Impaired cardiovascular recovery following stress predicts 3-year increases in blood pressure

Andrew Steptoe and Michael Marmot

Objective **To assess whether variation in the rate of cardiovascular recovery following exposure to acute psychological stress predicts changes in blood pressure longitudinally, independently of blood pressure at baseline and other covariates.**

Design **A 3-year longitudinal study.**

Participants **A total of 209 men and women aged 45–59 years at baseline, with no history of cardiovascular disease including hypertension.**

Method **Measurement of blood pressure, heart rate, heart rate variability, cardiac index and total peripheral resistance at rest, during two moderately stressful behavioural tasks and up to 45 min post-stress. Stress reactivity was defined as the difference in values between tasks and baseline, and post-stress recovery as the difference between recovery levels and baseline.**

Outcome measures **Resting blood pressure measured at baseline and 3 years later. Seven individuals had been prescribed hypertensive medication on follow-up.**

Results **Increases in systolic blood pressure (SBP) were predicted by impaired post-stress recovery of SBP ($P < 0.001$), diastolic blood pressure (DBP) ($P < 0.001$) and total peripheral resistance ($P = 0.003$), independently of baseline blood pressure, age, gender, socio-economic status, hypertensive medication, body mass and smoking. The adjusted odds of an increase in SBP ≥ 5 mmHg were 3.50 [95% confidence interval (CI) 1.19 to 10.8] for individuals with poor compared with effective post-stress recovery of SBP. Three-year increases in diastolic pressure were predicted by impaired recovery of SBP ($P < 0.001$) and DBP ($P = 0.009$) pressure and by heart rate variability during tasks ($P = 0.002$), independently of covariates.**

Conclusions **Impaired post-stress recovery and less consistently heightened acute stress reactivity may index disturbances in the regulation of cardiovascular stress responses that contribute to longitudinal changes in blood pressure in middle-aged men and women.** ***J Hypertens*** **23:529–536 © 2005 Lippincott Williams & Wilkins.**

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Introduction

The assessment of acute cardiovascular responses to mental stress tests has been used extensively for the investigation of psychosocial factors in the development of hypertension and coronary heart disease (CHD). A large number of studies have measured cardiovascular stress responses in hypertensives and normotensives, people varying in parental history of hypertension and in relation to factors such as social support, hostility and background chronic stress [1–3]. One concern about the value of this approach is whether stress responsivity is significant clinically and predicts changes in blood pressure and or future disease states longitudinally. A recent review of more than 20 prospective studies indicated that individual differences in blood pressure (BP) or heart rate stress responsivity predict future BP level and hypertension independently of baseline BP and other factors [4]. Nonetheless, there are a number of inconsistencies in this literature, and the additional variance accounted for by cardiovascular stress responses is often small [5,6].

Although the magnitude of cardiovascular responses during stress exposure is usually measured, rate of recovery during the post-stress period is also important. Recent stress research has highlighted impaired recovery as a key marker of psychobiological dysfunction [7]. Impaired or delayed cardiovascular recovery has been associated cross-sectionally with a family history of hypertension in young adults, low physical fitness and with high levels of background stress [3,8,9]. Our group has previously shown that post-stress recovery of BP is impaired in lower socio-economic status individuals, and that this effect is sustained by heightened total peripheral resistance

[10,11]. In the present study, measurements of clinic BP were carried out in healthy middle-aged men and women who had undergone mental stress testing 3 years earlier. The influence of cardiovascular responses to tasks and post-stress recovery on longitudinal changes in systolic blood pressure (SBP) and diastolic blood pressure (DBP) was tested, after controlling for initial BP, age, socio-economic status, smoking and body mass index (BMI).

