Cardiovascular control and plasma catecholamines during rest and mental stress: effects of posture

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ABSTRACT

In order to understand the complex autonomic adjustments that occur during the psychological challenges of normal daily life, autonomic responses to psychological stress were studied by evaluating the effects of body posture on various indices of sympathetic and parasympathetic regulation during performance of a psychological task. Twelve male subjects were studied in various postures (supine, sitting and standing), and during performance of the Colour Word Test (CWT) when sitting and then when standing. This procedure was subsequently repeated in reverse order (first standing and then sitting) after 15 min of supine rest. Blood samples for assay of plasma catecholamines were obtained before and during each CWT. Spectral analysis of beat-to-beat variations of heart rate (HR) and blood pressure (BP) was applied in order to obtain non-invasive indices of sympathetic and parasympathetic regulation. HR, diastolic BP, mid-frequency band power (0.07–0.14 Hz) of HR and systolic BP, and plasma adrenaline and noradrenaline concentrations showed significant increases when changing from supine to sitting to standing posture, whereas high-frequency band power (0.15–0.50 Hz) of HR decreased in a posture-dependent fashion. In the sitting position, the CWT caused significant increases in HR, BP and plasma adrenaline levels, and decreased HR and BP variability indices. In the standing posture, the CWT responses differed significantly from those during sitting for HR (a mild decrease during standing), high-frequency band power of HR (decreased more while sitting), high-frequency band power of BP (decreased more while standing), and plasma adrenaline responses (larger during sitting). Posture-related differential effects were observed on indices of sympatho–adrenomedullary activation during performance of a psychological challenge, whereas indices of parasympathetic activity indicated primarily less vagolytic effects when the task was performed in the standing posture. Our findings therefore underline the complexity of the adjustments that occur in neurohumoral and haemodynamic parameters during the psychological challenges of daily life.

INTRODUCTION

The interaction between psychological stress and cardiovascular control processes in relation to the exacerbation of cardiovascular diseases such as hypertension and coronary heart disease has been studied by evaluating the cardiovascular reactions to standardized stressful situations in the laboratory and/or to real-life stressful situations in the natural environment by means of ambulatory monitoring of cardiovascular signals. Subse-
sequently, these responses have been related to the future development or current aspects of cardiovascular disease. One of the key issues here is the hypothesis that consistently high levels of cardiovascular reactivity to psychological or behavioural stressors may contribute to the long-term development of hypertension and/or coronary heart disease, as well as affecting their prognosis. Some support for such relationships has been found (e.g. [1–3]), although increased reactivity may be only one of the factors involved in the association between psychological processes and cardiovascular disease.

Reactivity to psychological stressors in the laboratory, usually studied when the subject is in the sitting position, has been used to establish details of the temporal and situational stability of cardiovascular response profiles (e.g. [4–6]). In relation to temporal reactivity stability, a recent meta-analysis [7] revealed that the reproducibility of laboratory stress reactivity is highest for heart rate (HR), followed by systolic and diastolic blood pressure (BP), but a drop in stress reproducibility occurs as the test–retest interval increases; this poses limitations on the use of laboratory measures of cardiovascular reactivity as potential risk factors for coronary heart disease. Ambulatory cardiovascular studies have as an advantage that reactions to real-life psychological and behavioural stressors can be evaluated, which may be more advantageous for describing relationships with disease. However, posture- and mobility-related activities during daytime ambulation have substantial cardiovascular effects in themselves which may interact with the stress-related reactivity profiles, making unequivocal interpretation of situational and temporal aspects of reactivity difficult [8]. It is apparent that an association between reactivity and disease can only be evaluated accurately when reliable measures of reactivity are studied in their contextual nature.

Sherwood and Turner [9] addressed the stability of haemodynamic responses to mental stress reactions in seated and standing subjects in the laboratory. They found that, although BP responses always increased when the mental arithmetic task was performed in a sitting or standing position, the underlying haemodynamic determinants of the pressure responses were different for the two postures. Increased cardiac output appeared to be responsible for the rise in BP when the task was performed in the sitting position, whereas increased vascular resistance was responsible for the pressor response when the task was performed in the standing position. This study revealed that these haemodynamic responses to stress during different postures were reproducible. At present, no detailed data are available regarding the influence of body posture on parasympathetic and catecholaminergic reactions to mental stress, or the stability of these reactions to mental stress.

