

## Chapter 20

**Cardiovascular Reactivity to Work Stress Assessed by Ambulatory Blood Pressure, Heart Rate, and Heart Rate Variability**

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**Introduction**

Prospective studies have shown that high work stress is associated with increased risk for cardiovascular disease (Bosma, Peter, Siegrist & Marmot, 1998; Lynch, Krause, Kaplan, Salonen & Salonen, 1997; Siegrist, Peter, Junge, Cremer & Seidel, 1990). Despite the epidemiological evidence, the pathophysiological pathways underlying this relationship are still unclear. Exaggerated autonomic nervous system reactivity to work stress has been proposed as a possible etiological mechanism (Everson et al., 1997). Subjects who experience their work as stressful will be repeatedly exposed to protracted periods of emotional upheaval with autonomic activation. If this is accompanied by prolonged increases in heart rate (HR) and blood pressure (BP) this may cause target organ damage (Georgiades, Lemne, De Faire, Lindvall & Fredrikson, 1997) and increase the risk for atherosclerosis (Lynch, Everson, Kaplan, Salonen & Salonen, 1998). Ambulatory monitoring gives us the opportunity to test this hypothesis in a real life work setting. In fact, ambulatory monitoring may be the only alternative to study work stress in an ecologically valid way, because it is difficult or even impossible to envisage a laboratory test battery that could simulate the complex psychological factors involved in the work environment.

This chapter reports on the Amsterdam Work Stress Study where we used 24-hour registrations of continuous HR and intermittent BP on workdays and one non-workday in male white-collar workers and female nurses to investigate the relation between subjective work stress and cardiovascular activation during the workday. Using the data from this study, three main questions will be addressed. First, we tested the validity of the Root Mean Square of Successive Differences (RMSSD) as an index of ambulatory changes in respiratory sinus arrhythmia (RSA), by comparing it to the two "classical" indices of RSA, high frequency band (0.15–0.40 Hz) spectral power and peak-to-through RSA from the combined respiration signal and the Inter Beat Interval (IBI) time series. Next test-retest reliability of three indices

of cardiovascular reactivity to a work setting were assessed. Reactivity was defined as either work period versus sleep period on the workday, work period versus leisure period on the workday or workday versus non-workday differences. Finally, in the male white-collar workers the effects of chronic work stress will be examined on reactivity of ambulatory BP, HR, and RMSSD. Increasing evidence shows that work stress effects could be more pronounced during the process of unwinding after work than during the actual work time (Suadicani, Hein & Gyntelberg, 1993; Siegrist, Peter, Junge, Cremer & Seidel (1990). Therefore, special attention will be paid to the processes of recovery during the evening and sleep.

## The Amsterdam Work Stress Study

The Amsterdam Work Stress Study examines the relation between work stress and risk factors for cardiovascular disease in two different populations. One population consists of young female nurses ( $33.7 \pm 8.1$  y) working at three hospitals in Amsterdam (Riese, van Doornen, Houtman & de Geus, 2000) and the other of middle aged ( $47.2 \pm 5.3$  y) male white-collar workers, all working at the same large computer company (Vrijkotte, van Doornen & de Geus, 2000). None of the subjects received treatment for hypertension, hyperlipidemia or diabetes mellitus, and all subjects were free of overt cardiovascular disease.

### Ambulatory registration

Male participants underwent ambulatory monitoring on two workdays, Monday and Thursday, and one non workday (Saturday or Sunday) of the same workweek and always in that order. Female participants were monitored on a workday (third morning shift 7:30 am – 4:15 pm) and one non workday. BP was recorded by a Spacelabs 90207 device at 30-minute interval only during waking hours. HR and RMSSD were measured continuously for a 24-hour period by the VU-AMS (version 4.6, TD-FPP, Vrije Universiteit, Amsterdam). The VU-AMS has 2 channels for ECG and ICG registration. The memory capacity is 2 MB. The ECG-signal, derived from three electrodes, is led into a differential amplifier, with an input impedance higher than 1 MW, and a CMRR of 70 dB. The amplified ECG is passed through a bandpass filter of 17 Hz. The R-top is recognized with a level detector with automatic level adjustment. The output of the level detector is connected to an interrupted request line of the microprocessor. At each R-top, a millisecond counter is read and reset, yielding the raw inter-beat-interval (IBI). From the raw IBI's VU-AMS updates a running average of HR and RMSSD. To get an impression of the physical activity during the registration, the VU-AMS also monitors the amount of body movement (motility) of the subject by an active acceleration sensor. Because the device is always placed on the hip this indexes gross body movements, for instance, transitions in posture and activity. Motility has a range of 0 to 3.2 gsec with a resolution of 0.008 gsec. More information about the VU-AMS can be found on the internet page: [www.psy.vu.nl/vu-ams](http://www.psy.vu.nl/vu-ams). Reliability and validity aspects and recording method-

ology of the VU-AMS have been described previously (de Geus & van Doornen, 1996).

Average HR, RMSSD and motility over 30-second periods were stored throughout the 24-hour recording time. Approximately every 30 minutes (10 minutes randomized) the participants were prompted by an audible alarm from the VU-AMS, to fill in a diary. They were instructed to write down time, activities, body postures in chronological order and also consumed cups of coffee, glasses of alcohol and smoked cigarettes. Posture and activity information from the diary were combined with the motility signal using an interactive graphical program and stationary fragments (same posture, same activity) were coded for posture, activity, day and time of the day. Mean values for HR and RMSSD for these coded fragments were calculated by the program and stored simultaneously with start and end time and duration of the period. Each BP value from the Spacelabs device was similarly coded.

### **Comparison of RMSSD with HF and RSA**

Although HR is sometimes measured intermittently, for instance when derived from ambulatory BP monitors, many studies have measured continuous HR. This allows for the additional computation of heart rate variability in the respiratory range (RSA) that is widely regarded to index vagal influences on the myocardium (Hayano et al., 1991). The Framingham Heart Study (Sing et al., 1998) has identified decreased vagal tone as a determinant of the progression of hypertension. Apart from its effects on BP, low cardiac vagal tone may predispose to myocardial infarction and sudden death by compromising the electrical stability of the heart and by increasing HR, which is a risk factor for cardiac disease independent of BP (Palatini & Julius, 1997). Not surprisingly, low vagal tone has been found to be associated with higher risk for cardiac disease (Liao et al., 1997; Tsuji et al., 1996).