Methods

Participants

These data were collected as a follow-up to the psychobiology sub-study of the Whitehall II study. The Whitehall II cohort is a sample of 10 308 London-based civil servants recruited in 1985–1988 when aged 35–55 years, to investigate demographic, psychosocial and biological risk factors for coronary heart disease [12]. The psychobiology sub-study involved 228 volunteers (123 men, 105 women) who underwent detailed laboratory investigation [10,11]. Participation was based on the following criteria: white European origin, aged 45–59 years, living in the London area, not planning to retire for at least 3 years, no history or objective signs of coronary heart disease and no previous diagnosis or treatment for hypertension. The 3-year follow-up data were collected during a screening session from 209 individuals (111 men and 98 women, a 92% response rate). Of the remaining 19, one had died; four were lost to follow-up; two did not attend screening despite repeated requests; three had withdrawn from the Whitehall II study and nine had moved out of London, so were not invited to the screening session.

Laboratory mental stress session

Blood pressure and heart rate were monitored continuously from the finger, using a Portapres-2 (Finapres Medical Systems, Amsterdam, The Netherlands), a portable version of the Finapres device that shows good reproducibility and accuracy in a range of settings [13]. Absolute BP levels obtained with the Finapres were corrected against simultaneous brachial recordings. Cardiac output and stroke volume were determined from the Portapres using the aortic flow waveform method described by Wesseling *et al.* [14] and embodied in Modelflow 2.1 software (FMS, Amsterdam, The Netherlands). Stroke volume was calculated from the systolic area, i.e. the area under the arterial pressure wave between the onset of the blood pressure rise and the dichrotic notch, on a beat by beat basis corrected by a calibration factor related to aortic compliance. Total peripheral resistance (TPR) was predicted from mean pressure and computed aortic flow. Good agreement has been obtained between Modelflow computations of stroke volume and cardiac output from intra-arterial and finger blood pressure measures and between finger-based measures and thermodilution [15,16]. Heart rate variability was assessed as the root mean square of successive differences in R-R intervals (RMSSD) obtained from a three-lead electrocardiogram in 143 participants using an ambulatory cardiac impedance device (VU-AMS, Free University, Amsterdam, The Netherlands) [17].

Mental stress was induced by two behavioural tasks: a computerized colour-word interference task and mirror tracing. Both tasks have been extensively used in cardiovascular stress research [18–20]. The colour-word task involved the presentation of a succession of target colour words in incongruous colours. At the bottom of the computer screen were four names of colours displayed in incorrect colours and the task was to press a computer key that corresponded to the position at the bottom of the screen of the name of the colour in which the target word was printed. Mirror tracing involved the tracing of a star with a metal stylus, which could only be seen in mirror image. Participants were told that the average person completed five circuits of the star in the time available and were asked to give accuracy priority over speed on both tasks.

Participants were tested in either the morning or afternoon in a light and temperature-controlled laboratory. They were instructed not to have drunk tea, coffee, or caffeinated beverages, or to have smoked for at least 2 h prior to the study and not to have consumed alcohol or exercised on the evening before, or the day of testing. Following instrumentation and the insertion of a venous cannula for the periodic collection of blood samples (not described here), the participant rested for 30 min. BP and heart rate were recorded for the last 5 min and heart rate variability for the last 10 min of this period (baseline trial). Two measures using a standard arm cuff (A&D UA779) were also obtained and these values were averaged to index time 1 (T1) clinic SBP and DBP. The two tasks were then administered in random order with a 5 min inter-task interval. Each lasted for 5 min, during which BP and heart rate were recorded continuously. Post-stress recovery was assessed with 5 min recordings of BP and heart rate made 15–20 and 40–45 min after tasks. Participants rested quietly during the recovery period. The study was approved by the UCL/UCLH Committee on the Ethics of Human Research.

Three-year assessment

The 3-year follow-up BP data were collected as part of a screening session for the full Whitehall II cohort. The interval between the stress session (T1) and follow-up (T2) averaged 3 years, 21 days $\pm$ 110 days. Two BP readings were obtained by a research nurse with the participant seated. The correlations between the two readings were 0.89 for SBP and 0.95 for DBP, so measures were averaged. Body weight and height were measured and BMI calculated. Smoking and alcohol consumption were assessed by questionnaire. Seven individuals (four men

and three women) had been prescribed hypertensive medication at T2.