We aimed to evaluate the effects of body posture on various indices of sympathetic and parasympathetic autonomic control during performance of a challenging psychological task. Venous plasma adrenaline and nor-adrenaline concentrations were used as indices of adrenomedullary and sympathoneural regulation respectively, whereas spectral analysis of spontaneous beat-to-beat variations in HR and BP was used to obtain non-invasive indices of sympathetic and parasympathetic processes within short-term cardiovascular control [10–12]. Furthermore, we computed relationships between systolic BP (SBP) and inter-beat interval time series in the frequency domain, to obtain non-invasive indices of baroreflex sensitivity (reflecting the arterial baroreflex control of the HR) [13,14]. In the present study, the stability of cardiovascular and catecholaminergic responses to changes in body posture (supine, sitting, standing) was assessed by measuring each posture twice; similarly, the autonomic reactivity profiles during performance of a challenging psychological task were studied twice with a sitting and twice with a standing posture. By combining different indices of sympathetic and parasympathetic autonomic regulation, we hoped to gain more insight into some of the complex autonomic adjustments that occur during the psychological and behavioural challenges of normal daily life, as well as the reproducibility of these reactions. Moreover, this may contribute to a further understanding of the complexity of the relationship between reactivity and cardiovascular disease.

METHODS

Subjects and procedures

Twelve males with a mean age of 22 years (range 19–25 years) participated in this study after providing written informed consent. The subjects were paid volunteers, recruited from a student population by means of advertisement. The selection procedure included a comprehensive medical and psychological screening to exclude subjects with a personal or family history of psychiatric illness or substance abuse. All subjects were in good health; they had been medication-free for at least 2 months prior to the study, and did not smoke more than five cigarettes per day. The experimental procedures were in accordance with the Declaration of Helsinki (1989) of the World Medical Association, and were approved by the Medical Ethical Review Committee of the University Hospital Rotterdam – Dijkzigt.

At 3 days before the experiment, the subjects were instructed to keep a regular sleep/wake pattern and to avoid abnormal physical or psychic exertions. On the experimental day, the subjects were asked to take their normal breakfast at home, without coffee or tea. The experimental session in the laboratory was always per-
formed in the morning, between 8.15 and 12.15 hours. Coffee, tea and smoking were prohibited before or during the recordings, but drinking mineral water was allowed. Physiological and biochemical measurements were obtained during the following procedures: (a) two periods of supine rest (15 min each), (b) two periods of quiet sitting (10 min each), (c) two periods of active standing (10 min each); the subject stood for 10 min at ease in the upright position), and (d) four times during performance of the Colour Word Test (CWT) (10 min each); The CWT was performed twice while the subject was in the sitting position and twice while standing. The sequence of the procedures was as follows. After 15 min of supine rest and 10 min of sitting, the subjects performed the CWT in the sitting position. This was followed by 10 min of standing and performance of the CWT in the standing position. After 15 min of supine rest, the procedure was repeated in reverse order (first standing, then sitting). During each period of supine rest, sitting or standing, the subject was requested to relax, not to speak, to breath regularly and to stay awake. The CWT consisted of the presentation on videotape of four words (red, green, blue, yellow), one word at a time, in four different colours (red, green, blue, yellow). The subject had to indicate the colour of the word on an answer sheet, with the specific request to do his very best. The test induces cognitive conflict [15], while time-pressure effects are added due to the rapid presentation of the stimuli [16–18]. In order to become familiar with the requirements of the task, a brief practice session was allowed at the intake of the experiment.

**Measurements and analyses**

**Biochemical assays**

At 45 min prior to the start of the measurements, a catheter (Venflon, 18G; Viggo AB, Helsenborg, Sweden) was inserted into an antecubital vein of the non-dominant forearm, for serial blood sampling. For determination of plasma adrenaline and noradrenaline concentrations, blood (10 ml) was collected in chilled heparinized tubes containing 12 mg of glutathione, after each supine, sitting and standing period, and after 5 min during performance of the CWTs. The blood samples were centrifuged at 4 °C (15 min, 3000 g); plasma was subsequently stored at −70 °C until assay. Catecholamines were assayed by means of HPLC with fluorimetric detection after isolation from plasma by a specific liquid/liquid extraction method and derivatization with the selective fluorogenic agent 1,2-diphenylethylenediamine [19].

