Cardiovascular response to mental stress in offspring of hypertensive parents: the Dutch Hypertension and Offspring Study

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Objective: To compare blood pressure-regulating mechanisms during mental stress in two groups of offspring with contrasting risk for hypertension.

Design: Cardiovascular reactivity to two different types of mental stressors was studied in adolescents and young adults with two hypertensive or two normotensive parents. The two tasks used were intended to evoke either a predominantly adrenergic cardiac response (a memory search task) or a predominantly vascular response (a reaction-time task with visual search and tone avoidance).

Methods: Blood pressure and heart rate were recorded at rest and during stress. To study adaptations of the cardiovascular system to mental stress, cardiac output, total peripheral resistance and indices of vagal and sympathetic influences on the heart were measured.

Results: The reactivity of systolic blood pressure (SBP) to the memory search task was significantly higher in offspring of hypertensive parents, which resulted in a longer recovery after the task. In contrast, during the reaction-time task, offspring of hypertensive parents had a significantly enhanced reactivity of peripheral resistance, but no differences in heart rate or blood pressure response were observed. No differences between the two groups were found in sympathetic or vagal activity during either task measured by the ratio of pre-ejection time and left ventricular ejection time, and respiratory sinus arrhythmia, respectively.

Conclusion: Apart from a higher reactivity of SBP during the memory search task, no other indications supporting the presence of hyperadrenergic activation of the heart in early primary hypertension were found. On the contrary, the results of the present study support the hypothesis that blood pressure responses in prehypertensive subjects are characterized by enhanced vasoconstriction rather than by increased cardiac output.

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Introduction

The aetiology of primary hypertension is still being debated. Several mechanisms have been proposed to explain why blood pressure rises with age in some subjects and remains at normal levels in others. Hypertensive subjects show hyper-reactivity of both heart rate and blood pressure to acute stress compared with normotensive controls [1–4]. According to one of the theories, hyper-reactivity is attributed to an enhanced reactivity of the sympathetic nervous system, and is supposed to precede the hypertensive state [5–7]. This hyper-reac-

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tivity appears to manifest itself more during exposure to behavioural stressors [8,9].

Epidemiological studies [10–12] have indicated that high blood pressure clusters in families partly because of shared genes. In several studies [13–19], children of hypertensive parents and normotensive parents have been compared with respect to their reactivity of blood pressure and heart rate to mental stressors, with equivocal results. A factor limiting the comparability of studies is the great diversity of tasks that have been used. A clear distinction between cardiovascular reactivity patterns resulting from behavioural tasks and from physical tasks is known, but has rarely been considered [8,20,21]. The known difference in response patterns to distinct types of mental stress has been even more neglected [8,21,22].

Sympathetic hyper-reactivity can theoretically be the result of either an enhanced stimulation of the heart [23], or an augmented increase in total peripheral resistance (TPR) [24]. In most studies cardiovascular measurements have been limited to heart rate and blood pressure, which are used as indices of sympathetic activity. Restriction to these measures makes it hard to reveal information about the basic blood pressure regulatory mechanisms involved.

In the present study two groups of offspring with a highly contrasting risk for hypertension were compared, on the basis of their family history of hypertension. The study was conducted in order to examine cardiovascular adaptations during two distinct types of behavioural tasks, a memory search task and a reaction-time task with visual search and tone avoidance, in two groups of subjects with a different family history of hypertension. By measuring cardiac output and TPR together with indices of sympathetic and parasympathetic nervous system activity, differences in blood pressure-regulating mechanisms between the two groups were evaluated. Potential confounding factors were also considered in the present study by including indices for obesity and fitness [25].

Subjects and methods

Subjects

The present study is part of the Dutch Hypertension and Offspring Study [26]. For the present study, subjects (mean age ±SEM 22.6±0.7 years) were selected who had either two hypertensive parents or two normotensive parents. They were selected from a large epidemiological study in the Dutch town of Zoetermeer. Between 1975 and 1978 all the residents from two districts of Zoetermeer were invited to participate in a screening of blood pressure and other cardiovascular risk factors [27].