Statistical analysis

The SBP, DBP, heart rate, cardiac output, TPR and heart rate variability during the stress session were averaged into five trials: baseline, the two tasks and recovery periods 1 (15–20 min post-stress) and 2 (40–45 min post-stress). Cardiovascular responses to the two tasks were highly correlated, with increases in SBP and DBP responses to the tasks correlating 0.86 and 0.87, respectively across participants. Consequently, we averaged task measures to produce a mean task value for each variable. Cardiac output was transformed into cardiac index by correcting for body surface area.

Responses during the stress session were analysed using repeated measures analysis of variance with gender as the between-subject factor and trial (baseline, tasks, recovery 1, recovery 2) as the within-subject factor. The Greenhouse–Geisser correction was applied where appropriate. Stress reactivity was defined as the difference between the average task and baseline values and recovery, as the difference between recovery trial 2 and baseline.

The two BP readings obtained by standard sphygmomanometry after 30-min rest in the laboratory constituted T1 BP. The two readings obtained during the screening session 3 years later were the T2 follow-up data, and change scores were computed by subtracting T1 from T2 values. The predictors of change in BP over the 3-year period were analysed using multiple regression on T2 values controlling for T1 level. The other control variables in the regression analyses were factors that might be associated with increases in BP over time independently of stress responsivity. They included age, gender, grade of employment, hypertensive medication (entered as a binary dummy variable), BMI and smoking status at T2. The influence of stress reactivity was assessed by entering task values into the regression and the impact of recovery, by entering recovery trial 2 values. This procedure is statistically equivalent to entering reactivity and recovery change scores. The independent effects of reactivity and recovery are presented as unstandardized regression coefficients (B) with 95% confidence intervals (CI). The inclusion of alcohol consumption into the regression models did not alter the associations with cardiovascular stress response parameters, so alcohol was not added as a covariate.

Predictive effects were further analysed by calculating the proportion of participants who showed a definite increase in SBP or DBP. Based on the distribution of changes between T1 and T2, the sample was divided into those who showed an increase ≥ 5 mmHg SBP and ≥ 3.5 mmHg DBP, though other cutpoints produced similar results. These data were analysed by multiple logistic regression and the odds (with 95% CI) of showing an increase in BP above criterion for a standard difference in the levels of predictor variables were calculated, as detailed in the Results section. Data are presented as means $\pm$ standard deviations.

Results

Details of study participants are summarized in Table 1. Men were slightly older than women on average ($P = 0.045$), but there were no differences in socioeconomic status defined by grade of employment. The proportions of male and female smokers did not differ, and there was no difference in BMI or alcohol consumption. Average levels of SBP and DBP were low in both men and women at the two time points. There was a small decrease in SBP and DBP between T1 and T2 in men, which was significant in the case of DBP only ($P < 0.001$). Women showed an increase in SBP ($P = 0.013$) and a decrease in DBP ($P = 0.028$). Importantly, there was wide variation in the changes in SBP and DBP over the 3-year period, as evidenced by the large standard deviation for difference scores (12.0 mmHg for SBP and 8.10 mmHg for DBP).