**Physiological signals**

ECG, BP and respiration were recorded continuously during the session on a multichannel FM-type analogue recorder (Racal Store 14 DS, Sarasota, FL, U.S.A.). The ECG was derived using a precordial lead, amplified by means of a polygraph (Nihon Kohden, Tokyo, Japan). BP was recorded using a servo-plethysmomanometer for continuous non-invasive measurements of finger arterial BP, employing the volume-clamp technique of Penaz and co-workers [20,21] (Finapres 2300 NIBP; Ohmeda, Englewood, CO, U.S.A.). The cuffed middle finger of the non-dominant hand was kept at the level of the heart throughout the postural changes, in order to optimize the correspondence with intra-brachial pressure changes [22]. Thoracic and abdominal respiration were measured by means of impedance plethysmographs (Nihon Kohden). Adhesive disposable Ag/AgCl electrodes were used for the thoracic and abdominal recordings, placed at the level of the nipples and the abdomen respectively.

**Analyses of physiological signals**

The signals were digitized off-line at a sample frequency of 1000 Hz on a personal computer (Dell Optiplex GX5166) connected to an Analogue/Digital converter (DJ-200 PGH; Dataq Instruments). The interval between successive R-waves (interbeat interval) of the ECG was detected with an accuracy of 1 ms and transposed to HR series. SBP and diastolic BP (DBP) values were defined per interbeat interval of the ECG. Time series of interbeat interval, SBP and DBP were scrutinized for artifacts by means of visual inspection. Good-quality data were averaged in 5 min segments for the subsequent periods of supine, sitting and standing posture. For the CWT data, results from the first 1 min were discarded from analyses; the initial effects of the task induced too much instability for reliable interpretation of short-term variability in the cardiovascular time series. The data from the subsequent 4 min time period during performance of the CWT were used for spectral analyses.

**Spectral analyses of cardiovascular signals**

The 5- or 4-min segments of time series of HR, SBP and DBP were subjected to a discrete Fourier transformation based on non-equidistant sampling of the R-wave incidences (CARSPAN program) [23,24], in order to obtain power spectra of the rhythmic oscillations over a frequency range of 0.02–0.5 Hz, with a resolution of 0.01 Hz. For each time segment, the power was calculated for the low-frequency band (0.02–0.06 Hz), the mid-frequency band (0.07–0.14 Hz) and the high-frequency band (0.15–0.50 Hz), in addition to mean values and coefficients of variation for inter-beat interval, SBP and DBP (an example of beat-to-beat time series and spectra is shown in Figure 1). In order to reduce the number of parameters, presentation of spectral parameters is limited to the mid- and high-frequency band power of HR and SBP. Spectral energy was expressed in relative terms, i.e. as a fraction of the mean value of the considered signal during a particular time segment (squared modulation index, to be compared with squared variation coefficient) [25]. As an index of baroreflex sensitivity (BRS) per time
J. H. M. Tulen, F. Boomsma and A. J. Man in ’t Veld

Figure 1  Examples of 5-min periods of interbeat interval (IBI) variability (in ms) in one subject during supine rest, active standing and CWT performance (mental load)

The corresponding power spectra of the time series are presented next to the raw data. Note the changes in power of the relevant frequency bands from supine to standing, and the effects of the CWT.

segment, the gain (or modulus) in the mid-frequency band between the systolic pressure values and the interbeat interval times was computed, based on those frequency points within the 0.07–0.14 Hz range with a coherence of greater than or equal to 0.35 [14].

Respiration
Per time segment, samples of the respiratory signal were obtained at each incidence of the R-wave. Subsequently, these respiratory time series were subjected to spectral analyses [26]. Power spectra of the respiratory time series were evaluated primarily to assess if changes in cardiovascular variability due to body posture or mental load were related to changes in respiratory frequency. Per time segment, the dominant respiratory frequency in the power spectrum was assessed.

Statistical analysis
Data are presented as means (S.D.). A logarithmic transformation was applied to the cardiovascular spectral data because of skewness of the distributions. The data from the final 5 min of each supine, sitting or standing period were used for the statistical analyses, in addition to the data from the first part of the CWT performance (4 min period). Statistical analyses were performed using the SPSS for Windows Release 6.0 (SPSS Inc.). Two-factor multivariate analyses of variance (MANOVA) for repeated measurements were used to explore the posture-dependent effects and the reproducibility of these effects on the cardiovascular and catecholaminergic data: factor POSTURE (supine, sitting, standing) and factor REPEAT (CWT1, CWT2), as well as the interaction between factors POSTURE and REPEAT, were used to assess posture-related differences in response magnitudes as well as habituation effects to the CWT. A \( P \) value of < 0.01 was used to indicate a significant effect.

