Hz. The digitized signals were scored on a beat-to-beat basis and then the ensemble averaged with reference to the peak of the ECG R-wave (19, 21, 22). A trained researcher visually inspected each ensemble-averaged waveform after a procedure reported previously (19, 22). Beat-to-beat averages for HR and ensemble averages for PEP were calculated for the last minute of the baseline period preceding each task, during the 1 minute of each of the tasks, and during the 1 minute of recovery after each task. The rMSSD was calculated for each minute, using a program that was specially developed for that purpose.

Results

Figure 1 depicts the means and standard errors for HR, PEP, and rMSSD during the 1-minute baseline, task, and recovery periods for CP and MA. The rMSSD data were skewed significantly, so a natural log transformation was applied before analysis. Each cardiovascular measure was analyzed using the MANOVA approach to repeated measures with task (CP/MA) and period (baseline/task/recovery) as within-subject factors. The critical comparisons involved planned contrasts of recovery vs. baseline values and recovery vs. task values for HR, PEP, and rMSSD.

The main effect of period was significant (multivariate $F(6,102) = 15.92$, $p < .001$). More specifically, HR was lower, PEP was shorter,

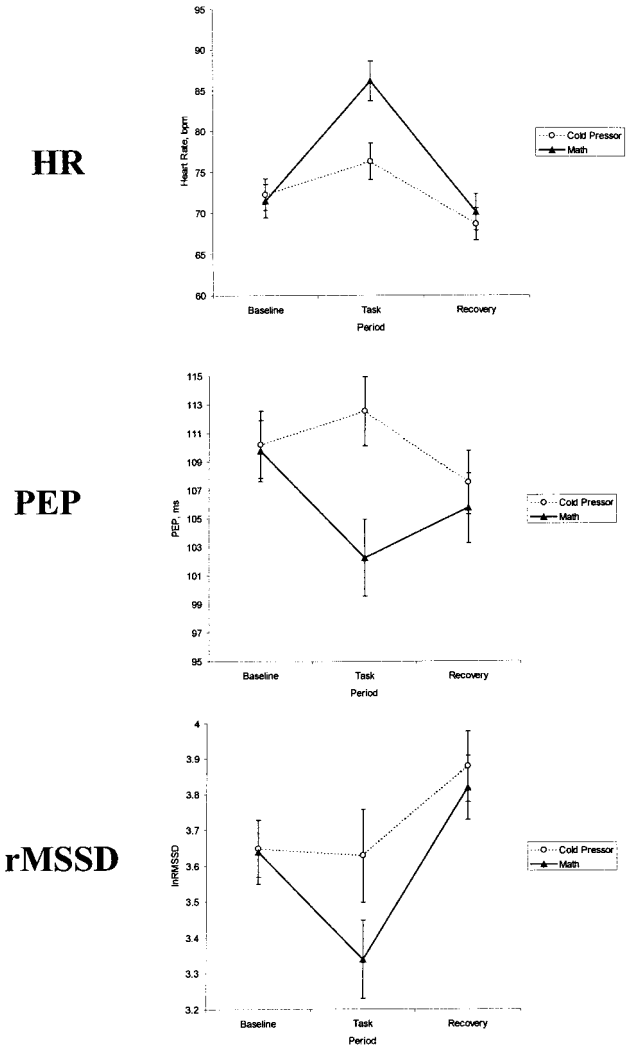

Fig. 1. HR, PEP, and rMSSD during baseline, task, and recovery for the MA and CP tasks in Experiment 1.

and rMSSD was greater in recovery than during baseline, $F(1,26) = 20.97$, 12.10, and 15.93, respectively, all $p < .01$. Moreover, HR was higher and rMSSD was lower during task than recovery, $F(1,26) = 86.34$ and 27.44, both $p < .001$; however, the difference between task and recovery was not significant for PEP. Significant task (multivariate $F(3,24) = 9.19$, $p < .001$) and task × period (multivariate $F(6,102) = 6.88$, $p < .005$) effects reflected greater increases in HR and decreases in PEP and rMSSD during mental arithmetic than during cold pressor. A decrease in rMSSD occurred in 55.6% of subjects during CP and in 74.1% of subjects during MA.

Vagal rebound. These data suggest that the HR deceleration in recovery is vagally mediated, as it is consistent with rMSSD changes rather than PEP changes. This HR deceleration during recovery from psychological stress is reminiscent of the postexercise recovery pattern that Cole et al. (5) linked to reduced mortality in heart disease patients 6 years after testing. As a preliminary test of whether vagally mediated cardiac recovery is linked to disease risk, the change in rMSSD from each task to recovery (rMSSD rebound) was calculated for each subject and tested for association with gender, an established risk factor for cardiovascular disease.