In laboratory settings, RSA is either derived by peak-valley estimation (Katona & Jih, 1975) that uses the time series of IBIs in combination with the respiration signal, or by various quantification techniques that use only the IBI time series, like spectral analysis (Akselrod, Gordon, Madwed, Snidman, Shannon & Cohen, 1985), time domain filtering (Porges & Bohrer, 1980) or Wavelet time-frequency analyses (Houtveen & Molenaar, 2000). Although some disagreement has arisen over which method "best" indexes RSA (Byrne & Porges, 1993) these measures have in fact shown excellent correspondence in various resting and task conditions (Grossman, van Beek & Wientjes, 1990; Hayano et al., 1991; Houtveen & Molenaar, 2000).

Ambulatory recording equipment is available that allows computation of all these time-domain and frequency based RSA measures. However, the practical value of these measures in ambulatory research is quite restricted. Valid use of spectral powers, for instance, depends on thorough inspection of each 2 to 3-minute IBI series that are typically used as input. A single missed beat can seriously distort the spectrum (Hilton, Ryan & Beattie, 1997). Reliable peak to trough assessment of RSA requires visual inspection of the combined respiration signal with the IBI time series to remove breathing artefacts (de Geus et al., 1995; Grossman et al., 1990).

Therefore, current methods available to extract RSA from continuous ambulatory HR data are very time consuming and almost unfeasible in epidemiology-scaled ambulatory studies in which large amounts of data are collected. A solution would be to use simple time domain measures like the RMSSD as a proxy for vagal tone. Below we test the validity of this approach by comparing ambulatory RMSSD to spectral high frequency power (HF) and peak-to-through RSA from the combined respiration signal and IBI time series.

Twelve women (mean age  $37.5 \pm 4.5$ ) were selected from the female population. Ambulatory registration of HR for 24 hour was performed by the VU-AMS on a workday and a non-workday. Every 30 ( $\pm 10$ ) minutes a beat to beat registration of 5 minutes was registered in which IBIs of consecutive R-peaks were recorded. By means of diary information and a motility signal, the 5 minutes periods were coded for posture (lying, sitting, standing, physical active).

For each of these 5 minutes periods we determined the RMSSD, the peak-to-through RSA and LF, HF and total spectral power. Full description of the methods to derive these measures are described in de Grossman, van Beek & Wientjes (1990) and Mulder (1988). Briefly, peak-to-through RSA combines the respiratory time intervals and the IBIs to obtain the shortest IBI during heart rate acceleration in the inspirational phase (which was made to included 750 milliseconds from the following expiration to account for phase shifts) and the longest IBI during deceleration in the expirational phase (including 750 milliseconds from the following expiratory pause/inspirational phase). The difference between the longest and shortest interval is used as an index of RSA. When no phase-related acceleration or deceleration was found, the breath was assigned a RSA score of zero. Frequency domain analysis of the interbeat intervals was done with the CARSPAN program that is based on sparse Discrete Fourier Transformation and that yields a power-frequency spectrum from 0.01 to 0.50 Hz. The spectrum is based on a series of equidistant samples representing HR. Three frequency bands were deemed of interest: the total power (TF), the power in the frequency band around the intrinsic blood pressure oscillations (0.07–0.14 Hz: MF) and the high frequency band that reflects vagal influence only (0.15–0.40 Hz: HF)

Table 1 shows the Pearson's correlation coefficients between the different indices of vagal tone within each subject with all values, regardless of posture, included. Within-subjects correlation between RMSSD and HF varied between .58 and .92 with median .82. Within subjects correlation between RMSSD and RSA varied between .39 and .89 with median .74. For HF and RMSSD between subject correlations were also calculated, and this was done separately for each posture. High between subject correlation of HF with RMSSD was found throughout: during sitting .91, during physical activity .71., and during sleep .73.

From the male population, a random subset of the 34 participants were selected for detailed RSA analysis. Twenty four hour continuous IBI time series were available on two workdays (Monday and Thursday) and a non-workday. Five minutes epochs from the morning, afternoon, evening and night periods were selected.

Table 1. Within-subject correlations

Subject	RMSSD HF Power	RMSSD MF Power	RMSSD TF Power	RMSSD RSA	RSA HF Power
1	0.71	0.67	0.62	0.74	0.92
2	0.92	0.35	0.60	0.89	0.88
3	0.58	0.51	0.55	0.39	0.62
4	0.75	0.42	0.55	0.79	0.94
5	0.82	0.51	0.78	0.86	0.92
6	0.83	0.38	0.61	0.82	0.86
7	0.83	0.38	0.67	0.74	0.90
8	0.81	0.29	0.50	0.44	0.50
9	0.64	0.19	0.32	0.66	0.72
10	0.70	0.33	0.38	0.67	0.74
11	0.91	0.49	0.72	0.69	0.70
12	0.91	0.16	0.43	0.86	0.87
Median	0.82	0.38	0.58	0.74	0.87

Diary and motility signal were used to assure that the subjects were sitting (lying during sleep). Figure 1 shows the between subject relationship between ambulatory RMSSD and HF as a function of period of the day. Correlations between RMSSD and HF were all highly significant and varied between .54 (Monday morning) and .95 (Non workday sleep) with median value of .89. On all three days, the correlations were lowest during the morning and highest during the night.