RESULTS

Postural effects

Plasma catecholamines
Plasma noradrenaline concentrations showed a significant and reproducible posture-dependent increase (Table 1). Based on the averaged data, plasma noradrenaline concentrations increased by 41% from supine to sitting, whereas from supine to standing a 161% increase was observed. Plasma adrenaline concentrations also increased significantly from supine to sitting to standing, although a significant interaction effect between factors POSTURE and REPEAT (\( F = 4.5, P < 0.05 \)) revealed that these effects were less stable. On average, the plasma adrenaline concentrations increased from supine to sitting by 50% and from supine to standing by 170%.

HR and BP
Mean HR increased significantly from supine to sitting and to standing (Figure 2; Table 1). For all body postures,
Table 1  Cardiovascular, respiratory and catecholaminergic parameters measured during the first and second periods of supine rest, sitting and active standing

Results are means (S.D.). F and P values of the MANOVAs for repeated measurements for factors POSTURE (supine, sitting, standing), and REPEAT (first period, second period) are also indicated. Abbreviations: Var. coeff., coefficient of variation; MFB, mid-frequency band; HFB, high-frequency band; Resp. freq., respiratory frequency; df, degrees of freedom; ns, not significant.

### MANOVA (F and P values)

<table>
<thead>
<tr>
<th></th>
<th>Supine 1</th>
<th>Supine 2</th>
<th>Sitting 1</th>
<th>Sitting 2</th>
<th>Standing 1</th>
<th>Standing 2</th>
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<tbody>
<tr>
<td>HR (beats/min)</td>
<td>63 (8)</td>
<td>61 (7)</td>
<td>66 (8)</td>
<td>64 (8)</td>
<td>88 (10)</td>
<td>83 (8)</td>
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<td>Var. coeff. (%)</td>
<td>6.7 (2.2)</td>
<td>9.0 (3.4)</td>
<td>7.4 (2.2)</td>
<td>8.3 (1.9)</td>
<td>8.6 (3.9)</td>
<td>9.1 (2.9)</td>
</tr>
<tr>
<td>MFB (log power)</td>
<td>6.8 (0.6)</td>
<td>7.4 (0.6)</td>
<td>7.3 (0.6)</td>
<td>7.6 (0.6)</td>
<td>7.5 (0.9)</td>
<td>7.8 (0.6)</td>
</tr>
<tr>
<td>HFB (log power)</td>
<td>7.4 (0.9)</td>
<td>7.8 (0.8)</td>
<td>7.4 (0.8)</td>
<td>7.7 (0.7)</td>
<td>6.1 (0.9)</td>
<td>6.4 (0.4)</td>
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<tr>
<td>SBP (mmHg)</td>
<td>111 (12)</td>
<td>112 (9)</td>
<td>117 (11)</td>
<td>122 (13)</td>
<td>117 (7)</td>
<td>121 (8)</td>
</tr>
<tr>
<td>Var. coeff. (%)</td>
<td>5.4 (1.8)</td>
<td>5.5 (1.4)</td>
<td>5.6 (1.3)</td>
<td>5.2 (1.2)</td>
<td>6.8 (1.9)</td>
<td>6.6 (1.5)</td>
</tr>
<tr>
<td>MFB (log power)</td>
<td>5.6 (0.8)</td>
<td>5.8 (0.3)</td>
<td>6.0 (0.6)</td>
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<tr>
<td>HFB (log power)</td>
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<td>4.8 (0.5)</td>
<td>5.2 (0.5)</td>
<td>5.2 (0.5)</td>
<td>5.8 (0.6)</td>
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</tr>
<tr>
<td>DBP (mmHg)</td>
<td>62 (9)</td>
<td>65 (5)</td>
<td>68 (8)</td>
<td>73 (8)</td>
<td>75 (8)</td>
<td>78 (7)</td>
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<tr>
<td>Var. coeff. (%)</td>
<td>5.4 (1.8)</td>
<td>5.5 (1.4)</td>
<td>5.6 (1.3)</td>
<td>5.2 (1.2)</td>
<td>6.8 (1.9)</td>
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<tr>
<td>MFB (log power)</td>
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<tr>
<td>HFB (log power)</td>
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<td>4.8 (0.5)</td>
<td>5.2 (0.5)</td>
<td>5.2 (0.5)</td>
<td>5.8 (0.6)</td>
<td>5.8 (0.5)</td>
</tr>
<tr>
<td>Noradrenaline (pg/ml)</td>
<td>159 (44)</td>
<td>163 (47)</td>
<td>226 (45)</td>
<td>227 (47)</td>
<td>418 (109)</td>
<td>421 (116)</td>
</tr>
<tr>
<td>Adrenaline (pg/ml)</td>
<td>10 (5)</td>
<td>9 (3)</td>
<td>11 (5)</td>
<td>18 (9)</td>
<td>26 (15)</td>
<td>27 (16)</td>
</tr>
</tbody>
</table>