Periods in Experiment 1 were 1 minute each; therefore, rMSSD rebound from each task was calculated by subtracting mean variability during the 1-minute task from mean variability in the minute after task termination. Degree of rMSSD rebound for women ($N = 15$) and men ($N = 12$) was compared using a MANOVA with gender as the between-subjects factor and task (cold pressor or mental arithmetic) as the within-subjects factor. The means and standard errors for the groups are presented in Figure 2. Collapsing across task, women showed significantly greater rMSSD increases from task to recovery than men, $F(1,25) = 6.70$, $p < .05$.

Because rMSSD rebound may have been associated with the degree of vagal withdrawal during the task, gender differences in rMSSD decreases from baseline to the tasks were analyzed. Vagal withdrawal during each task was calculated by subtracting mean variability during the 1-minute baseline immediately preceding the task from mean variability during the 1-minute task. MANOVA (gender, task) revealed no gender differences in vagal withdrawal.

Summary

One minute after termination of psychological stress, HR fell below baseline level. This fall in HR was apparently mediated by a sharp rebound in vagal activation after stress, as indicated by increased rMSSD during recovery, despite persistent residual sympathetic activation, as indicated by PEP. This vagal rebound may be crucial in restoring cardiovascular homeostasis.

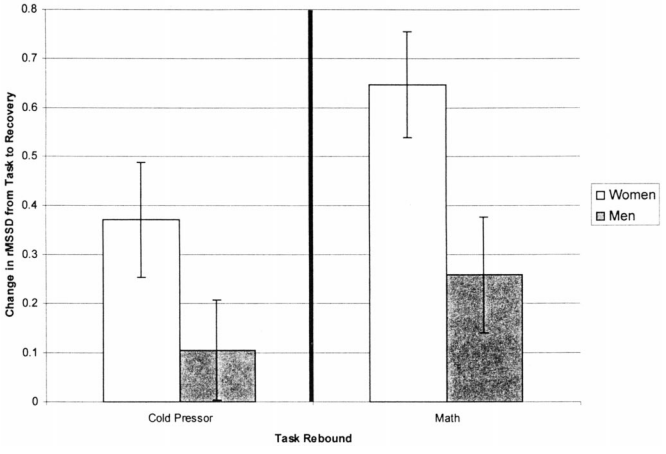

Fig. 2. Vagal rebound by gender in Experiment 1.

Vagal rebound was greater for women than for men. These findings are consistent with previous reports that women show greater HR overcompensation (HR that is lower in recovery than baseline) than men after harassment stress (8). The difference in rebound was not accounted for by differences in vagal withdrawal.

EXPERIMENT 2

Generalization of the findings of Experiment 1 may be limited because of the brief 1-minute stressor and recovery periods. The observed recovery phenomenon might disappear during longer stressor periods due to habituation of cardiovascular reactivity (23). Accordingly, Experiment 2 extended the original investigation by using longer task and recovery periods, as well as different tasks. In addition, measures of systolic and diastolic blood pressure were included to permit assessment of baroreflex sensitivity.

BRS reflects the ability to alter vagal and sympathetic activity in response to sudden changes in blood pressure (24). Baroreflex sensitivity can be reset and tends to decrease under stress (24, 25). In addition, derangements in resetting of baroreflex sensitivity are thought to play a role in the etiology of hypertension (26).

Because the baroreflex is an established PNS reflex that also has an association with future mortality (4), rMSSD rebound was tested for associations with BRS. In addition, because BRS changes in response to stress, rMSSD rebound was tested for associations with changes in BRS.

Methods

Subjects. Data came from 19 women and 12 men who volunteered for a study on the effects of fragrance on cardiovascular reactivity. Subjects were studied on two occasions, once to perform the tasks while exposed to fragrance and once to perform the tasks under normal room air conditions, with order of conditions counterbalanced. Data presented here are from the normal air session only. Analyses showed no significant effect of order of fragrance condition (normal air or fragrance condition first); therefore, data from the normal air sessions were combined.

The mean age of the subjects was 34.0 ± 8.6 years; 6% were of blended ethnicity, 6% were Latino, 19% were African American, and 68% were white. All subjects were free of any medical condition or medications that might affect the cardiovascular system. They were paid a total of $80 for the two sessions.

Tasks. Stroop Color-Word Task. Subjects were presented with color names (blue, green, yellow, and red) displayed in colors that were either congruent or incongruent with the names. Subjects pressed a key on a keypad that corresponded to the display color of the letters. The computer paced the task and delivered an error message whenever a subject entered an incorrect response or failed to respond rapidly enough.

