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Journal
Psychophysiology, 43(1)

ISSN
0048-5772

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Publication Date
2006

Peer reviewed

Hemodynamic Profile, Compensation Deficit, and Ambulatory Blood Pressure

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Abstract

This study hypothesized that physiologically grounded patterns of hemodynamic profile and compensation deficit would be superior to traditional blood pressure reactivity in the prediction of daily-life blood pressure. Impedance cardiography-derived measures and beat-to-beat blood pressure were monitored continuously in 45 subjects during baseline and four tasks. Ambulatory blood pressure measures were obtained combining data from one work and one off day. The mediating effects of gender and family history of hypertension were considered. Only gender was significantly associated with hemodynamicprofile. Regression analysis indicated that typical reactivity measures failed to predict everyday lifeblood pressure . After controlling for gender and baseline blood pressure, hemodynamic patterns during specific tasks proved to be strong predictors, overcoming limitations of previous reactivity models in predicting real-life blood pressure.

Descriptors: Ambulatory blood pressure, Cardiovascular reactivity, Gender, Family history of hypertension, Hemodynamic profile, Individual differences.

Hemodynamic Profile, Compensation Deficit, and Ambulatory Blood Pressure

It is widely held in physiology and medicine that altered stress reactions may signal the presence or risk of disease. In particular, the magnitude or pattern of an individual's hemodynamic response to behavioral stressors (cardiovascular reactivity) is hypothesized to mediate the relationship between stress and cardiovascular diseases. Despite the vast literature, findings of reactivity studies have not reached consistent consensus, and debates continue about the conclusiveness of the evidence. Some longitudinal studies are supportive of cardiovascular reactivity as a potential risk factor for hypertension or heart disease (Barnett et al., 1997; Carroll et al., 2003; Markovitz et al., 1998; Matthews, Woodall, & Allen, 1993), but others conclude that the pathogenic role of heightened reactivity in developing hypertension has only modest support (Carroll et al., 2001; Carroll et al., 1996). The larger prospective studies are not without limitations, such as the focus on the cold pressor test as an index of everyday life stress (Kasagi, Masazumi, & Shimaoka, 1995; Menkes et al., 1989), the small size of the samples (Borghi et al., 1986; Light, Dolan, Davis, & Sherwood, 1992), and the absence of women among subjects (Everson et al., 1996; Kamarck et al., 1997; Ming et al., 2004).

A review of recent findings bearing on the conceptualization and measurement of cardiovascular reactivity to psychological challenge (Kamarck & Lovallo, 2003) discusses methodological limitations of reactivity studies that may have restricted our ability to detect laboratory-to-life generalizability. One important limitation is the focus on blood pressure reactivity per se and not on underlying physiological determinants of blood pressure Recent studies suggest that reactivity to laboratory stressors may be distinguished by the degree to which cardiac or vascular mechanisms are predominant (Gregg, Matyas, & James, 2002; Kline et al., 2002). The rationale for investigations of responder type comes from the assumption that different patterns of compensatory changes in cardiac output and total peripheral resistance may be associated with blood pressureresponses of similar magnitude (Julius, 1988). The term

"hemodynamic profile" describes the relationship between cardiac output and total peripheral resistance in the homeostatic regulation of blood pressure (Gregg, James, Matyas, & Thorsteinsson, 1999). This approach was recently employed to characterize the hemodynamic effects of dietary caffeine, sleep restriction, and laboratory stress (James & Gregg, 2004). Moreover, besides the open debate about the pathogenic role of vascular or myocardial profiles, there is evidence of pattern differences in these variables as determinants of ambulatory blood pressure (Llabre et al., 1998).

A recent review documented the superiority of ambulatory over clinic blood pressure measurements in the prediction of major cardiovascular events (Verdecchia, Angeli, Gattobigio, & Porcellati, 2003). Ambulatory blood pressure monitoring has become a commonly used method of assessing the generalizability of laboratory-based cardiovascular reactivity to real-life settings. Significant associations between reactivity and ambulatory blood pressure means have been reported in a number of studies (Cornish, Blanchard, & Jaccard, 1994; Light, Turner, Hinderliter, & Sherwood, 1993; Steptoe & Cropley, 2000), but others have only found weak correlations between these two measures (Fredrikson, Robson, & Ljungdell, 1991; Majahalme et. al., 1998; Pickering & Gerin, 1988). Given that ambulatory 24-hr blood pressureis a highly established predictor of hypertensive target organ damage (Parati et al., 1987), the current study investigated whether profiles formed on the basis of patterns of cardiac output and total peripheral resistance reactivity to laboratory tasks relate more closely to ambulatory blood pressure levels than blood pressure reactivity itself.

Several strategies have been developed in order to obtain a taxonomy for classifying individuals based on their cardiac and vascular responses during psychological stress. A relatively common method is the use of residualized change scores to a beta-adrenergic task (Girdler, Turner, Sherwood, & Light, 1990; Kasprowicz et al., 1990; Sherwood, Dolan, & Light, 1990). In a recent paper, Gregg, Matyas, and James (2002) expressed caution about the categorical nature of earlier methods of classification. These authors criticized previous taxonomies, as they ignored the

continuous nature of cardiovascular measurements. In fact, previous attempts used linear, additive models (analysis of variance, bivariate regression, and multivariate regression), ignoring the multiplicative relationship between cardiac output and total peripheral resistance (Guyton, 1987). In addition, Gregg at al. (2002) suggested that the use of ratio scores instead of difference scores of reactivity could be a better way to deal with the physiological relationship between hemodynamic parameters. Furthermore, the authors noted that the same hemodynamic profile may differ in the extent to which total peripheral resistance and cardiac output compensate for each other and they demonstrated that failure to compensate is a critical issue in the prediction of blood pressure. Given these assumptions and considerations, the present paper assessed participants using two independent parameters (Gregg et al., 2002): "the extent to which they compensate" (compensation deficit) and "the way in which they compensate" (hemodynamic profile). These parameters were treated as continuous measures in the present study.