*From these two studies we conclude that RMSSD is an efficient alternative to RSA or HF to index vagal tone in ambulatory studies.*

### Reliability of HR, RMSSD and BP reactivity

Ambulatory monitoring studies have concentrated on HR and BP levels, because of the predictive power of these variables for CVD risk (Palatini & Julius, 1997; Kamarck et al., 1998; Mallion, Baguet, Siche, Tremel & De Gaudemaris, 1999). The quantification of BP, HR and their reactivity measures in an ambulatory setting is not as straightforward as in laboratory experiments. There is limited control over known sources of variation in cardiovascular activity during the day other than psychosocial demands like physical activity, posture, food intake, time of day, ambient temperature. Sophisticated statistical analysis have recently been applied to partial out the variance associated with these factors (Fahrenberg & Myrtek, 1996; Kamarck et al., 1998). Fortunately, previous studies have shown that most of the variance due to physical activity in ambulatory signals can be captured largely by looking at posture (Myrtek, Fichtler, Strittmatter & Brugner, 1999; Pieper, Warren & Pickering, 1993; Steptoe, Roy & Evans, 1996). The use of diaries and/or accelerometers are essential in retrieving this information so controlling for influence of physical activity is possible. After controlling for these confounding factors,

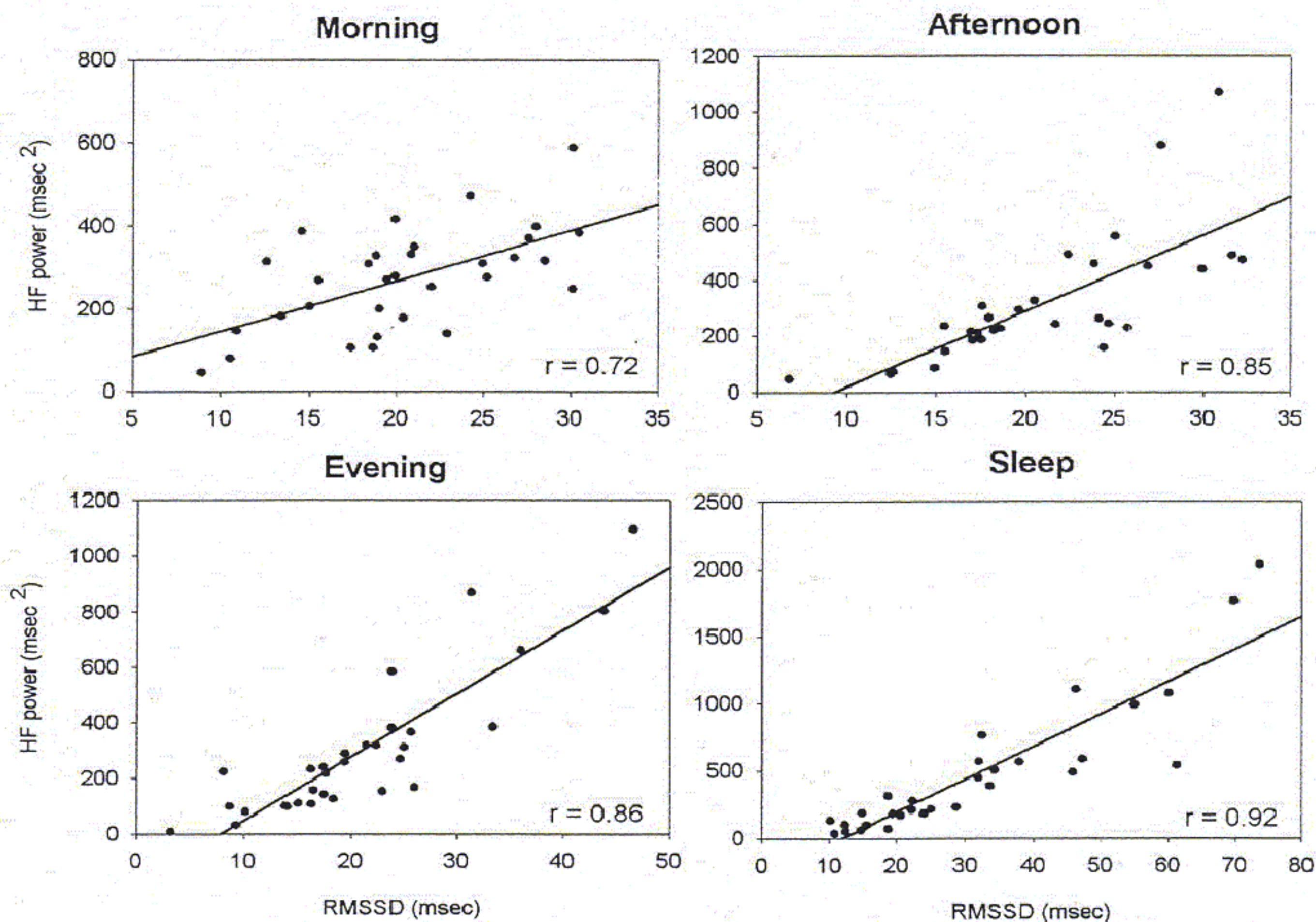


Figure 1. Correlation between two indices of vagal tone, Root Mean Square of Successive Differences (RMSSD) and high frequency (HF) power as a function of period of the day.

indices of real life stress reactivity, like work-sleep or work-leisure or workday-non workday differences will have high ecological validity. However, surprisingly little information is available on the temporal stability of these posture/activity controlled indices of real life stress reactivity.

There is an extensive literature exploring the temporal stability of cardiovascular reactivity in the laboratory (Manuck, Kamarck, Kasprovicz & Waldstein, 1993). Test-retest reliability of cardiovascular reactivity to laboratory stressors is low to moderate, with correlations on average in the .40 to .50 range. As Manuck (1994) stated in his review, reactivity is a more generalized propensity to respond to behavioral stimuli and therefore reactivity should be determined by aggregating subjects' responses over several tasks and several sessions. With this in mind, one would predict high temporal stability of ambulatory reactivity indices like the work period minus leisure period or work period minus sleep period, provided measurements were taken on comparable work-days. In principle, the difference between the average HR level at work and that during sleep can be regarded as the aggregation of the responses to many repeated stressors during the workday.

Ambulatory registrations from the sedentary male populations were used to test this hypothesis. Data from two workdays of the same work week, Monday and Thursday, were compared for the 109 male subjects to assess temporal stability. Mean values of HR, RMSSD and BP for the different periods work, leisure and

sleep (the latter only for HR and RMSSD) were determined as well as three reactivity scores: (1) work period minus leisure period (same day), (2) work period minus sleep period (same day), and (3) work period on a workday minus leisure period on a non workday.