Mental arithmetic. Subjects were presented with a four-digit number on a computer monitor and were instructed to subtract serially by 7s starting with this number, entering their response on a keypad. The first number disappeared after the first answer was entered on the keypad. Subjects received verbal prompts (eg, "please subtract faster") at random intervals. The computer did not pace this task, but subjects were instructed to subtract as quickly and as accurately as possible.

Signal acquisition and processing. ECG electrodes were placed on the right shoulder, on the left anterior axillary line at the 10th intercostal space, and in the right lower quadrant. Analog ECG signals were digitized at 500 Hz by a National Instruments A/D

board and passed to a microcomputer. The ECG waveform was submitted to a specially written R-wave detection routine, resulting in an RR interval series. Errors in marking of R-waves were corrected interactively. The rMSSD of the RR intervals served as the measure of HPV.

An Ohmeda Finapres 2300 monitor (Ohmeda, Englewood, CO) was used to measure BP. The finger cuff was placed on the left hand. Finapres measures were calibrated by moving the arm so that readings fell within ±10 mmHg of a manual blood pressure reading. The servo self-adjustment was disabled except for the last minute of each period, so that the calibration signal would not interfere with the derivation of the BRS. The analog blood pressure waveform was digitized at 500 Hz and collected by the microcomputer. Systolic and diastolic pressures were identified on the pressure waveform by a specially written program. Errors in marking systole and diastole were corrected interactively. The BRS was calculated from the ECG and SBP measures by the sequence method (27, 28). The BRS calculated for each period was based on its first 4 minutes, because the servo self-adjustment was enabled for the last minute.

Procedure. After giving informed consent, subjects were seated in a comfortable chair. Then electrodes for ECG were applied and the finger cuff for BP measurement was fitted on the subjects. After instrumentation, they completed questionnaires and procedures relevant to the fragrance study and then received instructions about the Stroop and mental arithmetic tasks. The tasks were presented in fixed order. Subjects rested for 5 minutes to provide baseline measures. Subjects then performed the 5-minute Stroop task, after which they rested for 5 minutes during an interim recovery period. Subjects then performed the 5-minute mental arithmetic task, after which they rested for a final 5-minute recovery period. The ECG and BP were recorded continuously. After additional questionnaires and procedures relevant to the fragrance study, instruments were removed, and subjects were paid and released.

Results

Heart rate and rMSSD. Figure 3 presents the means and standard errors for HR and rMSSD during the 5-minute baseline, interim, and final recovery periods, as well as the two stressor periods. The rMSSD data were skewed significantly, so a natural log transformation was applied before analysis. Heart rate and rMSSD were analyzed using a MANOVA approach to repeated measures with period (baseline/interim recovery/final recovery) and minute (1–5) as within-subjects factors. The critical comparisons were between the baseline and the two recovery periods (interim and final). Significant multivariate main effects occurred for period (multivariate $F(4,26) = 4.78$, $p < .005$) and minute (multivariate $F(8,22) = 4.06$, $p < .005$). Heart rate during baseline was significantly higher than during final recovery, $F(1,29) = 4.10$, $p < .05$. Moreover, as shown in Figure 3, rMSSD levels were higher than baseline during both the interim recovery period, $F(1,29) = 14.42$, $p < .0005$, and the final recovery period, $F(1,29) = 4.51$, $p < .05$. A decrease in rMSSD occurred in 83.3% of subjects during Stroop and in 67.7% of subjects during mental arithmetic.

Blood pressure. Data from three female subjects were dropped because of spurious readings (consistent extreme outlier values of SBP > 190 or DBP > 100). Figure 4 presents the SBP and DBP means and standard errors for minutes one through four during the baseline, interim, and final recovery periods, as well as the two stressor periods. The fifth minute of each period was deleted because the Finapres BP monitor was recalibrated during that time. Repeated measures MANOVA with period (baseline/interim recovery/final recovery) and minute (1–4) as within-subjects factors revealed a significant multivariate effect of period, ($F(4,104) = 3.38$, $p < .01$).