Reliability reflects the stability of a measure across time and settings, and it is considered one of the weakest points of the reactivity hypothesis (Gerin et al., 1998). Aggregation across multiple tasks is likely to increase the diversity of sampled situations so that they may better represent those encountered in the real world (Manuck, Kamarck, Kasprowicz, & Waldstein, 1993). Existing data suggest that tasks evoking different patterns of cardiovascular responding ("myocardial" vs. "vascular" tasks) may elicit similar dimensions of individual differences (Kasprowicz, Manuck, Malkoff, & Krantz, 1990). In order to have a stronger test of individual stereotypy of response, we chose tasks based on their ability to elicit alpha-, beta-, and mixed adrenergic patterns of response.

The primary purpose of this study was to test the model suggested by Gregg et al. (2002) in relation to ambulatory blood pressure. Specifically, we hypothesized that patterns of hemodynamic profile and compensation deficit would be superior to blood pressure reactivity measures in the prediction of daily-life blood pressure.

Furthermore, given the recognized role of gender (Sevre et al., 2001) and family history of hypertension (Cook et al., 2001; Lawler et al., 1998) in the incidence of cardiovascular disease, we

evaluated the mediating effect of these factors in the relationship between hemodynamic profile, compensation deficit, and ambulatory blood pressure.

Methods

Participants

Participants were drawn from a large sample of 150 subjects who had been involved in a study investigating interactions between family history of hypertension and psychosocial factors as related to ambulatory blood pressure Full -time employees were recruited by announcements and screened by phone for significant health problems and use of medications that affect cardiovascular functions. Exclusion criteria were: diagnosis of hypertension, health problems, menopause, and pregnancy or childbirth within the last 12 months. Parents of each subject were contacted in order to obtain health information so that subjects could be placed in appropriate family history groups (two hypertensive parents, one hypertensive parent, normotensive parents). For the present study, a letter was sent to all subjects inviting them to participate. Subjects were paid $50.

The participants were 16 men (mean age = 35.4 (8.1)) and 29 women (mean age = 32.6 (7.1)): 16 Asian, 14 Caucasian, 6 African, and 9 Latino Americans. The sample included approximately equal numbers of subjects with and without a positive family history of hypertension (n = 12, one hypertensive parent; n = 10, two hypertensive parents; n = 23, neither). The experimental protocol was approved by the UCLA Institutional Review Board.

Procedure

Participants were informed of the following restrictions: no caffeine, alcohol, nicotine, or strenuous exercise for 2 hours prior to the appointment. The experimental protocol consisted of an initial 10-min baseline period, followed by three 2.5-min tasks and one 2-min task; each task separated by a 10-min resting period. The session ended with a 10-min resting period. The order of tasks was counterbalanced across subjects.

Handgrip Exercise

Subjects were asked to squeeze a hand dynamometer for 2 minutes at 30% of maximal voluntary grip strength level. The handgrip exercise was selected as an example of a commonly used physical task. The 30% threshold was chosen considering that changes in cardiovascular function associated with low-intensity exercise are primarily mediated by parasympathetic withdrawal and, as exercise intensity increases, additional cardiovascular reactivity is mediated by increased sympathetic outflow (Kluess, Wood, & Welsh, 2000).

Mirror Tracing Task

While looking in a mirror, subjects were asked to trace a pattern that was on a board. The pen used for tracing had to remain between the lines of a star-shaped figure without touching the sides of the star. A shield prevented subjects from looking directly at their hand or the pattern. The trial was over when the subject returned to the starting point. Then, a new pattern was placed in the apparatus and a new trial started. Reviews focused on the measurement of cardiovascular reactivity point out that when significant intertask associations do appear, responses to cognitive challenges tend to be more highly correlated with each other than with physical challenges (Kamarck & Lovallo, 2003; Schwartz et al., 2003). Following these assumptions, the mirror tracing task was included in the assessment, as a standardized non-physical stressor, eliciting an alpha-adrenergic response (Kasprovicz, Manuck, Malkoff, & Krantz, 1990).

Computerized Logical-Mathematical Task

Participants were asked if the conclusion to a randomly generated syllogism (e.g., if "a>b" and "b<c" then "a<c?") was true or false. They responded by choosing the related button on the PC. A maximum of 5 s was allowed for the answer. If an answer was entered within the time limit, a window displaying "Correct answer!" or "Incorrect answer!" appeared. If an answer took too long, a window displaying "Too late!" appeared. After completion of each syllogism, a new one appeared immediately on the screen. Among the several stressors used to induce psychological

challenges in the laboratory, the logical-mathematical is an example of a predominantly beta-adrenergic task. A maximum of 5 seconds was allowed for each question as a means of increasing task difficulty and eliciting more substantial cardiovascular activity (Light & Obrist, 1983; Carroll, Turner, & Prasad, 1986). In addition, we gave feedback on subjects' answers because of the role of social evaluation in increasing cardiovascular reactions to laboratory stress (Kelsey et al., 2000; Smith, Nealey, Kircher, & Limon, 1997).

Rumination Task

The task required participants to recall an episode in which they felt intense anger or rage (i.e., being insulted, experiencing abusive or unfair treatment, witnessing others receiving unfair or abusive treatment). Participants were asked to ruminate on causes and consequences of this episode until the experimenter instructed them to "stop." Several studies have shown that social tasks may be more representative of daily life stressors than are the cognitive and physical tasks historically used in assessing cardiovascular reactivity (Linden & Rutledge, 1998). After a stressful event, people may ruminate about causes and consequences of their distress (Nolen-Hoeksema, Larson, & Grayson, 1999; Rusting & Nolen-Hoeksema, 1998), and such responses have been shown to perpetuate cardiovascular activation (Glynn, Christenfeld, & Gerin, 2002; Vitaliano, Russo, Paulsen, & Bailey, 1995). Following these assumptions, a rumination task was chosen as an example of a task with emotional and ecological validity.