The total amount of minutes spent in the activities other than sitting were comparable on Monday and Thursday (see Figure 3). Subjective report of mood, as assessed by the Profile Of Mood States (POMS) (Wald & Mellenbergh, 1990) showed no differences between the workdays in either positive or negative mood. Moreover, test-retest correlations were .58 for positive and .64 for negative mood. Thus, the assumption seems tenable that these workdays yielded comparable levels of work stress both at a group and at an individual level.

Figure 2 shows the relationship between Monday and Thursday for the four ambulatory cardiovascular variables obtained during work period. It is clear from this figure that the correlation between the two workdays is high for all four variables. Table 2 gives the corresponding correlation coefficients, also during leisure period and sleep period, and the reactivity indices. According to Bland and Altman (1986), high correlation coefficients do not always indicate a high repeatability. They recommend coefficient of repeatability ( $C_{rep}$ ), which describes the range of within

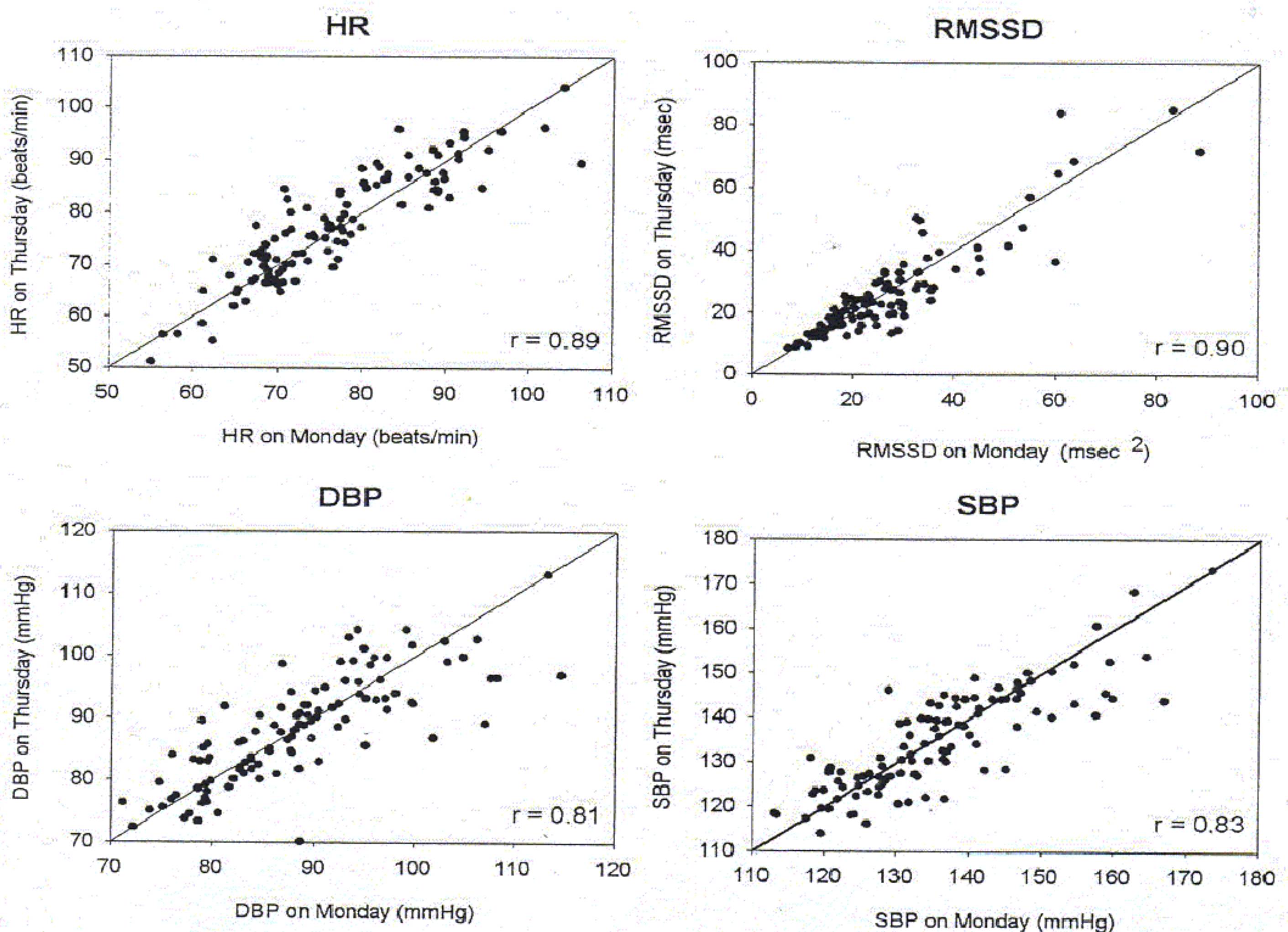


Figure 2. Monday to Thursday test-retest correlation for HR, RMSSD, DBP, and SBP during the work period.

Table 2. Pearson correlation coefficient ( $r$ ) and coefficient of repeatability ( $C_{rep}$ ) between Monday and Thursday as a function of period of the day. Mean values are the means of Monday and Thursday.