Blood pressure was measured in 10 532 out of 13 462 eligible residents (78%). This group included 1642 couples with children. A stringent selection procedure (described previously [26]) was applied to these couples to select families with a maximal contrast in familial predisposition to hypertension.

Biological children (aged 8–33 years) of two groups of parents participated: couples of whom both were normotensive, and couples both of whom had hypertension. Individual parents with both systolic blood pressure (SBP) and diastolic blood pressure (DBP) in the upper (hypertensive) or lower (normotensive) quartile of the age and sex-specific blood pressure distribution were selected. Blood pressure must have been stable in either of the two quartiles over a period of 10 years. Those who were receiving antihypertensive medication were included in the hypertensive group. The blood pressure values and other characteristics of the parents and their children (subjects) at the time of enrolment have been described previously [28].

Fifty-six of the subjects (36 male, 20 female) with two hypertensive parents and 43 of the subjects (26 male, 17 female) with two normotensive parents participated in the present study. At the time they participated, all of the subjects were without serious medical problems and were not taking any medication that might have influenced the tests. They were asked to refrain from smoking, drinking alcohol and using caffeine-containing products for 24 h before they visited the research centre.

The Dutch Hypertension and Offspring Study is a collaborative undertaking supervised by a steering committee representing five Dutch universities and clinical research centres. The study protocol was approved by the ethical committee of the Erasmus University Medical School, and written informed consent was obtained from the subjects and their parents.

Stressors

Two active coping tasks were used in the study. The first was a memory search task modelled after the Sternberg memory search paradigm used by Schneider and Shiffrin [29]. The task has been shown to evoke relatively large increases in cardiac output and in adrenergic cardiac drive. Participants had to remember a set of three letters (the memory set), given to them before the task started [22]. Thereafter, sets of one to four letters (the test sets) were presented on the monitor, in which either none or one of the letters of the memory set was present. Subjects had to press the 'yes' or 'no' button for the presence or absence of any one of the memorized letters. The number of points that they could win depended directly on the speed of reaction.

The other task was a reaction-time task with visual search and tone avoidance. This task has been shown mainly to evoke an increase in vascular resistance. Subjects were seated behind a response panel with four buttons, one in each corner of the panel. During the task a stimulus was presented very briefly (500 ms) in one of the four corners of a video screen. They had to respond as fast as possible to the stimulus by pressing the button at their response panel opposite to the corner in which the stimulus was presented. Two consecutive correct responses were rewarded with 10 points. Incorrect or slow responses were punished with a loud noise burst and a

reduction of 10 points after two mistakes. The tasks have been described in detail elsewhere [22].

All of the subjects had the opportunity to practice the tasks for 3 min just before the real task was executed. Because of the differences between subjects in age and education, four different starting levels were available for both of the tasks. For both tasks performance levels were adapted to the subject according to their performance in the last minute of the training period, and during the task the performance level was adjusted to the performance of the subject each minute. This resulted in a performance level close to the maximum for all participants.

Cardiovascular measurements

Signal recording

The electrocardiogram (ECG) was recorded using an amplifier with time constant 0.3 s and 1 MΩ impedance. The impedance cardiogram (ICG) was recorded with a Nihon Kohden Impedance Cardiograph [30] utilizing silver chloride spot electrodes, as described previously [31]. The respiration signal was recorded with a hollow tube around the chest at a level 7 cm above the umbilicus. Respiration was measured as a function of the change in length of the tube caused by breathing ($\delta Z/\delta t$).

The ECG, $\delta Z/\delta t$ and the respiration trace were recorded at 250 Hz using an Olivetti PC M250 in combination with a 12-bit anologue-to-digital converter. Data were stored on a tape (Tecmar) for later offline processing. Blood pressure was measured every second minute with a Dinamap Vital Signs Monitor (Critikon model 845 XT) [32,33].