Physiological recordings

For each individual in the current study, 24-hr ambulatory and casual blood pressure data were obtained from participation in a prior study. The average time between the two studies was 14.9 (7.6) months. The initial study recorded sitting blood pressure on 3 separate days, 3 readings per session, and the present study recorded 3 casual blood pressure measurements before electrode placement. Mean casual systolic and diastolic blood pressure were respectively 6.5 mmHg (t (1/44)

= 2.67, p = .01) and 5.1 mmHg (t (1/44) = 3.29, p = .002) higher during the second study. Correlations between the two measurements were significant (p = .001) for both casual systolic (r = .51) and diastolic blood pressure (r = .54). Elapsed time and change in blood pressurewere not significantly correlated.

Ambulatory Blood Pressure Monitor

Ambulatory 24-hr recordings of systolic and diastolic blood pressure were obtained during two separate sessions, one on a workday and one on a non-work day. The ambulatory blood pressure was recorded by the Accutracker II (Suntech Medical Instruments, Raleigh, NC), which has been widely used with established reliability and validity in clinical and research studies (Jyothinagaram, Watson, & Padfield, 1990). The Accutracker II was programmed to operate at random intervals three times an hour during waking hours and once an hour during sleep. Concurrent with the ambulatory monitoring, subjects filled out a diary each time the ambulatory recorder operated during waking hours. Ambulatory data were first edited for artifacts based on Accutracker reading codes (insufficient electrocardiogram or Korotkoff sounds) and extreme values (> 200/120 or < 70/40 mm Hg). Editing was done entirely by set rules (Goldstein, Jamner, & Shapiro, 1992). Using a stem-and-leaf plot (Systat, Evanston, IL) we identified and excluded outlying values. Of the total 6438 readings, there were 1229 exclusions (19%), with a mean number of 27.3 (*SD* 8.5) exclusions per subject out of 143.8 (*SD* 13.1). Classification of wake and sleep periods was based on diary entries. Of the total 5501 readings for the wake period, there were 1098 exclusions (19.9%) with a mean number of 24.4 (*SD* 8.0) exclusions per subject out of 122.2 (*SD* 13.6). For the sleep period there were 131 exclusions out of 937 (13.9%) with a mean number of exclusions per subject of 2.9 (*SD* 2.4) out of 20.8 (*SD* 6.3). Number of excluded values did not differ as a function of the major variables of the study.

Laboratory Recordings

Electrocardiographic and impedance cardiographic measures were obtained using two separate recording systems. A Minnesota Impedance Cardiograph measured the impedance signal. Four bands of disposable cardiograph electrode tape (Label Technologies, Inc.) were placed circumferentially around the neck, chest, and abdomen according to published guidelines (Sherwood et al., 1990). Measurements were taken to ensure that the electrodes were at the appropriate distance from one another. Each signal was continuously recorded in 30-s epochs during baseline, each stressor task, and recovery. The Cardiac Output Program (Bio-Impedance Technology, Chapel Hill, NC) acquired, stored, and processed the electrocardiographic (ECG) and impedance cardiographic signals via PC. The program calculated stroke volume for ensemble - averaged waveforms using the Kubicek equation (Kubicek et al., 1966). Each epoch was manually checked to ensure accuracy of the event waveform scoring. The Cardiac Output Program computed cardiac output as the product of stroke volume and heart rate for a given period.

ECG was monitored with a multitrace recorder (AcqKnowledge: Biopac System, Santa Barbara, CA). Disposable Ag-AgCl electrodes (ConMed Corp.) were affixed at standard thoracic monitor sites (right clavicle and precordial site V6).

Beat-to-beat blood pressure was measured non-invasively, using a Finapres Continuous NIBP Monitor (Ohmeda, Madison, WI). Continuous blood pressure readings were obtained via a finger cuff attached to the third finger of the non-dominant hand. The Finapres has been shown to compare well with intra-arterial blood pressure readings (Parati et al., 1989; Pe tersen, Williams & Sutton, 1995). Mean systolic and diastolic blood pressure readings were entered into the Cardiac Output Program to compute total peripheral resistance.

Traditional blood pressure reactivity scores were determined by subtracting the mean level obtained during the first baseline period from the average levels measured during each task.

Assessment of Hemodynamic Profile and Compensation Deficit

In the present study, hemodynamic profile and compensation deficit were assessed following

the new orthogonal, physiologically grounded model proposed by Gregg et al. (2002). The model is derived from the multiplicative relationship between cardiac output and total peripheral resistance in determining mean arterial pressure (Guyton, 1987). In order to obtain an additive function that maintains the integrity of relevant physiological theory concerning hemodynamics, data were treated using ratio scores and logarithmic transformations. The resulting equation used to address the concept of hemodynamic profile was log (cardiac output)r + log (total peripheral resistance)r = log (mean arterial pressure)r, where r indicates a ratio of task to baseline values. The orthogonal relationship between hemodynamic profile and compensation deficit was achieved by a 45-degree rotation of the two-dimensional space formed by the cardiac output and total peripheral resistance reactivity dimensions. Applying the criteria of Gregg et al. (2002), participants are described as more vascular when the algebraic increase in log (total peripheral resistance)r exceeds that in log (cardiac output)r, and more myocardial when the algebraic increase in log (cardiac output)r exceeds that in log (total peripheral resistance)r. Compensation deficit increases as the algebraic sum of the log (cardiac output)r and log (total peripheral resistance)r values increase. Specific details regarding computational method can be obtained by contacting JEJ (email: j.james@nuigalway.ie).

Data Analysis

All data are expressed as means (SD). Differences at $p < .05$ were regarded as significant. Data processing was performed with the software modules of Systat 9.0 (Systat Software Inc., Richmond, California, USA). Gender and family history were treated as categorical variables. Systolic blood pressure reactivity, diastolic blood pressure reactivity, hemodynamic profile, and compensation deficit were treated as continuous variables.

Gender and family history differences were analyzed by t-test. To test for stability of individual differences, intraclass correlations were calculated across tasks for blood pressure reactivity, hemodynamic profile, and compensation deficit. Consistent with the approach adopted by James et al. (2004), one-group *t*-tests were used to test the difference from zero of hemodynamic

profile and compensation deficit scores for each task.

All regression models were computed to predict ambulatory blood pressure (systolic blood pressure and diastolic blood pressure during wake and sleep). As mean work and nonwork day ambulatory blood pressure were similar and highly intercorrelated, models for blood pressure during wake and sleep were run combining data from both days.