	All values included			Only sitting values included		
	Mean	$r$	$C_{rep}$	Mean	$r$	$C_{rep}$
<b>HR (beats/minute)</b>						
work period	79.5 ± 10.1	.88	10.2	77.1 ± 9.9	.89	9.5
leisure period	79.1 ± 9.4	.62	18.5	75.3 ± 9.2	.79	12.6
sleep period	63.8 ± 7.6	.83	9.2	63.8 ± 7.6	.83	9.2
reactivity (work-sleep)	15.6 ± 7.9	.75	11.9	13.3 ± 7.7	.76	11.1
reactivity (work-leisure)	1.8 ± 5.5	.59	11.2	0.65 ± 6.6	.50	15.5
reactivity (workday-non workday)	-0.6 ± 9.0	.85	10.3	1.5 ± 7.6	.83	9.5
<b>RMSSD (msec)</b>						
work period	24.2 ± 9.4	.75	14.6	24.3 ± 9.8	.85	14.4
leisure period	24.8 ± 9.4	.75	14.6	25.0 ± 10.6	.79	11.2
sleep period	31.1 ± 15.1	.81	20.6	32.9 ± 15.1	.81	20.5
reactivity (work-sleep)	10.4 ± 15.9	.72	23.8	9.6 ± 16.0	.71	24.4
reactivity (work-leisure)	0.3 ± 6.5	.20	21.2	0.5 ± 5.4	.28	16.4
reactivity (workday-non workday)	-0.002 ± 8.0	.80	15.7	-.008 ± 9.2	.79	16.5
<b>DBP (mmHg)</b>						
work period	88.3 ± 8.6	.83	10.4	87.9 ± 8.6	.81	11.2
leisure period	85.2 ± 9.0	.84	10.7	84.5 ± 9.1	.83	11.2
reactivity (work-leisure)	3.5 ± 6.5	.48	12.9	3.1 ± 4.3	.36	11.9
reactivity (workday-non workday)	5.3 ± 5.8	.62	10.4	5.7 ± 6.7	.68	11.3
<b>SBP (mmHg)</b>						
work period	135.7 ± 11.4	.83	14.1	134.6 ± 11.6	.83	14.2
leisure period	133.9 ± 11.7	.82	14.8	132.4 ± 11.5	.82	14.4
reactivity (work-leisure)	2.2 ± 6.5	.50	15.1	1.9 ± 5.2	.30	15.2
reactivity (workday-non workday)	5.0 ± 6.5	.60	14.1	5.3 ± 7.4	.63	14.2

which 95% of repeated measurements will fall. The coefficient is calculated as 2 x standard deviation of the differences. A wide range relative to the mean indicates low repeatability. The  $C_{rep}$  are the lowest for HR and DBP, followed by SBP and RMSSD. Repeatability as a function of posture is described in Table 3. As expected the repeatability was highest during sitting and decreased during physical activity. Based on these findings we expected the repeatability to be higher when only sitting values were included, because it would reduce variance due to differences in physical activity. This was not the case, however, repeatability was the same as when periods with physical activity were included. Probably this reflects the finding that the activity patterns were on average the same on the two workdays (Figure 3). Also we selected a sedentary population: subjects were sitting almost 70% of their time during the work period. Nonetheless, in populations



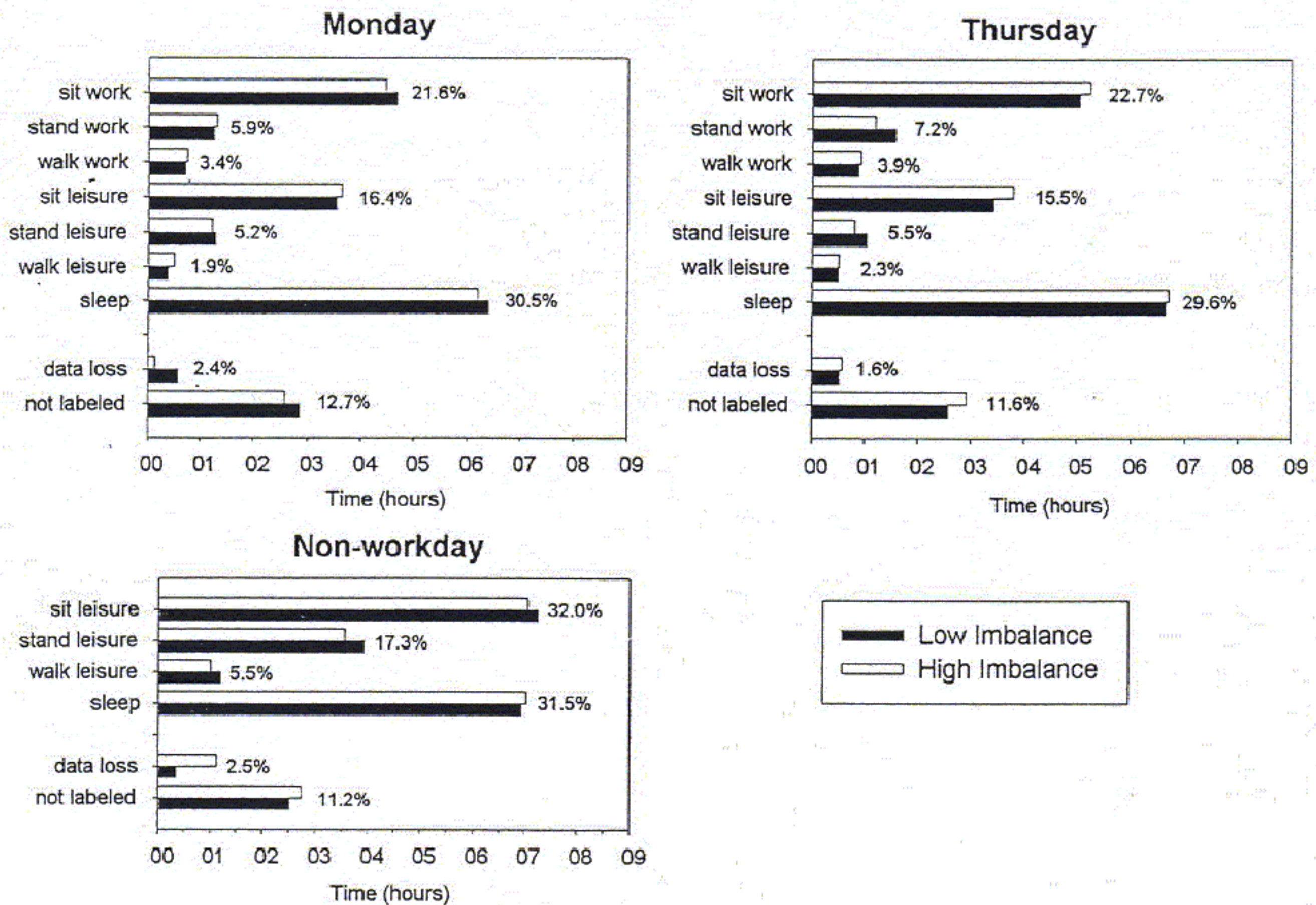


Figure 3. Amount of time spent in different postures during two workdays and one non-workday as a function of effort-reward imbalance.

that are more physically active, like nurses, it could be still be meaningful to select only sitting periods.