Signal processing

Of the original signals a mean ECG and mean ICG complex were computed for each 1 min period with reference to the ECG R-wave [34]. Of those signals the preejection period (PEP), the left ventricular ejection time (LVET), and the maximum value of $\delta Z/\delta t$ ($\Delta Z/\delta t_{max}$) were calculated. PEP was used as an index of β-adrenergic activity in the heart [35]. To correct for the influence of possible differences in afterload on PEP, PEP/LVET was used as an index of β-adrenergic activity. LVET and $\Delta Z/\delta t_{max}$ were used to calculate the stroke volume using a formula proposed by Kubicek *et al.* [30].

Heart rate was computed as the total number of interbeat intervals divided by the measuring time and expressed as beats/min. Cardiac output was computed by multiplying the stroke volume by the heart rate. Several authors have shown satisfactory correspondence between cardiac output derived from ICG and cardiac output assessed by other methods such as the dye-dilution technique [36], the thermodilution technique [37], and the Fick method [38]. The method was considered adequate because measurements have been found to be reliable in our laboratory. The correlation for LVET was high (0.97; two measurements with 3 weeks in between). The results for PEP were less reliable, although still quite high (correlation 0.83). The test–retest reliability of both cardiac output and TPR was acceptably high [difference in cardiac output 1.9 ± 3.1%, coefficient of variation 1.6 and regression coefficient 0.81 ± 0.11; difference in TPR 3.1 ± 4.4%, coefficient of variation 1.4 and regression coefficient 0.94 ± 0.14 (unpublished data)]. Mean values of blood pressure were calculated from all the measured values every 2 min. TPR was estimated from cardiac output and mean arterial pressure.

From the respiration signal and the ECG signal the magnitude of respiratory sinus arrhythmia (RSA) was computed using the peak-to-trough method [39], by subtracting the largest interbeat interval during the expiration period from the shortest interbeat interval during the inspiration period. Mean RSA in milliseconds was computed for each minute by averaging the RSA values over all breaths within that minute. Respiration rate was computed as the mean total cycle length in a 1 min period and expressed as cycles/min.

Physiological measures

Body weight and height were measured with the subjects wearing only light clothes and no shoes. The body mass index (BMI), an index of obesity, was calculated as body weight/(height)^2. To estimate the maximal aerobic power the subjects performed a supramaximal exercise test according to the protocol proposed by Åstrand and Rodahl [40] on an electrically braked bicycle ergometer (Tunturi EL400). The subjects started at a load of 0.5 W/kg body weight at a constant pedalling speed of 70 r.p.m. Children under the age of 14 years started at a load of 0.25 W/kg body weight. The load was increased every 3 min until the load reached a level 10% above the estimated maximal aerobic level in the previous period. The test was stopped when oxygen consumption rose no further or the subject gave up. Subjects were asked to breathe through a high-velocity, low-resistance mouthpiece with a minimal dead space that shunted all the expired air into an Oxycon (Mynhardt Ox4). This device calculates the amount of oxygen used and measures the air volume exhaled by the subjects.

Experimental protocol

After height and weight had been measured, the measurement devices were attached to the subjects. The subjects sat supine in a quiet temperature-controlled (20°C) and sound-shielded, dimly lit room. They faced a monitor on which the stimuli were presented. The physiological monitoring and the delivery of the stimuli were controlled from outside the room.

After an adaptation period, baseline measurements were recorded for 10 min during which the subject rested quietly. Then, the first task was explained to the subject and a 3 min period was given for practicing. Next, the first task was performed for 10 min followed by a recovery period of 5 min during which the subject sat quietly. The protocol was identical for both the tone avoidance reaction-time task and the memory search task. The order in which the tasks were presented to the subjects was randomized.

After a break of 25 min, during which the subjects were asked to relax, a new baseline was recorded for 10 min in order to assess the post-stress resting level. Maximal aerobic power was measured later the same day.

Data analysis

Mean values were calculated for all of the variables measured at rest (10 min) and during the tasks (10 min), and for the first 2 min of the recovery period after each of the two tasks. Post-stress resting levels were subtracted from levels measured during the tasks and the recovery periods in order to obtain reactivity measures of heart rate and blood pressure. All task levels of RSA and variables in which stroke volume was used were expressed as a relative change from the post-stress level.