The first group of regression analyses aimed at estimating which tasks provided a good test of whether conventional reactivity versus the hemodynamic profile/compensation deficit model was more useful in predicting blood pressure. Blood pressure reactivity, hemodynamic profile, compensation deficit, and hemodynamic profile x compensation deficit served as predictors. The next hierarchical regression models were restricted to the tasks that were useful, following different paths. In each case, we first examined the effects of gender and blood pressure baseline, the obvious frontline predictors. Inasmuch as family history of hypertension was consistently non-significant in all of the preliminary analyses, this variable was not subsequently included as a predictor in the hierarchical regression analysis. As conventional reactivity and ratio reactivity methods are mathematically related, we did not enter conventional reactivity in the models other than the first one.

Model 1: Gender and Baseline Blood Pressure were entered successively. The third predictor was conventional Blood Pressure Reactivity.

Model 2: Gender, Baseline Blood Pressure, then Hemodynamic Profile and Compensation Deficit were successively entered as predictors.

Model 3: Gender and Baseline Blood Pressure were entered, followed by Hemodynamic Profile, Compensation Deficit and the interaction between Hemodynamic Profile and Compensation Deficit.

The analyses aimed at estimating the additional variance that traditional blood pressure reactivity, or both hemodynamic profile and compensation deficit, or the interaction of profile and deficit accounted for in predicting ambulatory blood pressure, when other relevant variables were

controlled for (gender and baseline blood pressure).

Results

Blood Pressure Reactivity

Table 1 shows gender differences in blood pressure baseline and reactivity score values. Men had a 18.5 mmHg higher baseline systolic blood pressure and a 13 mmHg higher baseline diastolic blood pressure than women. Women showed a significantly higher blood pressure reactivity for the handgrip exercise and the mirror tracing task and a higher systolic blood pressure reactivity for the logical-mathematical task than men. Men and women did not differ in diastolic blood pressure reactivity during the logical-mathematical task, and in blood pressure reactivity during the rumination task.

There were no significant differences in blood pressure baseline or blood pressure reactivity values between subjects with a positive or a negative family history of hypertension.

Internal consistency across tasks was high for both systolic (Cronbach's alpha = .78) and diastolic blood pressure reactivity (Cronbach's alpha = .78).

Hemodynamic Profile and Compensation Deficit

Figure 1 shows the relationship between hemodynamic profile and compensation deficit scores for the different tasks. Following James and Gregg (2004), a significant t-test result for hemodynamic profile was taken to indicate either a vascular (positive t-value) or a myocardial profile (negative t-value). A non-significant hemodynamic profile result coupled with a significant compensation deficit result means that the response was mixed (i.e. neither vascular, nor myocardial). No hemodynamic response at all was deemed to have occurred when both hemodynamic profile and compensation deficit were not significant. Following these assumptions, one-group t-tests for hemodynamic profile and compensation deficit indicated that a vascular profile

was induced by the handgrip exercise ($t(44) = 3.62$, $p = .001$ and $t(44) = 10.43$, $p < .0001$, respectively), the mirror tracing task ($t(44) = 5.01$, $p < .0001$) and $t(44) = 4.91$, $p < .0001$), and the rumination task ($t(44) = 2.00$, $p = .05$ and $t(44) = 6.27$, $p < .0001$), while the logical-mathematical task produced a "mixed" profile ($t(44) = 0.42$, $p = .67$ and $t(44) = 4.34$, $p < .0001$).

Pearson intercorrelations between hemodynamic profile and compensation deficit were significant for all tasks ($r = .66$ for the handgrip exercise; $r = .68$ for mirror tracing task; $r = .57$ for the logical-mathematical task; $r = .57$ for the rumination task).

Cronbach's alpha among tasks was .86 for hemodynamic profile and .84 for compensation deficit.

The hemodynamic profile induced by tasks was significantly different for men and women for all tasks ($p < .05$), except for the logical-mathematical task. Specifically, women showed a markedly vascular profile during the handgrip exercise ($p < .0001$), the mirror tracing task ($p < .0001$), and the rumination task ($p = .002$), while the profile during the logical-mathematical task was mixed ($p = .10$). Conversely, men showed a mixed profile during all tasks ($p > .05$).

There were significant gender differences for compensation deficit scores during the handgrip exercise ($p = .001$) and the mirror tracing task ($p = .01$). Specifically, consistent with their higher level of blood pressure reactivity during those tasks, women showed a significantly higher compensation deficit score during the handgrip exercise and the mirror tracing task when compared to men.

T-test comparisons did not show significant differences in the patterns of hemodynamic profiles and compensation deficit for positive and negative history of hypertension.

Hierarchical regression: prediction of Ambulatory Blood Pressure

Multiple regressions were performed for each task. Only the mirror tracing task and the logical-mathematical task supported the role of hemodynamic profile and compensation deficit in the prediction of ambulatory blood pressure, consequently stepwise regression models following the

models outlined above are presented for these two tasks.

With regard to the mirror tracing task, Table 2 shows the results of the hierarchical regression for ambulatory systolic blood pressure during sleep. In all regression models gender was a significant predictor. Specifically, 28% of the variance of systolic blood pressure during sleep could be accounted for by the model including gender, baseline systolic blood pressure, and traditional systolic blood pressure reactivity (Model 1). Hemodynamic profile and Compensation Deficit (Model 2) improved the prediction of systolic blood pressure during sleep changes, accounting for an additional 8% of the variance. Specifically, Hemodynamic Profile was a significant predictor (β = -.37; $F(1, 45) = 4.28$; $p = .04$). The regression model including the interaction between hemodynamic profile and compensation deficit (Model 3) did not significantly add to the prediction of systolic blood pressure during sleep. For ambulatory diastolic blood pressure during sleep (Table 3), the model characterized by gender, baseline diastolic blood pressure and traditional diastolic blood pressure reactivity accounted for 44% of the variance. When other relevant measures were controlled for, hemodynamic profile and compensation deficit (Model 2) accounted for 50% of the variance of diastolic blood pressure sleep and both hemodynamic profile and compensation deficit were significant predictors ((β = -.43; $F(1, 45) = 7.34$; $p = .01$) and (β = .32; $F(1, 45) = 4.15$; $p = .04$), respectively). Scatterplots (Figure 2) show the patterns of hemodynamic profile during the mirror task for ambulatory systolic blood pressure and diastolic blood pressure during sleep.