The correlation coefficients of the reactivity measures are not as high as the absolute levels but are all highly significant ( $p < .001$ , except for RMSSD:  $p = .05$ ). The  $C_{rep}$  are relatively high compared to the mean reactivity scores, indicating low repeatability. At first glance, the reactivity index obtained by subtracting the non-workday from the workday levels seems the best choice in terms of repeatability. However, note that the reactivity values for Monday and Thursday use the same non-workday baseline. For a more fair comparison to the other two reactivity indices we should have repeated the non-workday measurement twice, taking one as baseline for Monday and the other as a baseline for Thursday. In fact, because mean reactivity scores are clustered around zero for  $HR_{(work-leisure)}$ ,  $HR_{(workday-non-workday)}$ ,  $RMSSD_{(work-leisure)}$  and  $RMSSD_{(workday-non-workday)}$  their repeatability will not be very good.

In conclusion, the repeatability of absolute HR, RMSSD, SBP and DBP in this sedentary white-collar population is high when mean values over relatively long periods, like work or leisure or sleep period, are compared. Repeatability of reactivity scores is low, but test-retest reliability still compares favourable to test-retest reliability of the reactivity of these variables to laboratory stressors. The most optimal reactivity scores are those that contrast day time levels to baseline values at sleep; these yield non-zero reactivity scores for most of the subjects and have the highest repeatability.

Table 3. Pearson correlation coefficient and coefficient of repeatability ( $C_{rep}$ ) between Monday and Thursday as a function of posture.

	Mean	r	$C_{rep}$
<b>HR (beats/minute)</b>			
sitting	76.4 ± 9.1	.87	9.7
standing	83.9 ± 9.9	.71	13.7
physical active	90.7 ± 10.1	.66	17.7
<b>RMSSD (msec)</b>			
sitting	25.3 ± 11.8	.83	13.8
standing	26.1 ± 12.6	.84	23.5
physical active	23.3 ± 10.6	.77	17.1
<b>DBP (mmHg)</b>			
sitting	86.4 ± 8.5	.85	9.6
standing	88.2 ± 9.4	.76	12.6
physical active	89.0 ± 9.5	.74	15.2
<b>SBP (mmHg)</b>			
sitting	133.8 ± 11.5	.87	10.8
standing	137.0 ± 12.7	.73	16.9
physical active	139.7 ± 14.0	.73	21.4

## Work stress effects on cardiovascular reactivity

### Work stress assessment

Although ambulatory monitoring has intrinsic ecological validity, to be useful in stress research it must also have content validity in that it discriminates between subjects who experience their work as high or low stressful. Previous analyses of our male white-collar workers suggest that this is true at least for some aspect of work stress (Vrijkotte, van Doornen & de Geus, 1999; Vrijkotte, van Doornen & de Geus, 2000). The remainder of this chapter will summarize and extend these findings to demonstrate the power of ambulatory recording for psychosocial effects on cardiovascular regulation.

The Effort-Reward Imbalance questionnaire, based on Siegrist's model (Siegrist & Peter, 1994), was used to measure perceived chronic work stress. This model defines chronically stressful experiences at work in terms of a mismatch between high effort spent and low reward received in occupational life. This mismatch is expressed by two summary measures of work stress: imbalance and overcommitment. Of 109 subjects, 23 (21%) experienced a mismatch between extrinsic effort (demanding aspects of the work environment) and reward (esteem reward, monetary gratification and status control) and were classified as high imbalance. Overcommitment states an individual's style of coping with work demands. This

component is by itself a mismatch score because those individuals who score high tend to spend an inadequate amount of effort that is not met by the externally defined rewards. Overcommitment was dichotomized so that subjects in the upper tertile were considered high in overcommitment. The groups did not differ in possible confounding factors like: age, body mass index, consumed cups of coffee and glasses of alcohol, smoking, years of services, habitual physical activity. Support for the use of this work stress model comes from the Kuopio Ischaemic Heart Disease Risk Factor Study in which men with high stress induced blood pressure reactivity who also experienced high work demands (Everson et al., 1997) or had low socioeconomic status (Lynch, Krause, Kaplan, Salonen & Salonen, 1997) had the greatest atherosclerotic progression in four years.

### Effect of posture

As we mentioned before, posture is the most important confounding factor and our results confirmed this. Ambulatory HR and BP levels increased from lying (only HR) to sitting to standing to walking ( $p < .001$ ) (Table 3). RMSSD also showed a main effect of posture ( $p < .001$ ), but this was completely due to a higher RMSSD during lying (sleep) compared to sitting, standing, and walking (Table 3). This means that any ambulatory cardiovascular differences between the work stress groups could reflect different activity patterns during the ambulatory monitoring days. However, a comparison of the time (number of minutes) spent in different postures showed that the work stress groups did not differ in the total duration of sitting, standing, physical active, and lying (only during sleep) during the various periods of the three days (Figure 3). In fact, statistical analyses on (1) values during sitting-lying only and (2) all values, i.e., regardless of posture, gave essentially the same results. Only the latter values will be presented.

### Effect of work stress

On the workdays, DBP and SBP were significantly higher (3.1 and 1.8 mmHg, respectively) during work compared to leisure period (Figure 4). This work-leisure reactivity was, however, independent of work stress. Workday versus non-workday showed that DBP and SBP were lower during the non-workday, even during leisure period on the workdays. On average these DBP/SBP differences were 4.2/3.6 mmHg. And again, this was not different for the high and low imbalance groups.