Cardiovascular stress responses

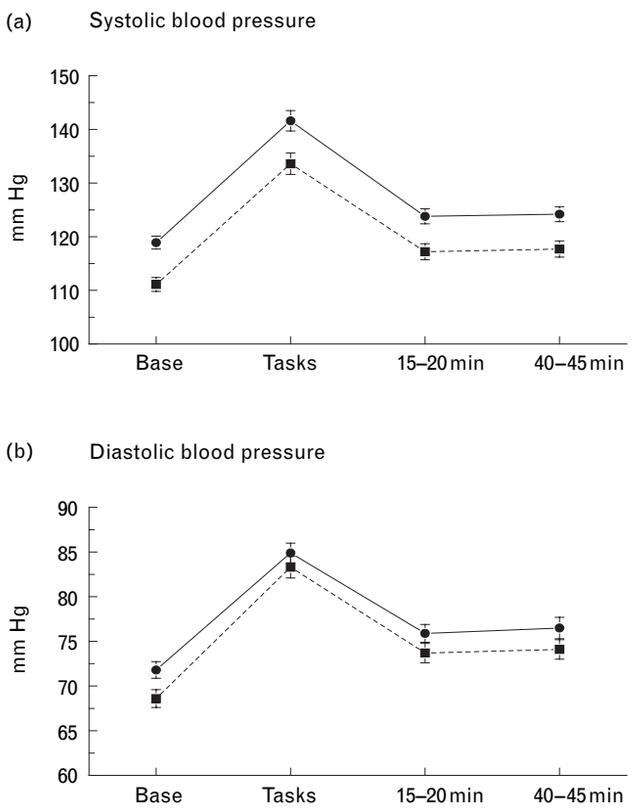
SBP and DBP rose during stress, with increases averaging 22.8 and 13.9 mmHg, respectively ($P < 0.001$). Blood pressure fell between tasks and post-task recovery trials ($P < 0.001$), but nevertheless remained above baseline levels for recovery trials 1 (15–20 min) and 2 (40–45 min, both $P < 0.001$). Interestingly, neither SBP nor DBP decreased between the two post-task recovery trials (Fig. 1). There was no interaction between gender and trial in these analyses. Some 36.5% of participants showed an increase in SBP ≥ 5 mmHg over the 3-year follow-up period, while 18.7% increased DBP ≥ 3.5 mmHg.

Heart rate was higher on average in women than men ($P = 0.02$, Fig. 2). In addition, there was a significant gender by trial interaction ($P = 0.004$), since heart rate responses to tasks were larger in women than men [means 8.17 versus 6.07 beats/min (bpm), $P = 0.01$]. Unlike the pattern for BP, heart rate was below baseline levels

Table 1 **Study participants**

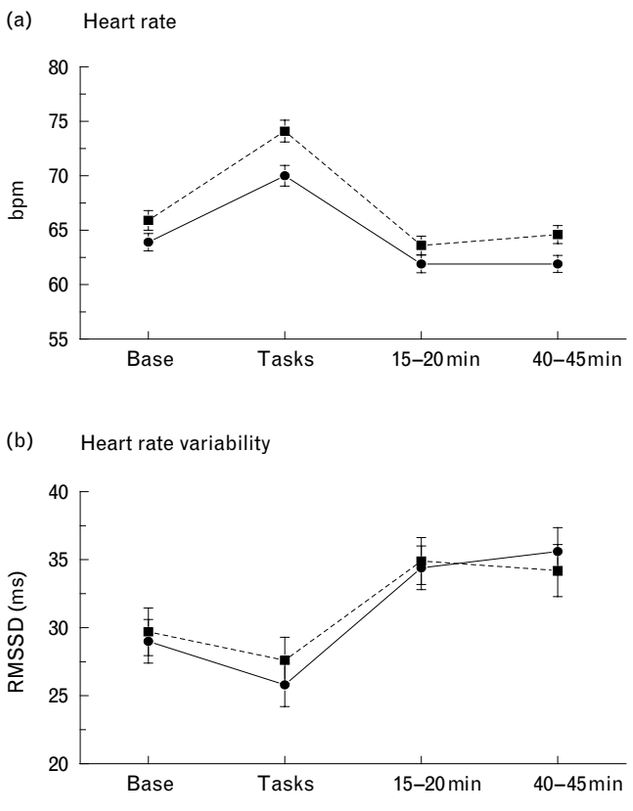
	Men ($n = 111$)	Women ($n = 98$)
Age T1 (years)	52.6 ± 2.6	51.8 ± 2.8
Grade of employment		
Higher	44 (39.6%)	35 (35.7%)
Intermediate	40 (36.0%)	33 (33.7%)
Lower	27 (24.3%)	30 (30.6%)
Smoking T2 (%)	14 (12.6%)	6 (6.1%)
Body mass index T2 (kg/m^2)	25.9 ± 3.8	25.4 ± 4.4
Alcohol consumption at least daily T2 (%)	48 (41.4%)	39 (38.2%)
Systolic blood pressure T1 (mmHg)	121.3 ± 12.4	113.4 ± 14.2
Systolic blood pressure T2 (mmHg)	119.5 ± 11.9	116.7 ± 15.0
Diastolic blood pressure T1 (mmHg)	74.5 ± 8.9	71.5 ± 9.9
Diastolic blood pressure T2 (mmHg)	70.4 ± 10.3	69.7 ± 10.5