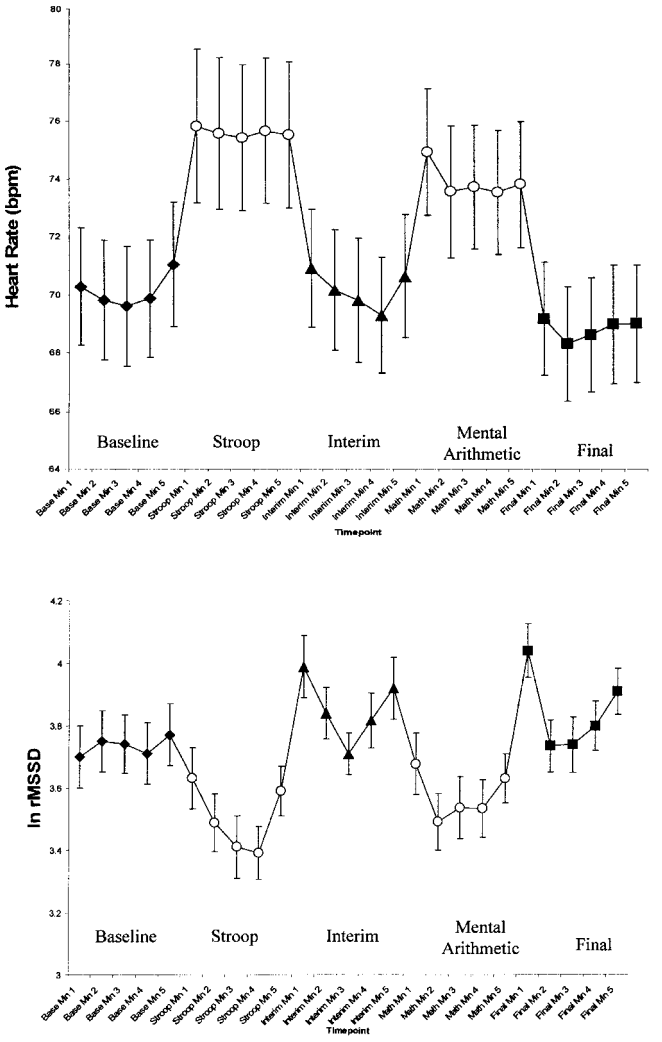

Fig. 3. HR and rMSSD throughout the session for baseline, Stroop, interim recovery, mental arithmetic, and final recovery in Experiment 2.

Separate analyses indicated that this effect held for both SBP ($F(2,52) = 6.70$, $p < .005$) and DBP ($F(2,42.88) = 6.49$, $p < .01$). Contrast analyses indicated that SBP and DBP were significantly higher than baseline during both the interim and final recovery periods (SBP, $F(1,26) = 8.35$ and 9.64, respectively, both $p < .01$; DBP, $F(1,26) = 5.73$ and 8.55, respectively, both $p < .05$).

Vagal rebound and baroreflex sensitivity. The mean and standard errors for BRS are presented in Figure 5. Consistent with previous reports, BRS decreased under stress and rose again after termination of stress (25). Correlations between BRS for each period and rMSSD rebound were calculated for each task. In addition, BRS change scores were calculated from pretask baseline to task (Stroop and mental arithmetic) and task to recovery. These change scores represent the resetting of the BRS from period to period. Correlations were calculated between these change scores and rMSSD rebound.

Except for a significant correlation between rMSSD rebound from MA and BRS during MA ($r = -.40$) there was little relationship between rMSSD rebound and BRS levels. However, rMSSD rebound correlated significantly with the change in BRS from periods of rest to stress and from periods of stress to rest. That is, rMSSD rebound

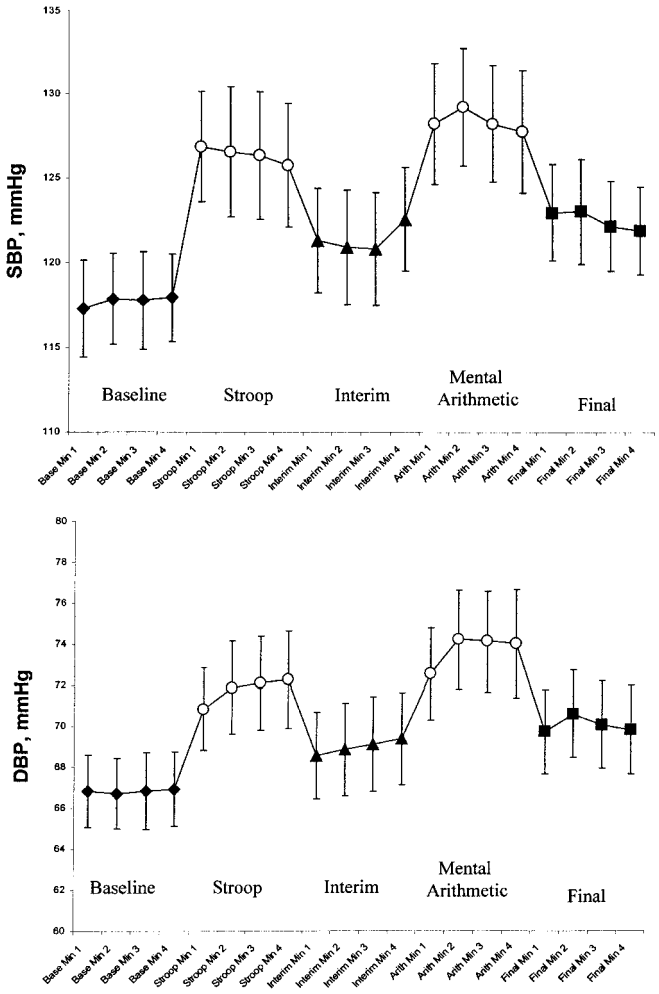

Fig. 4. SBP and DBP throughout the session for baseline, Stroop, interim recovery, mental arithmetic, and final recovery in Experiment 2.