Within each workday no significant differences between HR and RMSSD during leisure and work periods were found, suggesting that HR and RMSSD reactivity to work was protracted in leisure time. On all days sleep HR was significantly lower and sleep RMSSD significantly higher than the levels during the day. Average reactivity of HR and RMSSD derived by subtracting the non workday from the workday level was very low. Again, for HR and RMSSD, reactivity computed as work minus sleep appears most useful.

The RMSSD<sub>(work-sleep)</sub> reactivity was not different between the imbalance groups but the high imbalance group had a higher HR<sub>work-sleep</sub> reactivity compared to the

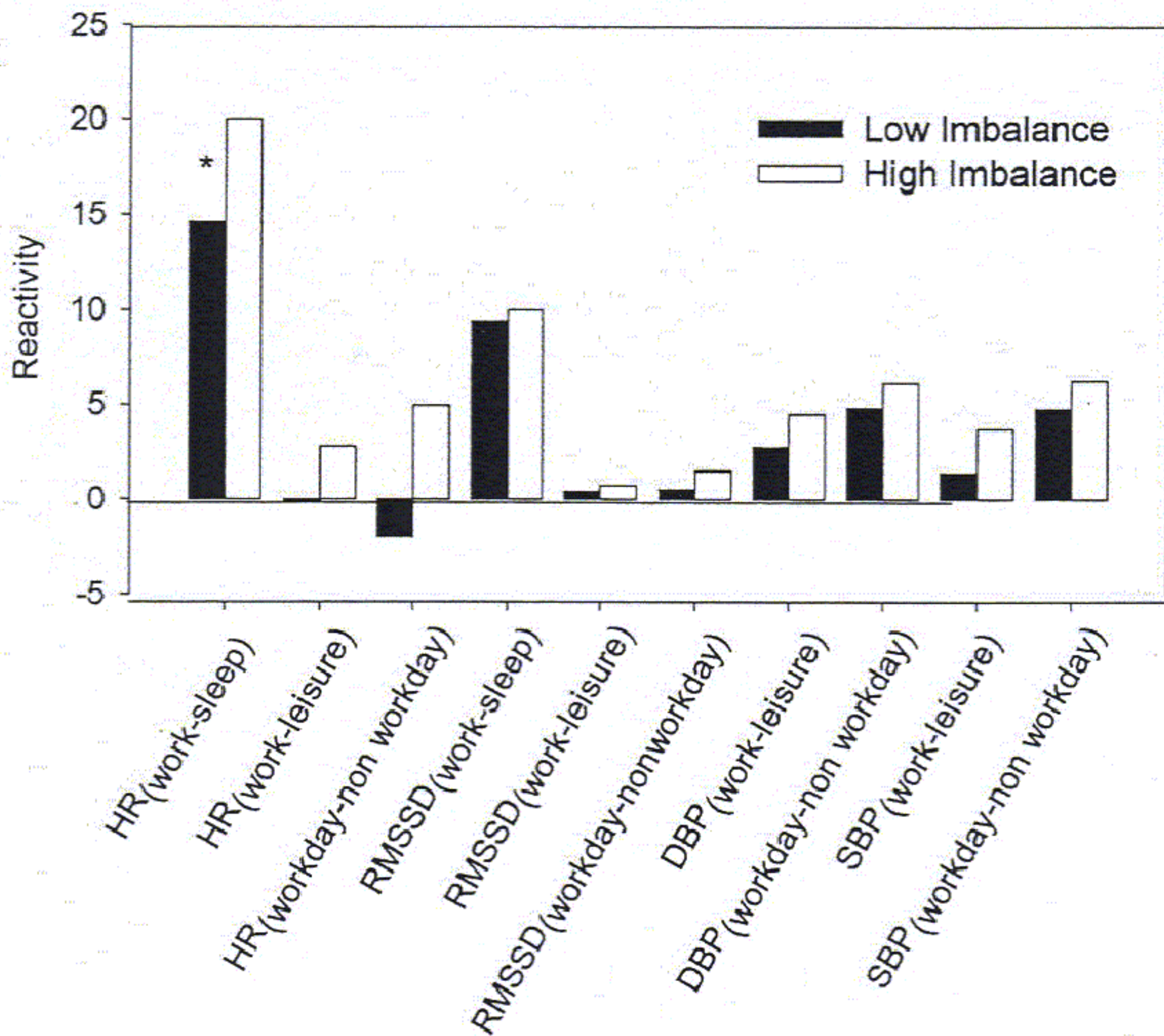


Figure 4. Cardiovascular reactivity to the work period as a function of effort-reward imbalance. Mean reactivity over the two workdays is shown. HR difference in beats/minute, RMSSD difference in msec, BP difference in mmHg. \* Significant ( $p < .05$ )

low imbalance group. This difference was on average 6.8 bpm. Higher HR reactivity of this high work stress group remained evident throughout the leisure time period.

Neither overcommitment, the other aspect of work stress in the Siegrist model, nor the interaction of overcommitment with imbalance, had an effect on SBP, DBP, HR and RMSSD reactivity.

### Recovery from work stress

A number of recent findings suggests that slow or incomplete recovery from stress may be as important to disease risk as reactivity during stress (Siegrist, Peter, Junge, Cremer & Seidel, 1990; Suadicani, Hein & Gyntelberg, 1993). McEwen used the term 'allostatic load' to refer to the chronic overactivity of stress-related physiological systems, and has argued that chronic allostatic load is frequently manifest as failure to recover following termination of demands (McEwen, 1998). Ambulatory recording provides an excellent setting to test slow recovery processes during sleep after the workday. Our previous analyses showed that HR not only increased more during the workday in the high imbalance group, but also stayed higher during leisure time after work. Overall sleep levels, however, were again comparable. This