With regard to the logical-mathematical task, Table 4 and Table 5 show the results of the hierarchical regression for ambulatory systolic blood pressure during wake and sleep, respectively. When other relevant measures were controlled for, the interaction of hemodynamic profile and compensation deficit (Model 3) accounted for an additional 6% of the variance of systolic blood pressure wake (β = .27; $F(1, 45) = 4.29$; $p = .04$), and 10% of the variance of systolic blood pressure sleep (β = .28; $F(1, 45) = 6.85$; $p = .01$) compared to the traditional systolic blood pressure reactivity model. Specifically, the interaction of hemodynamic profile and compensation deficit to

the logical-mathematical task predicted ambulatory systolic blood pressure such as that myocardial reactors exhibited increasing systolic blood pressure as compensation deficit increased, whereas an increase in compensation deficit was not coupled by a remarkable change in systolic blood pressure for vascular reactors (see Figure 3).

No other hierarchical multiple regressions models were significant.

Discussion

Cardiovascular reactivity is theoretically considered a unitary construct although in practice it is measured by parameters that are physiologically linked, but typically analyzed as independent factors. When different statistical models were examined, the two-factor classification of individuals in terms of the relationship between cardiac output and total peripheral resistance appeared to be the best method of reducing the variability of each single occasion of measurement and emphasizing the presence of individual differences (Kamarck & Lovallo, 2003). However, the two-factor solution in the context of blood pressure responses to laboratory stress is not without problems. Earlier classifications of individuals as myocardial or vascular reactors used linear additive models, ignoring the physiological, multiplicative relationship between cardiac output and total peripheral resistance. Present findings confirmed the usefulness of the hemodynamic profile/compensation deficit model in the prediction of ambulatory blood pressure.

The reactivity hypothesis is founded on the assumption that cardiovascular stress response is a relatively stable trait (Manuck, 1994). Several studies showed that one's tendency to react with cardiac or vascular responding persists across different types of tasks (Kasprowicz, Manuck, Malkoff, & Krantz, 1990; Sherwood et al., 1990). Consistent with this finding, subjects from our sample maintained their characteristic response tendencies, even though tasks were selected to elicit different cardiovascular response patterns. Traditional blood pressure reactivity values were intercorrelated across laboratory tasks, but failed in predicting everyday-life blood pressure. The lack of relationship between traditional blood pressure reactivity and ambulatory data is in

accordance with several previous studies (e.g. Seibt, Boucsein, & Scheuch, 1998).

Compared to traditional blood pressure reactivity, the use of hemodynamic response patterns improved our ability to predict ambulatory blood pressure, even after controlling for the frontline predictors: gender and baseline. This could be partially ascribed to the increase in reliability, both across time and tasks, associated with impedance-derived reactivity measures when compared to blood pressure reactivity measures (Kasprowicz, Manuck, Malkoff, & Krantz, 1990; McGrath & O'Brien, 2001; Saab et al., 1992). In particular, Matthews, Salomon, Brady, and Allen (2003) demonstrated the effectiveness of impedance hemodynamic measures in predicting future rises in blood pressure. Furthermore, in a prospective 10-year follow-up study, Jokiniitty et al. (2002) demonstrated that the predictive value of casual blood pressure measurements for future blood pressure level could be improved by using hemodynamic responses to different tasks.

Our first aim was to answer to the following question: which tasks provided a good test of whether the conventional reactivity model or the hemodynamic profile/compensation deficit model was more useful in predicting blood pressure? Indeed, one of the occasional problems in literature is to find tasks that are sufficiently potent elicitors of reactivity (Lovallo & Gerin, 2003). In general, the results indicate that over and above gender and baseline a) the interaction between hemodynamic profile and compensation deficit during the logic task predicted ambulatory systolic blood pressure across sleep and wake; b) hemodynamic profile during the mirror tracing task predicted ambulatory systolic blood pressure and diastolic blood pressure during sleep; and c) compensation deficit during the mirror tracing task predicted diastolic blood pressure during sleep.

The usefulness of the logical-mathematical stressor confirmed the frequent use of beta-adrenergic tasks as the most suitable for hemodynamic classifications (Girdler et al., 1990; Kasprowicz, Manuck, Malkoff, & Krantz, 1990; Sherwood & Turner, 1993). Specifically, the interaction of hemodynamic profile and compensation deficit in response to the logical-mathematical task predicted ambulatory systolic blood pressure such that myocardial reactors exhibited increasing systolic blood pressure as compensation deficit increased, whereas there was

little change in systolic blood pressure for vascular reactors. Interestingly, hemodynamic profile during the mirror task was a strong predictor for nighttime ambulatory blood pressure A possible explanation considers hemodynamic circadian patterns. At nighttime, blood flow to the skeletal muscles is decreased through local autoregulation, which increases total peripheral resistance and decreases cardiac output, while a steep increase in cardiac output and a decrease in total peripheral resistance characterizes the morning period (Veerman et al., 1995). Thus, a predominantly alpha-adrenergic task could be a better predictor of sleeping blood pressure, characterized by a total peripheral resistance increase. Another explanation is related to the nature of demands in everyday life. Beta-adrenergic activity increases as cognitive functioning become more complex (Streufert et al., 1988), so that we can speculate that the mirror tracing task did not predict waking blood pressure because demands are less vascular in nature during waking hours. In line with Majahalme et al. (1998), the isometric exercise (handgrip) did not prove to be a very useful predictor of ambulatory blood pressure level. Thus, the present data support the use of responses to cognitive rather than physical laboratory tasks as a potential marker of risk for hypertension (Kamarck & Lovallo, 2003; Schwartz et al., 2003). Surprisingly, the rumination task proved to be the worst predictor. It is well known that social challenges in the lab pose special difficulties because they are more complex than cognitive tasks and involve many dimensions. Verbal reports obtained immediately after the task emphasized individual differences in terms of the ability to identify with the situation described in the task. In addition, we found important differences between subjects in terms of elapsed time since the anger-related episode occurred. With all the limitations previously described, the present study does not support the usefulness of this specific emotional task in the prediction of daily life blood pressure levels.