correlated inversely with subsequent BRS changes to the onset of stress ($r = -.34$ to $-.52$, all $p < .05$) and correlated positively with subsequent BRS changes to the offset of stress ($r = .36$ to $-.49$, all $p < .05$). In general, the greater the rMSSD rebound, the greater the BRS decline to stress and the greater the BRS enhancement after stress. Thus, vagal rebound may contribute to the resetting of BRS in response to the onset and offset of stress.

Vagal rebound. A prominent feature of cardiac recovery from stress is a rapid increase in HPV in the first minute of recovery, suggesting a PNS surge that decelerates HR quickly and promotes recovery from stress. To examine the relevance of this PNS response to health, the degree of rMSSD recovery from each task was calculated for each subject and tested for association with standard risk factors for cardiovascular disease: gender and family history of heart disease.

Periods in Experiment 2 were 5 minutes long. Following the procedures of Cole et al. (5), rMSSD was calculated as the change in rMSSD from its minimum minute value during Stroop or mental arithmetic to the first minute after termination of each task. That is, rMSSD rebound was calculated from maximal vagal withdrawal during the stressor to the first minute of recovery.

In Experiment 2, degree of rMSSD rebound was compared between women ($N = 18$) and men ($N = 12$) and between those with a positive ($N = 11$) and negative ($N = 18$) family history of cardiovascular disease. Family history was assessed through an interview in response to the question "Is there any history of cardiovascular disease in your family?" Research on undergraduates indicates that family history information gathered this way might be inaccurate (29). However, in this study, the subjects were primarily medical personnel, older and more sophisticated than undergraduates in their knowledge of cardiovascular disorders. Moreover, for all but one subject, the information reported involved highly salient events (death from cardiovascular disease, surgery), which are typically reported accurately.

The rMSSD rebound of the groups was compared using MANOVA with gender or family history as the between-subjects factor and tasks (Stroop and mental arithmetic) as the within-subjects factor. The means and standard errors for the groups are presented in Figure 6, which shows that subjects with a positive family history of cardiovascular disease had significantly smaller rebound than subjects with a negative family history ($F(1,27) = 4.37$, $p < .05$). A significant task × family history interaction indicated that this difference primarily was due to responses to the mental arithmetic task ($F(1,27) = 9.17$, $p < .005$).

To examine possible effects of vagal withdrawal during tasks on vagal rebound, family history differences in rMSSD decreases to the tasks were analyzed. Vagal withdrawal during each task was calculated by subtracting the mean variability during the 5-minute baseline immediately preceding each task from the mean variability during the 5-minute task. A MANOVA (family history of cardiovascular disease, task) revealed no family history differences in vagal withdrawal.

Similar results were obtained when rebound was calculated as the difference in rMSSD between the first minute of task (not peak withdrawal) and the first minute of the recovery. Although the main effect of family history was no longer significant, a highly significant task × family history interaction remained ($F(1,27) = 10.84$, $p < .005$). Separate follow-up t tests for Stroop and mental arithmetic revealed that subjects with a positive family history of cardiovascular disease had significantly smaller rebound after mental arithmetic than subjects with a negative family history ($t(27) = 2.86$, $p < .01$). Family history showed no association with rebound after Stroop. There were no significant effects of gender on rMSSD rebound calculated by either method, although the pattern of mean differences during mental arithmetic was similar to that during mental arithmetic in Experiment 1.

Summary

Results from analyses of HR and rMSSD levels from Experiment 2 were generally consistent with those from Experiment 1. HR during recovery from the second task (mental arithmetic), but not the first task (Stroop), was slower than during baseline. Also as in Experiment 1, rMSSD was greatest during both the interim and final recovery periods. Moreover, as Figure 3 reveals, rMSSD increased sharply in the critical first minute of recovery, then declined. In contrast, both SBP and DBP climbed progressively higher from baseline, to interim recovery, to final recovery. Vagal rebound, calculated as the increase in rMSSD from maximal or first minute of task to first minute of recovery, was associated with family history of cardiovascular disease and degree of BRS change to the onset and offset of stress, but not with gender.