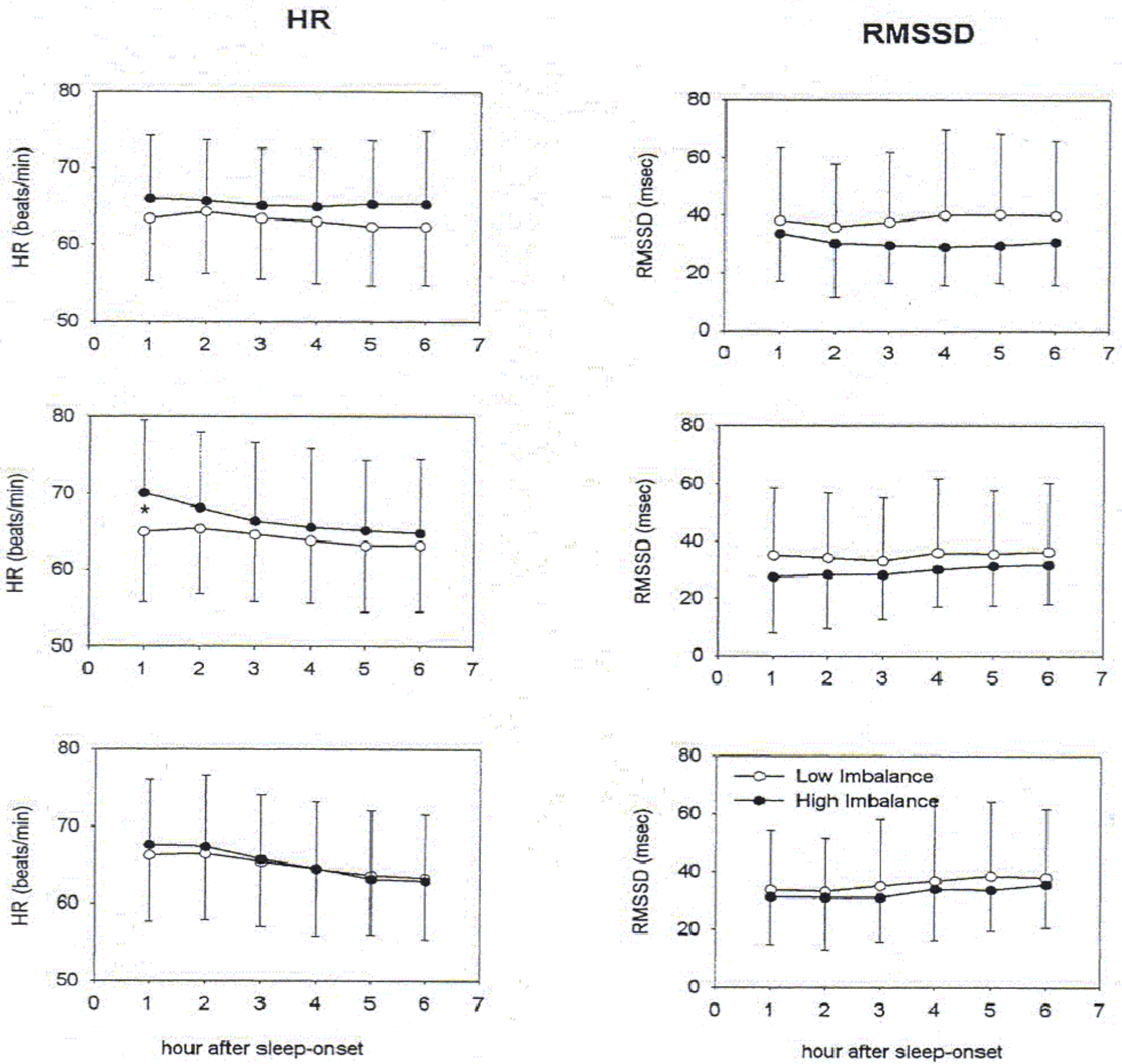


Figure 5: HR and RMSSD (mean and SD) during sleep on Thursday for the high and low imbalance group. \* Significant ( $p < .05$ )

work stress effects disappear gradually in the course of the night. To test this idea, one hour mean values for HR and RMSSD after time to bed were calculated and compared between the work stress groups. Because overcommitment is characterized by worrying and the inability to withdraw from work obligations, we further expected this scale to show the largest sensitivity to individual differences in recovery.

The results, first of all, showed that alcohol and coffee consumption during the day affected HR and RMSSD during the first three hours of sleep. After adjusting for these confounders, HR was higher during the first hour of sleep in the high imbalance group, although significant only on Thursday (Figure 5). RMSSD was not significant different between the imbalance groups. Most surprisingly, overcommitment did not show any effect on HR and RMSSD.

Our results provide only partial support for the importance of compromised recovery to explain the effects of work stress on cardiovascular health. With longer exposure to work stress, however, the high HR found during work, leisure and the first hours of sleep may extend to the whole sleep and possibly non workdays. At some point the overall level of HR may increase to a point where HR dynamics are reduced and work stress is associated with decreased rather than increased HR response to work. This gradual change from HR reactivity to tonic increases in HR with loss of HR dynamics was already proposed by Siegrist (two-stage model of cardiovascular reactivity), with a putative down-regulation of cardiac beta-receptors as an underlying mechanisms (Siegrist & Klein, 1990). Indeed, down-regulation of beta-receptors has been linked previously to chronic stress (Dimsdale, Mills, Patterson, Ziegler & Dillon, 1994) and to diminished HR responses to stress (Eisenhofer, Lambie & Johnson, 1985; Mills, Dimsdale, Ziegler, Berry & Bain, 1990).

## Conclusion

In conclusion, 24 hour registration of HR, RMSSD and BP produces reliable and valid information about individual differences in physiological responses to work stress. Also detailed analyses of cardiovascular recovery processes can be made. RMSSD appears to be an efficient index of RSA to index vagal tone in large scale ambulatory studies. Posture and activity are important confounding factors in ambulatory cardiovascular registrations and on-line analysis of posture and motion, described in chapter 13, 14, and 15, would facilitate data analysis and would produce more accurate posture and activity related data. The test-retest reliability of absolute HR, RMSSD and BP during various periods of the day is high. The test-retest reliability of often used ambulatory reactivity measures is lower, but higher than most reliability scores found in the laboratory. The highest and most reliable reactivity scores are obtained by taking mean values during sleep as a baseline. Finally, ambulatory HR reactivity discriminates between high and low chronic work stress. The higher HR found in the high imbalance group during work and leisure period and during the first hours of sleep clearly shows that "real life" work stress has "real life" physiological consequences.

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