The present findings may provide insights into the role of hemodynamic profiles in the pathogenesis of essential hypertension. Julius et al. (1983) posited an initial state of myocardial activation, progressing to a later state of increased total peripheral resistance. Consequently, borderline hypertension is characterized by increased stroke volume and cardiac output and normal

total peripheral resistance, whereas sustained hypertension is characterized by normal cardiac output and increased total peripheral resistance. The competing theory is that the initial phase is characterized by increased total peripheral resistance, associated with trophic factors in the blood vessel wall. This hypothesis is consistent with Sung et al. (1993) who found elevated vascular resistance in borderline hypertensives. The higher ambulatory blood pressure found in this sample for participants who show a more myocardial profile suggests that the early rise of blood pressure may be indeed characterized by myocardial rather than resistance responsivity to stress. However, Figure 3 suggests that a more myocardial profile might have a pathogenetic role for the development of hypertension only when associated with a higher deficit in compensating. These findings demonstrated that the model proposed by Gregg et al. (2002) offers improved predictive potential for future studies on cardiovascular reactivity and pathogenesis. It remains for future studies to examine the health implications of these hemodynamic patterns by relating them to the development of specific cardiovascular diseases.

The present study emphasized the superiority of Gregg et al. (2002) model compared to traditional reactivity in the prediction of daily-life blood pressure, but showed a discrepancy with the model. In fact, we found a significant correlation between hemodynamic profile and compensation deficit, a finding that is contrary to the model, which is predicated on hemodynamic profile and compensation deficit being orthogonal. Gregg et al. (2002) anticipated the possibility that the model needed adjusting and the present study supports the need of further research to elucidate the relationship between hemodynamic profile and compensation deficit. Gregg et al. observed substantial though not complete orthogonality, and suggested that the non-orthogonality may have been due to the "accumulation of measurement errors" across several parameters. A possible source of non-orthogonality is related to potential measurement errors, especially when different measurement approaches are used (Gregg and colleagues used the Finapres to measure cardiac output and total peripheral resistance, whereas impedance cardiography was used in the present study). At the same time, Gregg et al. noted that if non-orthogonality continues to be

observed in further research this could indicate grounds for revising the generally accepted theoretical equation Mean Arterial Pressure = Cardiac Output x Total Peripheral Resistance. It is only after independent sources of empirical data have been collected that we will be in a position to say whether or not the theory regarding hemodynamics needs to be revisited.

The present study is not without limitations. First, the sample size was relatively small and may not have been adequate in some of the comparisons. Second, with regards to the failure to show that family history was a significant mediator to the effects, we used two groups to differentiate between positive and negative parental hypertension history, while Manuck et al. (1996), recommended three groups. Because we were unable to discern differences between our two small subgroups (individuals with two hypertensive parents (n = 10) and those with one hypertensive parent (n = 12)), they were grouped together under positive family history of hypertension. Third, there is a strong prevalence of women in the sample (p = .05). In contrast to Girdler, Turner, Sherwood, and Light (1990) but in agreement with Kline et al. (2002), women were more likely to be vascular responders. As previously reported, women demonstrated a lower baseline and ambulatory blood pressure (Knox, Hausdorff, & Markovitz, 2002), but higher blood pressure reactivity (Alfie et al., 1995). Fourth, none of the tasks used in this study seemed to elicit a very strong cardiac response. This is an unfortunate limitation, given that activation of beta-adrenergic processes has been linked to the maximal laboratory blood pressure increase (Harrison et al., 2001) and ambulatory blood pressure prediction (Markovitz et al., 1998).

Limitations notwithstanding, the study showed that the prediction of ambulatory blood pressure could be improved by applying hemodynamic profile and compensation deficit data even after controlling for gender and baseline, and was better than using traditional blood pressure reactivity as a predictor. Obviously, replication with a larger sample size and a wider range of tasks is needed to test the robustness of the present findings.

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NOTES

This research was supported by Research Grant HL-52102, National Heart Lung and Blood Institute.

Tables

Table 1. *Gender Differences in Baseline and Reactivity for Systolic and Diastolic Blood Pressure*

Blood Pressure	Men (n = 16)	Women (n = 29)	p
Baseline			
Systolic (mmHg)	133.4 (16.6)	114.9 (15.2)	.001
Diastolic (mmHg)	76.3 (11.0)	63.3 (10.5)	.0001
Reactivity			
Handgrip			
Systolic (mmHg)	10.9 (11.1)	27.4 (17.3)	.0001
Diastolic (mmHg)	6.7 (5.6)	14.8 (9.3)	.003
Mirror Tracing			
Systolic (mmHg)	- 0.3 (16.8)	13.7 (29.8)	.02
Diastolic (mmHg)	1.4 (7.4)	7.9 (10.9)	.03
Logical-Mathematical			
Systolic (mmHg)	4.8 (15.2)	15.4 (17.6)	.05
Diastolic (mmHg)	- 0.6 (12.5)	5.6 (10.6)	.08
Rumination			
Systolic (mmHg)	5.9 (13.0)	13.9 (19.3)	.14
Diastolic (mmHg)	4.4 (9.5)	4.9 (10.0)	.87

Note. Values are mean (standard deviation). Each reactivity score is the mean (SD) response level during the task minus baseline.