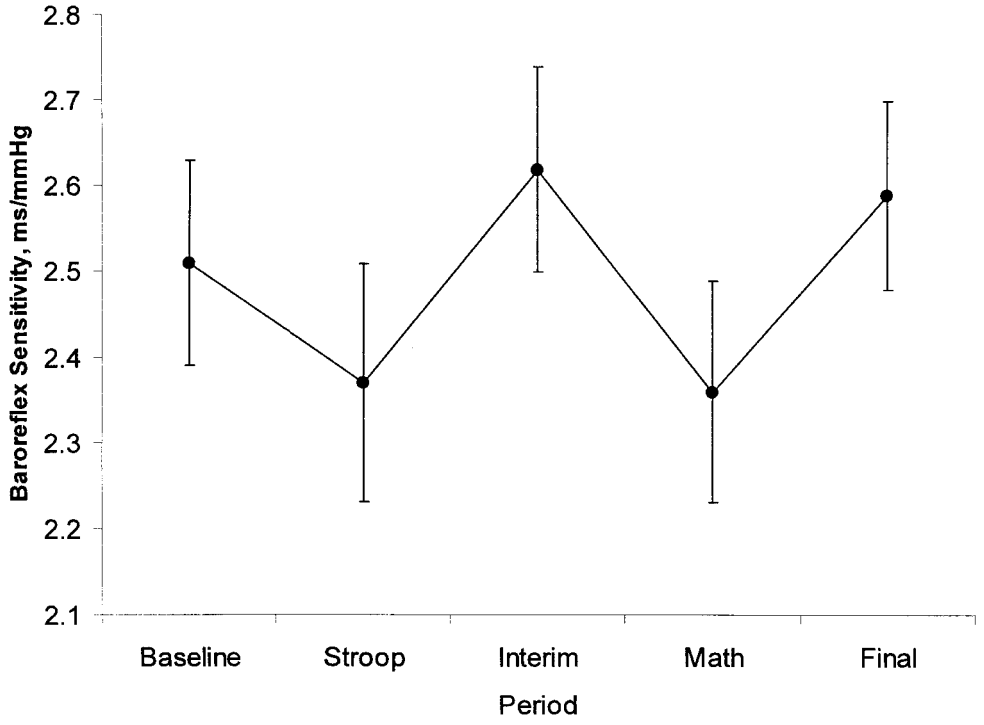

Fig. 5. BRS throughout the session for baseline, Stroop, interim recovery, mental arithmetic, and final recovery in Experiment 2.

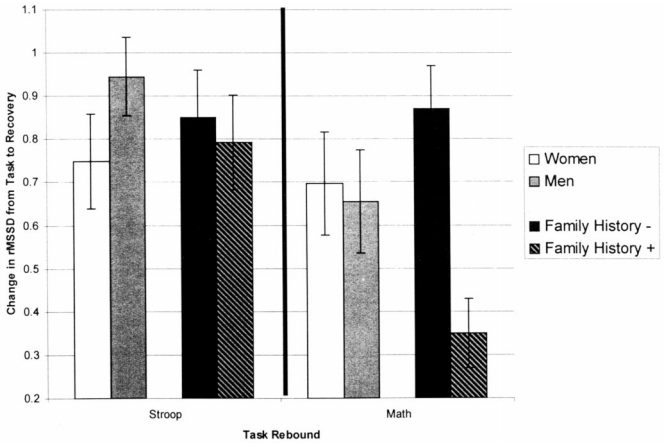

Fig. 6. Vagal rebound by gender and family history in Experiment 2.

DISCUSSION

Vagal Rebound and Recovery

Recent research suggests that recovery of heart rate and blood pressure after stress is a process closely related to resting and reactivity measures, and may be described by the general parameters of level, amount, and speed of recovery (30). Results from our research examining the underlying autonomic components identify a sharp increase in HPV as a distinct feature in the parasympathetic recovery process, indicating a surge in vagal activity at the offset of psychological stress. Thus, our work adds to the formation of general concepts, descriptions, and topography of recovery processes, especially the concept of vagal rebound.

Refinement of the notion of vagal rebound should be the focus of future parametric investigations. Vagal rebound may be calculated in several different ways with varying degrees of association with resting and reactivity measures. In Experiment 2, vagal rebound was calculated as a change score from the maximal rMSSD withdrawal to the first minute of recovery. Because Cole et al. (5) used a similar method to demonstrate a link between HR recovery from stress and subsequent mortality, this method of calculating rebound seemed a logical first step.

Yet, alternative methods could be used to calculate vagal rebound. Differences between first, last, mean task or resting values and first or maximal recovery rMSSD are possible variants. Moreover, derived amplitude/magnitude as well as timing/speed measures are also of interest. Although logically related, the different computations of vagal rebound may reflect differing components. Depending on task length, vagal rebound calculated from the overall task means may reflect varying processes of onset, offset, and habituation during the task (23).