For comparisons between the two groups, means and SEM are given, with two-sided P for the difference. Adjustments for the differences in age and the proportion of males across the two groups were made by multiple linear regression. Associations between variables for the group as a whole were adjusted for differences in group characteristics (using indicator variables for group) and for age, sex, BMI and fitness by multiple regression analysis when appropriate. The BMDP statistical software package was used for data analysis.

Results

Table 1 gives the baseline values of the physiological variables that were measured at rest. Although all subjects were still normotensive, blood pressure was significantly higher at rest in offspring of hypertensive parents [mean differences in SBP (±SEM) 6.0±1.6 mmHg; $P<0.01$; DBP 5.6±1.3 mmHg; $P<0.01$]. There was no difference between the two groups in maximal aerobic power or BMI. Similarly, no differences in performance on the two tasks between the two groups were observed, evaluated by mean reaction time and final score for the two tasks (Table 2).

Table 1. Measurements during the post-stress resting period in offspring of normotensive and hypertensive parents.

	Mean±SEM*			
	Offspring of two normotensive parents (A)	Offspring of two hypertensive parents (B)	Difference between A and B	P
Systolic blood pressure (mmHg)	111.2±1.3	118.2±1.2	6.99±1.77	<0.01
Diastolic blood pressure (mmHg)	65.7±1.0	71.2±0.9	5.57±1.31	<0.01
Heart rate (beats/min)	65.8±1.6	64.3±1.4	−1.53±2.11	0.47
PEP/LVET	0.4±0.0	0.4±0.0	0.01±0.02	0.47
RSA† (ms)	98.2±5.9	102.9±5.4	4.65±8.14	0.57
Respiration (cycles/min)	18.8±0.5	19.8±0.5	0.98±0.71	0.17

*Differences between groups are adjusted for differences in age and sex. †Adjusted for differences in respiration rate between the two groups. PEP/LVET, pre-ejection period/left ventricular ejection time; RSA, respiration sinus arrhythmia.

Table 2. Body composition, fitness and the performance on the two tasks in offspring of normotensive and hypertensive parents.

	Mean±SEM*			
	Offspring of two normotensive parents (A)	Offspring of two hypertensive parents (B)	Difference between B and A	P
Sex (male/female)	26/17	36/20		
Mean age (years)	21.5±1.1	23.4±0.9	1.97±1.43	0.17
Range	9.4–33.4	7.2–33.0		
No. smokers	6 (14%)	13 (23%)		
Height (cm)	172.0±1.7	173.9±1.5	1.89±2.29	0.41
Weight (kg)	65.7±1.9	68.2±1.6	2.52±2.50	0.32
BMI (kg/m^2)	21.5±0.4	22.3±0.3	0.74±0.52	0.16
Relative VO_2max (ml/kg×min)	40.3±1.1	38.5±0.9	−1.78±1.44	0.22
Reaction-time task score	343.7±28.5	301.6±23.7	−41.6±37.3	0.27
No. faults	51.1±3.8	56.3±3.2	5.22±4.98	0.30
No. trials	207.5±1.3	208.4±1.1	0.83±1.69	0.62
Memory search task score	−219.7±40.5	−163.7±34.0	56.04±53.23	0.30
Mean reaction time (ms)	1171.2±72.4	1243.8±60.8	72.56±95.17	0.53

*Differences between groups are adjusted for differences in age and sex. BMI, body mass index; VO_2max, maximal aerobic oxygen uptake.

Physiological reactivity

Memory search task

Table 3 gives the results of the measures of reactivity to the memory search task, which is supposed to induce greater cardiac activation than the reaction-time task. Subjects with hypertensive parents had a higher SBP response to this task (difference in response 3.0±1.5 mmHg; $P=0.05$). No differences were found in DBP or heart-rate reactivity, or in change of sympathetic or vagal activation of the heart, as indicated by PEP/LVET and RSA. Neither were there any differences in change of stroke volume, cardiac output and TPR for this task.