Fig. 1



Mean (a) systolic blood pressure and (b) diastolic blood pressure, during the baseline, task and post-stress recovery trials. Values for men are shown in solid lines and women in dashed lines. Error bars are standard error of the mean (SEM).

Fig. 2



Mean (a) heart rate and (b) heart rate variability, during the baseline, task and post-stress recovery trials. Values for men are shown in solid lines and women in dashed lines. Error bars are standard error of the mean (SEM). bpm, beats per minute; RMSSD, root mean square of successive differences in milliseconds (ms).

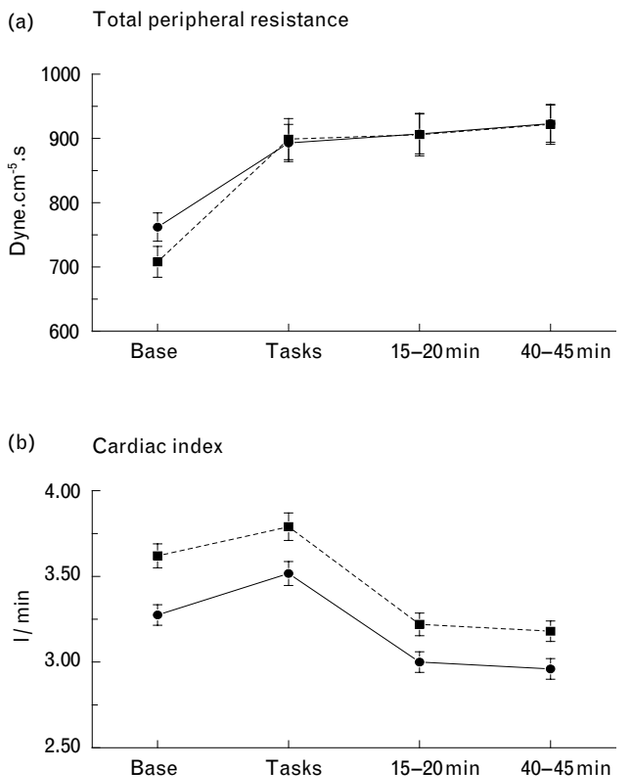
during post-task recovery trials 1 and 2 (both $P < 0.001$, Fig. 2). The increase in heart rate during trials was associated with a significant reduction in heart rate variability ($P < 0.001$), followed by an increase above baseline levels during the recovery period ($P < 0.001$). There was no interaction between gender and trial in the analysis of heart rate variability.

Total peripheral resistance did not differ significantly at baseline in men and women ($P = 0.86$), but resting cardiac index was higher in women ($P = 0.005$). There was a significant rise in both TPR and cardiac index during tasks ($P < 0.001$, Fig. 3). The gender by trial interaction was significant for TPR ($P = 0.043$), but not cardiac index. This was due to the increase in TPR during tasks being greater in women than men ($P = 0.04$). Total peripheral resistance remained elevated above baseline throughout the post-task recovery period ($P < 0.001$), but cardiac index fell below baseline during the two recovery trials ($P < 0.001$). Thus the elevation in SBP and DBP during the recovery period was sustained by higher vascular resistance, with low heart rate, cardiac index and enhanced parasympathetic cardiac control.