Table 2. *Summary of Hierarchical Regression Analysis for the Prediction of Ambulatory Systolic Blood Pressure During Sleep. Hemodynamic Profile, Compensation Deficit, and Traditional Systolic Blood Pressure Reactivity relates to the Mirror Tracing Task (N = 45)*

Model 1					**Model 2**					**Model 3**				
	B	*SE*	*β*	*VIF*		*B*	*SE*	*B*	*VIF*		*B*	*SE*	*β*	*VIF*
Gender	-8.09	2.68	-.46**	1.35	Gender	-7.05	2.61	-.40*	1.39	Gender	7.08	2.65	-.40*	1.40
Baseline	0.05	0.16	.06	1.59	Baseline	-0.02	0.15	-.01	1.61	Baseline	0.00	0.16	-.00	1.66
Reactivity	-0.08	0.13	-.09	1.35	Profile	-74.86	36.17	-.37*	2.05	Profile	-71.74	39.52	-.36	2.39
					Deficit	28.76	66.47	-.08	1.98	Deficit	22.33	73.93	.06	2.39
										Profile x Deficit	146.08	697.49	.03	1.33
R^2			.28		R^2			.36		R^2			.36	

Note: Hemodynamic Profile and Compensation Deficit were centered at their means before calculating the Interaction term.

*$p < .05$, **$p < .01$.

Table 3. *Summary of Hierarchical Regression Analysis for the Prediction of Ambulatory Diastolic Blood Pressure During Sleep. Hemodynamic Profile, Compensation Deficit, and Traditional Systolic Blood Pressure Reactivity relates to the Mirror Tracing Task (N = 45)*

Model 1					**Model 2**					**Model 3**				
	B	*SE*	*β*	*VIF*		*B*	*SE*	*B*	*VIF*		*B*	*SE*	*β*	*VIF*
Gender	-2.67	0.75	-.48*	1.37	Gender	-2.21	0.73	-.40**	1.45	Gender	-2.12	0.74	-.38**	1.45
Baseline	0.15	0.06	.35	1.61	Baseline	0.11	0.06	.25	1.62	Baseline	0.11	0.06	.25	1.62
Reactivity	0.11	0.07	.31	1.31	Profile	-27.02	9.98	-.43*	2.01	Profile	-30.74	10.67	-.49**	2.31
					Deficit	38.09	18.69	.32*	2.02	Deficit	46.85	20.71	.40*	2.48
										Profile x Deficit	-186.13	188.94	-.12	1.29
R^2			.44		R^2			.50		R^2			.51	

Note: Hemodynamic Profile and Compensation Deficit were centered at their means before calculating the interaction term.

*$p < .05$, **$p < .01$.

Table 4. *Summary of Hierarchical Regression for the Prediction of Ambulatory Systolic Blood Pressure During Wake. Hemodynamic Profile, Compensation Deficit, and Traditional Systolic Blood Pressure Reactivity relates to the Logical-Mathematical Task (N = 45)*

Model 1					**Model 2**					**Model 3**				
	B	*SE*	*β*	*VIF*		*B*	*SE*	*B*	*VIF*		*B*	*SE*	*β*	*VIF*
Gender	-5.27	1.72	-.45**	1.33	Gender	-5.18	1.76	-.44**	1.37	Gender	-5.31	1.71	-.45**	1.37
Baseline	0.14	0.11	.22	2.04	Baseline	0.09	0.11	.14	1.85	Baseline	0.12	0.11	.19	1.88
Reactivity	0.03	0.11	.05	1.69	Profile	-9.75	21.56	-.07	1.54	Profile	1.38	21.64	.01	1.65
					Deficit	-6.46	43.41	-.03	1.92	Deficit	8.55	42.69	.03	1.97
										Profile x Deficit	505.83	261.14	.27*	1.21
R^2			.33		R^2			.33		R^2			.39	

Note: Hemodynamic Profile and Compensation Deficit were centered at their means before calculating the interaction term.

*$p < .05$, **$p < .01$.

Table 5. *Summary of Hierarchical Regression Analysis for the Prediction of Ambulatory Systolic Blood Pressure During Sleep. Hemodynamic Profile, Compensation Deficit, and Traditional Systolic Blood Pressure Reactivity relates to the Logical-Mathematical Task (N = 45)*

Model 1					**Model 2**					**Model 3**				
	B	*SE*	*β*	*VIF*		*B*	*SE*	*B*	*VIF*		*B*	*SE*	*β*	*VIF*
Gender	-8.40	2.64	-.48**	1.33	Gender	-8.21	2.65	-.47**	1.37	Gender	-8.39	2.56	-.48**	1.37
Baseline	-0.03	0.18	-.03	2.05	Baseline	-0.05	0.16	-.06	1.85	Baseline	-0.01	0.16	-.02	1.89
Reactivity	-0.19	0.16	-.21	1.69	Profile	-20.63	32.41	-.10	1.54	Profile	-3.25	32.39	-.02	1.65
					Deficit	-79.29	65.21	-.22	1.92	Deficit	-55.86	63.90	-.15	1.97
										Profile x Deficit	789.61	390.84	.28*	1.21
R^2		30			R^2		34			R^2		40		

Note: Hemodynamic Profile and Compensation Deficit were centered at their means before calculating the interaction term.

*$p < .05$, **$p < .01$.

Figure Captions

Figure 1. Scatterplots for Hemodynamic Profile and Compensation Deficit During Each Task.

Note. A "more vascular" profile is associated with more positive values along the Hemodynamic Profile axis and a "more myocardial" profile is associated with more negative values along the Hemodynamic Profile axis. A "higher deficit" in compensating is associated with more positive values on the Compensation Deficit axis and a "lower deficit" in compensating is associated with more negative values on the Compensation Deficit axis**.**

Figure 2. Scatterplots for Ambulatory Systolic Blood Pressure and Diastolic Blood Pressure during Sleep and Hemodynamic Profile during the Mirror Tracing Task.

Figure 3. Interaction between Hemodynamic Profile and Compensation Deficit in relation to Systolic Blood Pressure during Wake.

Note. Endpoints of the lines in the graph represent one standard deviation above and below the mean for Hemodynamic Profile and Compensation Deficit.

Figure 1.