Reaction-time task with tone avoidance

As expected the reaction-time task mainly showed a peripheral vasoconstrictive effect and only a small cardiac effect. As indicated in Table 4 the rise in calculated TPR was higher in subjects with hypertensive parents (difference 16.2±7.1%; $P=0.03$). This did not result in a more pronounced rise in blood pressure, most probably because of a greater decrease in stroke volume in subjects with hypertensive parents (difference 6.3±3.0%; $P=0.04$). The difference in cardiovascular reactivity patterns between the two tasks is clearly depicted in the response of cardiac output and TPR (Fig. 1).

To assess whether the difference in reactivity to each of the two stress tasks was confounded by a difference in blood pressure between the two groups, adjustments for baseline SBP were made by addition of SBP to the regression analysis. However, the difference in reactivity of SBP to the memory search task between the two groups became larger (difference adjusted for rest level 5.6±1.8 mmHg; $P<0.01$). Adjustment for differences in blood pressure between the two groups resulted in a slight reduction in the difference in reactivity of stroke volume and TPR (difference in stroke volume adjusted for blood pressure −5.7±3.2%; $P=0.08$; difference in TPR after adjustment for blood pressure 15.0±7.6%; $P=0.05$).

In the recovery period after each of the two stressors all of the physiological variables returned to baseline levels for both groups, with the exception of SBP and PEP/LVET after the memory search task. The SBP remained 2.9±1.3 mmHg ($P=0.06$) above the resting lev-

Table 3. Reactivity to the memory search task in offspring of normotensive and hypertensive parents.

	Mean reactivity±SEM*			
Change in	Offspring of two normotensive parents (A)	Offspring of two hypertensive parents (B)	Difference between B and A	*P*
Systolic blood pressure (mmHg)	4.8±1.1	7.7±1.0	2.99±1.51	0.05
Diastolic blood pressure (mmHg)	4.1±0.9	4.9±0.8	0.80±1.17	0.50
Heart rate (beats/min)	7.9±0.9	7.6±0.8	−0.23±1.21	0.85
PEP/LVET ($\times 10^{-2}$)	−0.4±0.7	−0.7±0.6	−0.34±0.90	0.70
RSA† (%)	−34.6±3.4	−35.9±3.1	−1.30±4.59	0.78
Stroke volume (%)	−2.7±2.3	−6.4±1.9	−3.71±3.01	0.22
Cardiac output (%)	4.1±4.1	3.0±3.5	−1.11±5.41	0.84
Total peripheral resistance (%)	5.3±5.4	10.2±4.6	4.94±7.16	0.49
Respiration rate (cycles/min)	2.4±0.5	2.2±0.5	−0.18±0.70	0.78

*Differences between groups are adjusted for differences in age and sex (post-stress resting levels are taken as baseline). †Adjusted for differences in respiration rate between the two groups. PEP/LVET, pre-ejection period/left ventricle ejection time; RSA, respiration sinus arrhythmia.

Table 4. Reactivity to the reaction-time task in offspring of normotensive and hypertensive parents.

	Mean reactivity±SEM*			
Change in	Offspring of two normotensive parents (A)	Offspring of two hypertensive parents (B)	Difference between B and A	*P*
Systolic blood pressure (mmHg)	6.3±1.2	8.0±1.1	1.72±1.63	0.29
Diastolic blood pressure (mmHg)	5.0±1.0	5.8±0.9	0.77±1.30	0.56
Heart rate (beats/min)	7.5±1.0	8.3±0.8	0.79±1.31	0.55
PEP/LVET ($\times 10^{-2}$)	−0.2±0.7	−0.6±0.6	−0.42±0.94	0.65
RSA†(%)	−30.1±3.1	−30.5±2.8	−0.87±4.19	0.84
Stroke volume (%)	−5.2±2.4	−11.5±1.9	−6.27±3.02	0.04
Cardiac output (%)	1.6±3.9	−3.7±3.3	−5.34±5.08	0.30
Total peripheral resistance (%)	4.0±5.5	20.2±4.5	16.23±7.12	0.03
Respiration rate (cycles/min)	2.2±0.5	1.9±0.5	−0.36±0.72	0.62

*Differences between groups are adjusted for differences in age and sex (post-stress resting levels are taken as baseline). †Adjusted for differences in respiration rate between the two groups. PEP/LVET, pre-ejection period/left ventricle ejection time; RSA, respiration sinus arrythmia.