Prediction of SBP change

SBP at T2 was positively associated with T1 resting SBP ($P < 0.001$) and with BMI at T2 ($P = 0.034$), and was lower in smokers than non-smokers ($P = 0.006$). It was not related independently to gender, grade of employment, or to SBP responses to behavioural tasks. However, the change in SBP between T1 and T2 was predicted by SBP during recovery trial 2, independently of T1 resting BP, age, gender, grade of employment, hypertensive medication, BMI and smoking (B = 0.33, CI 0.16–0.49, $P < 0.001$). Participants whose BP failed to return to baseline levels during post-stress recovery showed greater SBP increases over the 3-year follow-up period. This effect was explored further by analysing the factors predicting an increase in SBP of ≥ 5 mmHg between T1 and T2, dividing SBP during recovery trial 2 into tertiles. The proportion of individuals with a SBP increase ≥ 5 mmHg ranged from 26.1% in the lowest recovery trial tertile to 50.1% in those in the highest tertile (Table 2). Compared with the lowest tertile, the odds of an increase ≥ 5 mmHg were 3.59 (CI 1.19–10.8, $P = 0.024$) for the highest tertile of SBP during recovery,

Fig. 3



Mean total (a) peripheral resistance and (b) cardiac index during the baseline, task and post-stress recovery trials. Values for men are shown in solid lines and women in dashed lines. Error bars are standard error of the mean (SEM).

independently of T1 resting SBP, age, gender, grade of employment, hypertensive medication, BMI and smoking.

Table 2 Prediction of 3-year increase in systolic blood pressure by post-task recovery effects

Predictor	Percentage showing increase in SBP $\geq$ 5 mmHg	Odds of increase in SBP $\geq$ 5 mmHg (95% CI)
SBP recovery trial 2		
Lowest tertile	26.1	1
Middle tertile	37.2	1.86 (0.85 to 4.08)
Highest tertile*	50.1	3.59 (1.19 to 10.8)
DBP recovery trial 2		
Lowest tertile	28.1	1
Middle tertile	27.5	0.94 (0.39 to 2.23)
Highest tertile†	58.1	4.10 (1.28 to 13.1)
Total peripheral resistance recovery trial 2		
Lowest tertile	32.0	1
Middle tertile	27.3	0.78 (0.33 to 1.83)
Highest tertile‡	52.3	2.82 (1.18 to 6.77)

*Adjusted for age, gender, grade of employment, hypertensive medication, BMI, smoking and T1 systolic blood pressure (SBP). †Adjusted for age, gender, grade of employment, hypertensive medication, body mass index, smoking, T1 SBP and baseline diastolic blood pressure (DBP). ‡Adjusted for age, gender, grade of employment, hypertensive medication, body mass index, smoking, T1 SBP and baseline total peripheral resistance. CI, confidence interval.

Change in SBP between T1 and T2 was also predicted by DBP during task ($P = 0.003$) and recovery trials ($P = 0.002$), independently of resting T1 DBP and other covariates. When these two effects were entered competitively into a stepwise regression, recovery DBP entered the model first and the effect of task values was no longer significant. The regression coefficient for the association between DBP during recovery trial 2 and SBP at T2 was $B = 0.32$ (CI 0.10–0.55, $P = 0.005$), independent of covariates. As shown in Table 2, 58.1% of participants with DBP in the highest recovery tertile showed an increase in SBP $\geq$ 5 mmHg, compared with 28.1% of those in the lowest tertile. The adjusted odds of an increase in SBP $\geq$ 5 mmHg were 4.10 (CI 1.28–13.1, $P = 0.018$) for participants whose DBP was in the highest tertile during the recovery period.

Changes in SBP over the 3-year period were not associated with heart rate, heart rate variability, or cardiac index during the stress session. But an effect of TPR was observed, since the increase in SBP between T1 and T2 was greater in people whose TPR was higher in recovery trial 2 ($B = 0.012$, CI 0.004–0.020, $P = 0.004$), independently of resting TPR, resting T1 SBP, gender, grade of employment, hypertensive medication, BMI and smoking. The proportion of participants with SBP increases $\geq$ 5 mmHg was 32.0% in the lowest and 52.3% in the highest tertile of TPR during recovery (adjusted odds 2.82, CI 1.18–6.77, $P = 0.02$). Gender did not interact with recovery effects in any of these analyses.