Heart Rate and Recovery

Results from both experiments showed that heart rate was slower in recovery than during baseline. It

might be argued that this HR overcompensation resulted from anticipatory stress and elevated HR during the initial baseline period, and that this recovery effect would disappear in comparisons with longer baseline periods. However, as Figure 2 suggests and statistical analyses confirmed, HR was stable within the initial 5-minute baseline rest period, so inclusion of a longer baseline period probably would not have altered the results from Experiment 2. Moreover, Experiment 1 included an initial baseline period of 10 minutes and yielded similar HR results. Thus, consistent with recent findings of equivalent cardiovascular reactivity to stress after initial baseline periods lasting from 5 to 16 minutes (23), it seems that HR overcompensation during recovery is largely unaffected by initial baseline duration. Accordingly, it seems likely that the decreased HR during recovery resulted from an active compensatory recovery process.

Vagal Reflexes: Withdrawal and Rebound

As noted, some subjects did not show a vagal withdrawal in response to stress, although vagal rebound was calculated in the same manner for all subjects. For participants who do not show vagal withdrawal under stress, it seems logical to assume that there would be little or no rebound. Traditionally, attenuated reactivity has been conceived of as protective, and recovery has been conceived of as repairing normal or exaggerated perturbations induced by stress; however, this may not always be so. An attenuated stress response, when coupled with attenuated recovery, may predict later illness. Cole et al. (5) reported that patients with attenuated HR recovery (and increased risk of mortality 6 years later), also showed attenuated HR responses to stress. Thus, it may be that the vagal reflexes that control responses to stress are mechanistically related to the vagal reflexes that control recovery from stress. That is, a lack of vagal withdrawal and lack of vagal rebound may reflect the same general failure of the parasympathetic nervous system to adapt. Investigations using simultaneous assessment of reactivity and recovery with sympathetic and parasympathetic measures are needed to uncover underlying mechanisms linking responses to stress and risk for illness.

Finally, reactivity and recovery may be more or less independently predictive, with attenuated or exaggerated responses additively or interactively determining risk. Group difference comparisons in these studies support this stance, because differences in vagal rebound during recovery were not explained readily by group differences in vagal withdrawal during task.

Vagal Rebound and Risk for Cardiovascular Disease

The results of Experiment 1 suggest that the offset of psychological stress involved a rapid surge in PNS activity that decelerates HR quickly and promotes recovery despite any residual sympathetic activation. The HR deceleration during recovery is reminiscent of the postexercise recovery pattern that Cole et al. (5) linked to reduced mortality 6 years after testing. These findings parallel the reports of Cole et al. (5) and thus provide an insight into mechanisms underlying that association. Moreover, they suggest that vagal rebound after psychological stress may be predictive of cardiovascular health.

Vagal rebound was reduced in subjects with a family history of cardiovascular disease and gender, but only in Experiment 1. The conflicting findings may stem from the modest sample sizes and lack of power, or from differences in the tasks used. Also relevant is the finding that vagal rebound may contribute to changes in BRS in response to the onset and offset of psychological stress. These findings suggest that additional research on vagal rebound in recovery from psychological stress may provide important information relevant to cardiovascular disease risk.

Summary and Significance

Immediately after the termination of a psychological stress there seems to be a sharp increase in HPV, a vagal rebound that results in slower HR during recovery than during baseline. Vagal rebound is attenuated in subjects who have relatively higher risk for later cardiovascular disease (men and those with a positive family history of disease) compared with subjects who have relatively lower risk (women and those with a negative family history of disease). Additional prospective longitudinal investigations are necessary to determine the use of vagal rebound in predicting cardiovascular disease. Moreover, the results indicate that vagal rebound is associated with alterations in the baroreflex sensitivity, another vagal reflex that is predictive of mortality (4). Our results and those of others (4, 5) support an association between an attenuated vagal reflex and risk for cardiovascular disease.

REFERENCES

1. Berntson G, Bigger J, Eckberg D, Grossman P, Kaufman P, Malik M, Nagaraja H, Porges S, Saul J, Stone P, Van der Molen M. Heart rate variability: origins, methods, and interpretive caveats. Psychophysiology 1997;34:623–48.
2. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate