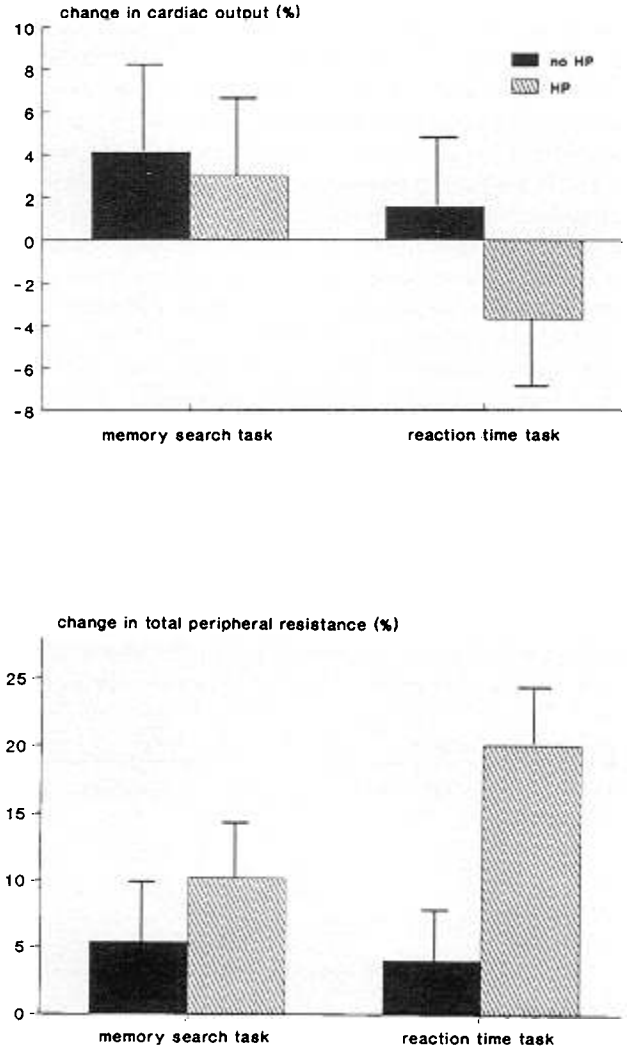

Fig. 1. Reactivity of cardiac output and total peripheral resistance to the two mental stress tasks in offspring of two hypertensive parents (HP) and of two normotensive parents (no HP).

els in the offspring of hypertensive parents 2 min after the task had been finished. This was probably the result of the higher SBP during the memory search task, because the difference between the groups disappeared after adjustment for the reactivity of SBP to the task (difference 1.0 ± 1.1 mmHg; $P=0.37$). PEP/LVET was marginally lower (-0.015 ± 0.008; $P=0.06$) in offspring of hypertensive parents during the recovery period, suggesting a higher sympathetic drive to the heart. This appeared not to be the result of a higher sympathetic drive during the task, because after adjustment for PEP/LVET reactivity during the task the difference in the ratio did not disappear (difference -0.013 ± 0.007; $P=0.07$). For both the tasks and the recovery periods no indications of any influence of performance level, BMI or fitness on the reactivity measures were detected.

Discussion

In the present study two groups of adolescents and young adults at different risks for hypertension were compared in their reactivity to two different mental stress tasks. The results indicate that the SBP response to the memory search task is enhanced in offspring of hypertensive parents. Furthermore, in this group average SBP levels are higher in the recovery period following the task, probably as a result of the higher SBP level during the task. A larger increase in calculated TPR in offspring of hypertensive parents was observed during the reaction-time task. This is not accompanied by clear differences in blood pressure or heart rate, probably because of a concomitant decline in stroke volume.

During the two tasks no detectable differences in sympathetic or vagal drive to the heart could be detected. This is particularly remarkable for the memory search task. Confrontation with stressors of this kind has been reported to result in a marked cardiac response. This has been attributed to a higher sympathetic drive [21,22]. The absence of a difference in neural activity between the two groups might be the result of the moderate sensitivity of the PEP and RSA measurements, although we cannot rule out the possibility that the two groups do not differ in neural activity on the heart.