Prediction of DBP change

DBP at T2 was positively associated with T1 resting DBP ($P < 0.001$), BMI ($P = 0.01$) and negatively with smoking ($P = 0.02$). The change in DBP was also predicted by SBP during the post-stress recovery period, but not by reactions to tasks. DBP increased to a greater extent over the 3-year period in those with greater SBP during recovery trial 2, independently of T1 resting SBP and DBP, age, gender, grade of employment, BMI and smoking ($B = 0.20$, CI 0.08–0.33, $P < 0.001$). Table 3 summarizes the associations between post-task recovery BP and increases in DBP $\geq$ 3.5 mmHg between T1 and T2. Individuals in the highest tertile of SBP during post-task recovery trial 2 had substantially elevated odds of an increase in DBP $\geq$ 3.5 mmHg independently of covariates (odds ratio 8.59, CI 2.08 to 35.4, $P = 0.003$).

Changes in DBP between T1 and T2 were associated both with DBP measured during stress tasks ($P = 0.013$) and recovery trial 2 ($P = 0.01$), independently of resting DBP at T1. In stepwise regression, DBP during recovery entered the model first and the effect of task levels was not independently significant ($B = 0.22$, CI 0.06–0.38, $P = 0.009$). As shown in Table 3, increases in DBP $\geq$ 3.5 mmHg over 3 years were observed in 30.9% of participants with recovery DBP in the highest tertile,

Table 3 Prediction of 3-year increase in diastolic blood pressure by post-task recovery effects

Predictor	Percentage showing increase in DBP $\geq$ 3.5 mmHg	Odds of increase in DBP $\geq$ 3.5 mmHg (95% CI)
SBP recovery trial 2		
Lowest tertile	4.2	1
Middle tertile	17.1	2.77 (0.95 to 8.12)
Highest tertile*	34.1	8.59 (2.08 to 35.2)
DBP recovery trial 2		
Lowest tertile	12.8	1
Middle tertile	12.5	0.86 (0.29 to 2.60)
Highest tertile†	30.9	3.38 (1.14 to 10.1)

*Adjusted for age, gender, grade of employment, hypertensive medication, body mass index, smoking, T1 diastolic blood pressure (DBP) and baseline systolic blood pressure (SBP). †Adjusted for age, gender, grade of employment, hypertensive medication, body mass index, smoking and T1 DBP. CI, confidence interval.

compared with 12.8% for those in the lowest tertile ($P = 0.029$, see Table 3).

The change in DBP was not associated with heart rate, TPR, or cardiac index responses during the stress session. There was, however, a significant relationship with heart rate variability. Lower heart rate variability during stress tasks predicted changes in DBP from T1 to T2 independently of T1 resting DBP, resting heart rate variability, age, gender, grade of employment, hypertensive medication, BMI and smoking ($B = -0.83$, CI -0.30 to -0.07, $P = 0.002$). Some 26.4% of participants with heart rate variability in the lowest tertile increased DBP $\geq$ 3.5 mmHg over the 3-year period, compared with 9.6% of individuals in the highest tertile, but the adjusted odds were not significant (2.72, CI 0.71–10.5, $P = 0.15$).

Discussion

The main findings of this study were that increases in SBP and DBP over a 3-year period were predicted by impaired post-stress cardiovascular recovery. Although some associations with cardiovascular activity during stress tasks were observed, these were generally less strong than the recovery effects. Increases in SBP were predicted by high levels of SBP and DBP and TPR during the recovery period, while increases in DBP were predicted by high levels of SBP and DBP during recovery and by reduced heart rate variability during stress tasks. The impact of these variables was independent of the influence of baseline BP at T1, age, gender, socioeconomic status, hypertensive medication, smoking and BMI. Response rates were high, so selection factors are unlikely to have accounted for the pattern of results.