Figure 2  Effects of body posture (supine, sitting, standing) on (a) HR, (c) log power mid-frequency band (MFB) fluctuations of HR, and (e) log power high-frequency band (HFB) fluctuations of HR sup1 and sup2: first and second periods of supine rest; sit1 and sit2, first and second periods of sitting; stand1 and stand2, first and second periods of standing. The effects of sitting and standing on the corresponding CWT response magnitudes of the HR parameters are depicted next to the postural effects: (b) HR response magnitude, (d) log power mid-frequency band response magnitude, and (f) log power high-frequency band response magnitude.
the second period induced significantly lower HR values than the first period (Table 1; factor REPEAT, \( P < 0.01 \)). Based on the averaged data, the mean HR increased by 5% from supine to sitting and by 39% from supine to standing. Regarding HR variability measures, overall variability (variation coefficient) did not change in a posture-dependent fashion. Mid-frequency HR fluctuations showed a trend towards a significant posture-dependent increase (\( P < 0.05 \); supine to sitting, +5%; supine to standing, +8%), whereas high-frequency HR fluctuations decreased significantly, primarily from supine to standing (supine to sitting, −1%; supine to standing, −18%). However, all HR variability measures showed non-reproducible effects, with the second period of measurement for each body posture revealing significantly greater variability than the first period (Figure 2; Table 1).

Regarding SBP and variability in SBP, significant and reproducible posture-related effects were found for all parameters (Table 1). SBP increased slightly from supine to sitting, but did not change much from sitting to standing (supine to sitting, +7%; supine to standing, +6%). The SBP variability measures increased primarily during active standing (mid frequencies: supine to sitting, +4%; supine to standing, +23%; high frequencies: supine to sitting, +7%; supine to standing, +20%). DBP showed a significant posture-dependent increase (supine to sitting, +11%; supine to standing, +20%). In addition, during the second period of measurement for each posture, DBP appeared higher than during the first period (\( P < 0.05 \); Table 1).

BRS

BRS was reduced in a posture-dependent fashion, particularly during active standing (Table 1) (supine to sitting, −3%; supine to standing, −53%). During the second period of each posture, BRS was higher than during the first period (\( P < 0.01 \); Table 1).

Respiration

Body posture had no significant effect on dominant respiratory frequency (Table 1).

Body posture and CWT response magnitudes

Plasma catecholamines

The CWT had no significant effect on plasma noradrenaline concentration (Table 2); the response magnitudes were variable and, on average, similar for sitting and standing (sitting, +0%; standing, +2%). For plasma adrenaline responses, we observed a small postural effect (trend effect: \( P < 0.05 \)): when performed while being seated, the CWT induced an average increase in the plasma adrenaline concentration of 55%, whereas the adrenaline responses were less strong when the CWT was performed in the standing position (+17%) (Table 2).

HR and BP

Mean HR responses to the CWT were posture-dependent (Figure 2; Table 2). During sitting, the CWT
induced an average increase in HR of +10%, whereas the HR response to the CWT during standing was a minor decrease (−2%). HR response magnitudes to the second CWT were smaller than responses to the first CWT, for both sitting and standing (interaction effect: POSTURE × REPEAT, F = 9.1, P < 0.01) (Figure 2). The CWT significantly reduced overall HR variability, as well as mid- and high-frequency variability (Figure 2). However, of the HR variability parameters, only the CWT response magnitudes of the high-frequency fluctuations revealed a posture-dependent effect: when the CWT was performed standing, the decrease in high-frequency variability was smaller (on average −4%) than when the CWT was performed in the sitting position (−9%).