The enhanced increase in TPR in offspring of hypertensive parents during the reaction-time task could be an indication of an increased α-adrenergic responsiveness of peripheral vessels, resulting in vasoconstriction. This notion is also supported by the tendency toward a larger response of the TPR to the memory search task. Enhanced reactivity of diastolic blood pressure described in literature is an indication in the same direction [14,41]. In this respect it is worth noting that we have observed an increased density of α_2-adrenoreceptors on platelets in the offspring of hypertensive parents (unpublished observations). This finding and its relation to the present observations requires further attention.

Subjects were selected if SBP and DBP of both their parents were in either of the most extreme quartiles of the blood pressure distribution. Larger contrasts in blood pressure measured between the parents are related to larger differences in risk for hypertension in the offspring [42]. Consequently, the number of real prehypertensive subjects in the group offspring of hypertensive parents depends directly on the selection method. However, because hypertension clusters in families and blood pressure tracks within subjects, blood pressure is most likely to be already elevated at an early age in children of hypertensive parents [10,43,44]. Higher levels of baseline blood pressure have been described, and differences have been depicted mainly in terms of daytime SBP [45,46]. Although none of the subjects in the present study had

evident hypertension it is conceivable that the difference in blood pressure between the groups might have caused the differences in cardiovascular reactivity rather than vice versa. Although it is difficult to exclude this possibility, one approach could be to adjust the observed differences for the difference in blood pressure between the groups. Adjustment for differences in SBP did not clearly affect the difference in response of SBP to the memory search task or of stroke volume and TPR to the reaction-time task. However, it should be noted that adjustment for blood pressure level might obscure true differences in characteristics related to the development of high blood pressure, because the offspring with the highest blood pressure may be those with the highest risk for future hypertension.

The results presented here provide arguments against sympathetic overactivation of the heart (the hyper-β-adrenergic hypothesis) and are in favour of the hypervascular reactivity theory. The results are also in accordance with the recently proposed idea that blood pressure responses in subjects at risk for hypertension are primarily the result of peripheral vasoconstriction during stress, whereas in low-risk subjects blood pressure responses are largely caused by an increased cardiac output [47,48]. This difference in haemodynamic pattern is considered to be the result of a more pronounced vascular activation pattern in prehypertensive subjects.

In most previous studies [13–19], small but statistically significant differences between offspring of normotensive and hypertensive parents in terms of the SBP or DBP response to mental stress were observed. Comparison between those studies and the present study is difficult because of the variety in relative contribution of vascular and cardiac components in reactivity to different types of mental stress. In general, tasks based on mental effort, such as memory search tasks or mental arithmetic tasks, induce relatively strong β-adrenergic cardiac impulses and vasodilation. Greater increases in forearm blood flow during mental arithmetic in offspring of hypertensive parents have been demonstrated [49]. This could result in a higher SBP in offspring of hypertensive parents. However, sensory intake tasks, such as reaction-time tasks, give a more pronounced rise in peripheral resistance, which might result in a higher response of the DBP in offspring of hypertensive parents [8,21].

When comparing mean reactivity measures over stress periods the duration of the tasks might be important. If hyper-reactivity in prehypertensive subjects is the result of overactivation of the cardiovascular system in adaptation to the novelty of the stressor then it will only manifest itself in the first minutes of the task. However, analyses of cardiovascular reactivity data in which only the first 2 min of each of the two tasks were included show results that are essentially similar to those presented here for the whole task period.

In conclusion, our findings suggest that adolescents and young adults with a positive family history of hypertension have already elevated resting blood pressure levels and may also have an enhanced cardiovascular reactivity to mental stressors compared with subjects with normotensive parents. Despite a higher SBP response to a memory search task, no indications were found for the presence of β-adrenergic hyper-reactivity of the heart during mental stress. The results of the present study support the hypothesis that blood pressure responses in prehypertensive subjects are caused by an enhanced vasoconstriction rather than an increased cardiac output. Moreover, it is essential to consider the type of mental stress task when interpreting the differences between groups with and without parental history of hypertension.

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