A number of previous studies have shown associations between longitudinal changes in BP and cardiovascular stress reactivity [4]. The literature is not all consistent, and it is notable that positive associations between stress reactivity and longitudinal changes have generally been recorded in young samples, but not in middle-aged cohorts such as the one studied here [6,21]. Impaired cardiovascular recovery has been related to longitudinal changes in BP in only one previous study, but the follow-up rate in that investigation was only 39%, so selection factors may have been operating [22]. It is plausible that post-stress recovery becomes more important as people grow older for two reasons. First, post-stress recovery tends to be rapid and efficient in young people; indeed, the majority of studies of recovery in young adults and adolescents have not continued monitoring for more than 10 min after stress termination [9]. The scope for individual differences to have an impact is therefore limited. Second, impairment of post-stress recovery may emerge after prolonged and repeated exposure to moderate stress activation over many decades. McEwan and colleagues [7,23] have described as allostasis the process of achieving stability of physiological systems in the face of environmental challenge. Chronic or repeated challenge over long periods may lead to a condition of allostatic load, a key element of which is chronic dysregulation of biological response systems. One manifestation of this state is failure to adapt efficiently post-stress. Several of the studies that have failed to show strong associations between cardiovascular stress reactivity and future BP have not included post-stress recovery in the analysis [6,21].

Predictors of increases in BP were analysed using both linear and logistic regression techniques in this study. Linear regression utilizes the complete range of data, but correlation and regression coefficients are typically low and relatively little of the variance in BP is accounted for. It has been argued that such methods may be misleading and underplay the clinical significance of associations [24]. By way of analogy, the substantial long-term mortality risk associated with raised BP that was described in the Chicago Heart Association Detection Project in Industry corresponds to a correlation of less than 0.1 (accounting for 1% of the variance) [25]. We therefore computed the odds of a definite increase in BP being associated with impaired post-stress recovery. The odds of definite increases in SBP were three-fold for individuals in the upper compared with the lower tertile of recovery BP and as high as 8.11 in the case of DBP.

This study was carried out with healthy middle-aged men and women with no signs of hypertension, CHD or diabetes at baseline, although a small number had been diagnosed and treated for hypertension by the time of follow-up. Their relatively healthy status is reflected in the fact that there were small decreases in DBP over the 3-year period in both men and women on average. The associations that were observed with post-stress recovery may therefore underestimate the significance of these factors in more representative samples, where upward drift in BP is more apparent [6].

The behavioural tasks utilized in this study elicited marked acute increases in SBP and DBP. These were sustained during task periods by a combination of raised cardiac output and TPR (Fig. 3). But during the post-task recovery period, heart rate and cardiac index fell below baseline levels, while the increased TPR was sustained. Thus the elevation in BP recorded during the recovery period was underpinned by vascular rather than cardiac responses. A similar pattern of transient increase in cardiac index and prolonged changes in TPR has been recorded during extended mental stress testing [26]. We do not know how long BP would take completely to return to baseline, since monitoring stopped after 45 min. Raised peripheral resistance in response to stress may be associated with enhanced cardiovascular disease risk [27]. For example, BP stress responses are maintained to a greater extent by vascular than cardiac adjustments in black compared with white hypertensives [28], young adults with hypertensive parents [29] and in lower socioeconomic status (SES) men and women [11]. Our group has also shown that lower SES individuals show more prolonged responses to stress in pro-coagulant haemostatic factors [30] and in inflammatory cytokine release [31].

This study has a number of limitations. Data were collected from middle-aged white men and women and results may not generalize to other populations. The sample size was not sufficient to investigate possible interactions between post-stress recovery and socioeconomic position, but this is potentially important in view of the greater risk of CHD in less privileged sectors of the population. Because of equipment problems, heart rate variability was only assessed in a proportion of participants and this may have limited our ability to investigate parasympathetic influences. Nonetheless, the findings add to the evidence that disturbances of post-stress cardiovascular regulation and the restitution of normal function are potentially significant for cardiovascular disease.

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