SBP and DBP response magnitudes to the CWT showed no posture-dependent effect: the CWT always induced a significant increase in BP (Table 2). However, significant habituation effects were present: the response magnitudes to the second CWT during sitting or standing were lower than those to the first CWT. On average, SBP during sitting increased by 10% during CWT performance (DBP: +13%), and SBP during standing increased by 11% (DBP: +11%). The CWT significantly reduced BP variability parameters when performed in the sitting or standing position. Changes in body posture did not affect response magnitudes of mid-frequency fluctuations of SBP; on average, the CWT induced a decrease of mid-frequency SBP fluctuations of 12% during sitting and of 6% during standing. Regarding the CWT response magnitudes of the high-frequency SBP fluctuations, a significant posture-dependent effect was found: response magnitudes during standing (−15%) were larger than those during sitting (−12%).

**BRS**

BRS was not significantly affected by the CWT, either during sitting or during standing (Table 2).

**Respiration**

Body posture did not influence the significant increase in respiratory frequency observed during performance of the CWT (on average: sitting, +25%; standing, +17%) (Table 2).

**CWT performance**

Body posture had no significant effect on CWT performance (Table 2).

**DISCUSSION**

**Postural effects**

All parameters changed in the expected direction (e.g. see [27–29]): HR, DBP, mid-frequency band power of HR and SBP, and plasma adrenaline and noradrenaline concentrations increased, whereas high-frequency band power of HR decreased. In addition, BRS decreased in a posture-dependent manner, while no significant changes were observed in respiratory frequency. In this group of subjects, mean SBP increased slightly on moving from supine to sitting to standing positions. Thus different non-invasive and invasive indices (mid-frequency band power, plasma noradrenaline and adrenaline) supported the presence of reflex sympathetic (cardiovascular, sympathoneural, adrenomedullary) stimulation during active standing, whereas the high-frequency band power of HR reflected a posture-dependent cardiac vagal inhibition. The increase in high-frequency band fluctuations in SBP during active standing probably reflects non-neuronal processes, related to postural changes in the mechanical thoracic coupling between respiration and the vasculature; the respiration-induced fluctuations of HR appear to play only a minor role in the origin of these fluctuations in BP [13,30].

Although these postural effects are clear, a word of caution is required regarding an oversimplified interpretation of some parameters. The increase in plasma noradrenaline concentration during active standing is often used clinically to reflect reflex sympathoneural activation. However, Esler and co-workers (e.g. [31]) have shown that clearance of plasma noradrenaline decreases with upright posture (which may be due to a reduction in cardiac output and organ blood flows): the rise in plasma noradrenaline concentration therefore may be due to both a fall in plasma noradrenaline clearance and an increase in noradrenaline release. Our study neatly demonstrated a posture-dependent increase in plasma adrenaline concentration, reflecting adrenomedullary activation during standing, but it may also have been caused in part by reduced clearance. The extent to which this increase in adrenomedullary activity during active standing is related to the increase in plasma noradrenaline concentration is unclear. The increase in mid-frequency band power of HR and BP, as an indicator of increased cardiac sympathetic tone during active standing, has been documented in numerous studies (e.g. [27–29]), although it has been shown that age and changes in breathing pattern may greatly affect the mid-frequency power of HR and BP. In the present study with young healthy adults, we observed no significant posture-related changes in dominant respiratory frequency.

With the exception of the SBP and catecholaminergic parameters, all cardiovascular variables measured during supine, sitting and standing periods showed significant changes on repeated performance: the mean HR decreased, while HR variability indices, DBP and BRS increased. Thus, within a relatively short period of several hours, ‘habituation’ effects of the cardiovascular responses (particularly the HR parameters) to a physical task (active standing) were apparent. Plasma catechol-
amine concentrations proved to be reproducible on repeated performance. These data suggest that the (central and peripheral) interplay between sympathetic and vagal cardiovascular processes reflects habituation effects to the physical tasks and/or experimental procedures more accurately than do peripheral biochemical indices of sympathoneural or adrenomedullary activity during postural changes. However, it should be noted that the sequence of procedures used in our study (first supine, sitting, standing; then supine, standing, sitting) may have contributed in part to our findings.