variability: standards of measurement, physiological interpretation, and clinical use. Circulation 1996;93:1043–65.
3. Kleiger R, Bigger J, Bosner M, Chung M, Cook J, Rolnitzky L, Steinman R, Fleiss J. Stability over time of variables measuring heart rate variability in normal subjects. Am J Cardiol 1991;68: 626–30.
4. La Rovere M, Bigger J, Marcus F, Mortara A, Schwartz P. Baroreflex sensitivity and heart-rate variability in prediction of total cardiac mortality after myocardial infarction. Lancet 1998;351: 478–84.
5. Cole C, Blackstone E, Pashkow F, Snader C, Lauer M. Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med 1999;341:1351–7.
6. Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, Takeda H, Inoue M, Kamada T. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. J Am Coll Cardiol 1994;24: 1529–35.
7. Brosschot J, Thayer J. Anger inhibition, cardiovascular recovery, and vagal functions: a model of the link between hostility and cardiovascular disease. Ann Behav Med 1998;20:326–32.
8. Earle T, Linden W, Weinberg J. Differential effects of harassment on cardiovascular and salivary cortisol stress reactivity and recovery in women and men. J Psychosom Res 1999;46:125–41.
9. Faber S, Burns J. Anger management style, degree of expressed anger, and gender influence cardiovascular recovery from interpersonal harassment. J Behav Med 1996;19:31–53.
10. Gerin W, Pickering T. Association between delayed recovery of blood pressure after acute mental stress and parental history of hypertension. J Hypertens 1995;13:603–10.
11. Gillin J, Mills P, Nelesen R, Dillon E, Ziegler M, Dimsdale J. Race and sex differences in cardiovascular recovery from acute stress. Int J Psychophysiol 1996;23:83–90.
12. Gregg M, James J, Mataya T, Thorsteinsson E. Hemodynamic profile of stress-induced anticipation and recovery. Int J Psychophysiol 1999;34:147–62.
13. Jackson R, Treiber F, Turner J, Davis H, Strong W. Effects of race, sex, and socioeconomic status upon cardiovascular stress responsivity and recovery in youth. Int J Psychophysiol 1999;31: 111–9.
14. Mills P, Berry C. Menstrual cycle, race and task effects on blood pressure recovery from acute stress. J Psychosom Res 1999;46: 445–54.
15. Schuler J, O' Brien W. Cardiovascular recovery from stress and hypertension risk factors: a meta-analytic review. Psychophysiology 1997;34:649–59.
16. Vitaliano P, Russo J, Paulsen V, Bailey S. Cardiovascular recovery from laboratory stress: biopsychosocial concomitants in older adults. J Psychosom Res 1995;39:361–77.
17. Berntson G, Cacioppo J, Binkly P, Uchino B, Quigley K, Fieldstone A. Autonomic cardiac control. III. Psychological stress and cardiac response in autonomic space revealed by pharmacological blockades. Psychophysiology 1994;31:599–608.
18. Mezzacappa E, Kelsey R, Katkin E. The effects of epinephrine administration on impedance cardiographic measures of cardiac function. Int J Psychophysiol 1999;31:189–96.
19. Sherwood A, Allen M, Fahrenberg J, Kelsey R, Lovallo W, van Doornen L. Methodological guidelines for impedance cardiography. Psychophysiology 1990;27:1–23.
20. Sherwood A, Turner J. A conceptual and methodological overview of cardiovascular reactivity research. In Turner JR, Sherwood A, Light KC, editors. Individual differences in cardiovascular response to stress. New York: Plenum Press; 1992. p. 3–32.
21. Kelsey R, Guethlein W. An evaluation of the ensemble averaged impedance cardiograph. Psychophysiology 1990;27:24–33.
22. Kelsey R, Reiff S, Wiens S, Schneider T, Mezzacappa E, Guethlein W. The ensemble averaged impedance cardiogram: an evaluation of scoring methods and interrater reliability. Psychophysiology 1998;35:337–40.
23. Kelsey R, Blascovich J, Tomaka J, Leitten C, Schneider T, Wiens S. Cardiovascular reactivity and adaptation to recurrent psychological stress: effects of prior exposure. Psychophysiology 1999; 36:818–31.
24. La Rovere M, Mortara A, Schwartz P. Baroreflex sensitivity. J Cardiovasc Electrophysiol 1995;6:761–74.
25. Steptoe A, Sawada Y. Assessment of baroreflex function during mental stress and relaxation. Psychophysiology 1989;26:140–7.
26. Zanchetti A, Mancia G. Cardiovascular reflexes and hypertension. Hypertension 1991;18 Suppl III:III-13–III-21.
27. Blaber A, Yamamato Y, Hughson R. Methodology of spontaneous baroreflex relationship assessed by surrogate data analysis. Am J Physiol 1995;268:H1682–7.
28. Watkins L, Grossman P, Sherwood A. Noninvasive assessment of baroreflex control in borderline hypertension: comparison with the phenylephrine method. Hypertension 1996;28:238–43.
29. Hastrup J, Hotchkiss A, Johnson C. Accuracy of knowledge of family history of cardiovascular disorders. Health Psychol 1985; 4:291–306.
30. Christenfeld N, Glynn L, Gerin W. On the reliable assessment of cardiovascular recovery: an application of curve-fitting techniques. Psychophysiology 2000;37:543–9