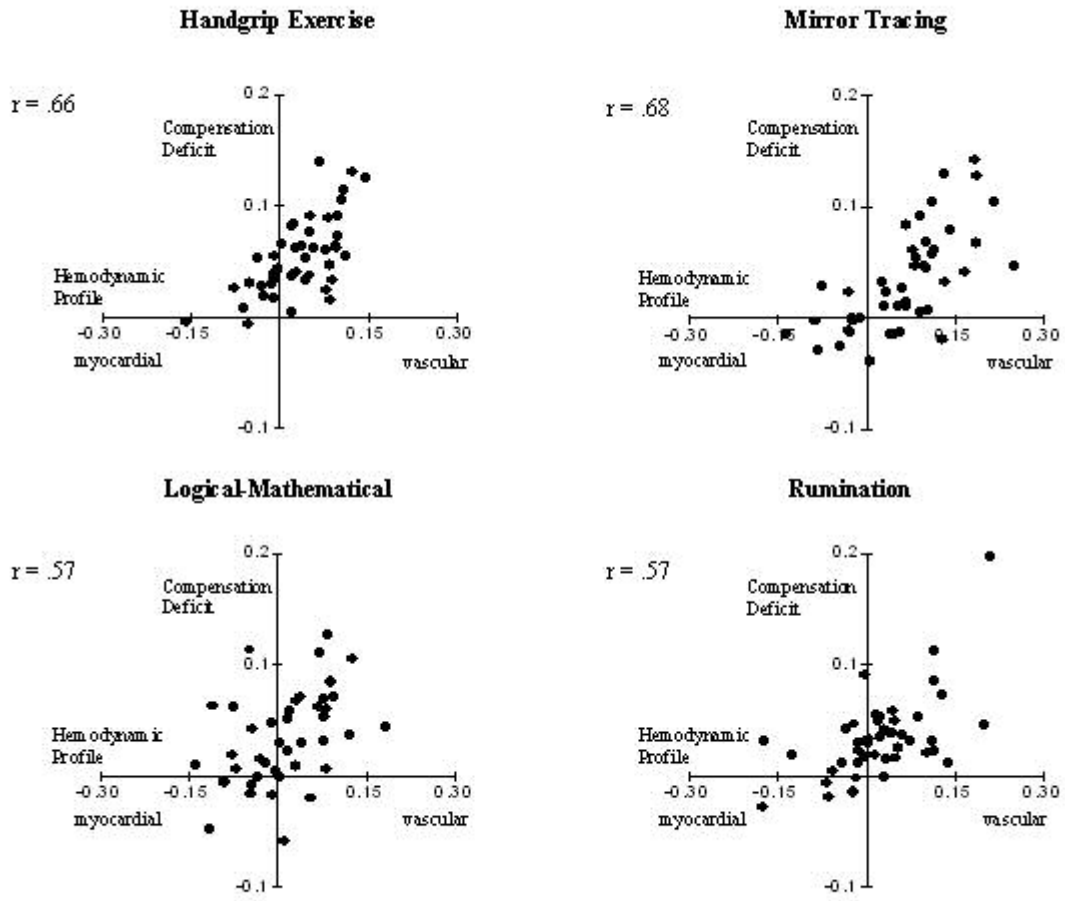

Handgrip Exercise
r = .66
Compensation Deficit
Hemodynamic Profile
myocardial
vascular
0.2
0.1
-0.1
-0.30
-0.15
0.15
0.30
Mirror Tracing
r = .68
Logical-Mathematical
r = .57
Rumination
r = .57

Figure 2.

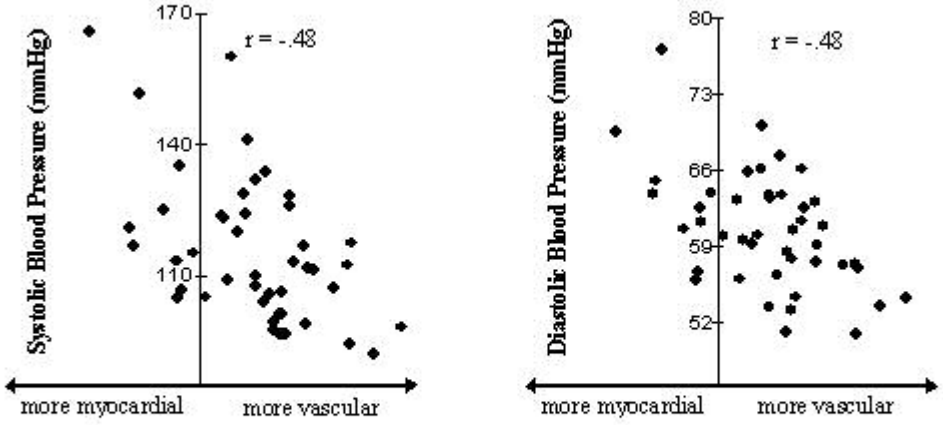
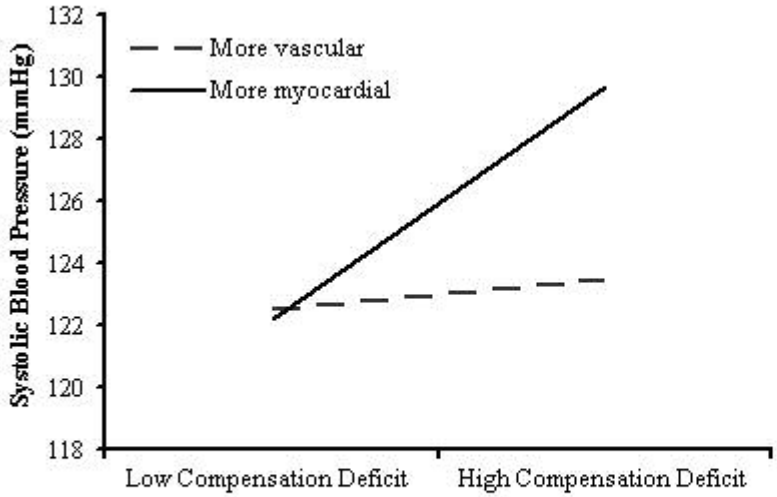

Systolic Blood Pressure (mmHg)
170
140
110
r = -.48
more myocardial
more vascular
Diastolic Blood Pressure (mmHg)
80
73
66
59
52
r = -.48
more myocardial
more vascular

Figure 3.


132
130
128
126
124
122
120
118
Systolic Blood Pressure (mmHg)
More vascular
More myocardial
Low Compensation Deficit
High Compensation Deficit