**CWT response magnitudes**

When performed in the sitting position, the CWT induced the following significant effects: HR, BP, respiratory frequency and plasma adrenaline concentrations increased, whereas indices of cardiovascular variability decreased. We observed variable and non-significant results with regard to plasma noradrenaline concentrations and BRS. Regarding the haemodynamic and plasma catecholamine responses to mental challenge, our findings are in line with those of previous studies (e.g. [16,32–34]). Similarly, reduced HR and BP variability in the two frequency bands during a psychological task has been reported previously (e.g. [35,36]), although some studies reported task-induced increases in mid-frequency band power of HR (e.g. [37]). Apparently, mid-frequency band power of HR and BP during performance of a psychological task may not be stable indicators of cardiac sympathetic activity. Also, the direction of change in cardiovascular variability may be greatly affected by the specific requirements of the task involved. In combination with the increase in respiratory frequency, the reduction in variability indices of HR and BP during performance of the CWT resembles a pattern of vagal withdrawal, whereas the increases in BP and plasma adrenaline concentration indicate sympathoadrenomedullary activation. The variable data with regard to plasma noradrenaline concentration during the CWT can be explained by the fact that the antecubital venous noradrenaline concentration largely represents the venous drainage from skeletal muscles. Sympathetic nervous outflow to forearm muscle tends to show variable effects during mental tasks [33,34]; however, cardiac noradrenaline spillover can be increased during mental challenge [33].

When comparing the magnitude of responses to the CWT during sitting and standing, the following significant differences were observed: (1) mean HR increased during sitting, whereas it slightly decreased during standing, (2) high-frequency band fluctuations of HR decreased to a greater extent when the CWT was performed in the sitting position than when it was performed in the standing position, (3) high-frequency band fluctuations of SBP during the CWT decreased to a greater extent during standing than during sitting, and (4) plasma adrenaline responses to the CWT were larger during sitting than during standing, although the effect was small. With regard to the haemodynamic effects, our findings are similar to those of Sherwood and Turner [9]: whereas a CWT-induced increase in BP was observed during both sitting and standing, the mean HR increased only during sitting, suggesting different haemodynamic determinants of posture-related BP responses to mental tasks. When the CWT was performed during standing, plasma noradrenaline concentrations continued to show variable results. Plasma adrenaline concentrations appeared somewhat reduced in comparison with responses during sitting, indicating less adrenomedullary activation during CWT performance in the standing position. The index of BRS did not differentiate between sitting and standing in responsiveness to the CWT, showing that alterations in BRS during the CWT were not responsible for the lack of increase in HR during standing CWT performance. Since we observed less reduction of the high-frequency band power of HR and a slight decrease in mean HR during the CWT when it was performed standing, our data suggest reduced vagolytic responsiveness in comparison with performance of the task when sitting. High-frequency band power of SBP, primarily representing non-neuronal mechanisms related to the mechanical thoracic coupling between respiration and the vasculature [13,30], decreased to a greater extent when the CWT was performed in the standing position. Although we observed no significant changes in respiratory frequency, alterations in respiratory tidal volume, leading to changes in intrathoracic pressure and venous return, may have contributed to these effects. For most parameters, active standing induced significant effects; these differences in baseline during sitting and standing may have influenced responsiveness to the CWT.

In summary, in the present study we investigated the effects of body posture on various indices of sympathetic and parasympathetic autonomic regulation during performance of a challenging psychological task. By combining different indices of sympathetic and parasympathetic autonomic regulation, some of the complex autonomic adjustments that occur during the psychological and behavioural challenges of normal daily life were evaluated. Overall, our data, obtained in a controlled laboratory setting, indicated posture-related differential effects on indices of sympathoadrenomedullary activation during performance of a psychological challenge, whereas indices of parasympathetic activity indicated primarily less vagolytic effect when the task was performed in the standing position. Habituation to the test and to experimental procedures may have been partly responsible for the low reproducibility of some of the cardiovascular parameters. Generalization of our laboratory findings to real-life situations could benefit greatly from studies with larger numbers of subjects in
which similar parameters are explored during challenging situations in an ambulatory setting, whereby control for posture-related effects are essential for a reliable interpretation of stress-related autonomic nervous system activity. In this respect, recent developments regarding ambulatory activity monitoring with body-mounted accelerometers [38] may offer new possibilities for objectively studying stress-related cardiovascular activity in the behavioural context.

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