Short-term Variability of Blood Pressure and Heart Rate in Borderline and Mildly Hypertensive Subjects

Reijo Takalo, Ilkka Korhonen, Väinö Turjanmaa, Silja Majahalme, Martti Tuomisto, Arto Uusitalo

Abstract Electrocardiogram and intra-arterial blood pressure were recorded in 96 men (aged 35 to 45 years) by the Oxford method over a 30-hour period. The study involved 33 normotensive, 29 borderline hypertensive, and 34 mildly hypertensive individuals, as assessed by the cuff method. Five-minute periods during sleep and with subjects in supine, sitting, and standing positions were extracted from the recordings for frequency domain analysis. Power spectrum density estimates of systolic blood pressure, diastolic blood pressure, and heart rate were calculated by an autoregressive method over the bandwidths of 0.02 to 0.075 (low-frequency), 0.075 to 0.15 (mid-frequency), and 0.15 to 0.35 Hz (high-frequency), attributable to thermoregulatory, baroreceptor, and respiratory activity. No significant intergroup differences were observed at nighttime, but in different body positions the borderline hypertensive subjects frequently had either greater low-frequency variability or smaller mid-frequency variability than the other groups. In this respect, the power spectra for systolic and diastolic blood pressures provided better statistical differentiation between the groups than those for heart rate. Furthermore, the borderline hypertensive subjects exhibited attenuated night-day changes in the low-frequency band for all time series. The results suggest that in borderline hypertension the baroreceptor oscillations are shifted to lower frequencies, presumably reflecting altered function of the sympathetic nervous system. In conclusion, spectral analysis of blood pressure variability for controlled test situations made it possible to detect differences in the cardiovascular regulatory systems between normotensive, borderline hypertensive, and mildly hypertensive individuals. (Hypertension. 1994;23:18-24.)

Key Words • blood pressure • hypertension, borderline • heart rate • spectrum analysis

By means of spectral analysis, short-term (time scale of seconds to minutes) variability in blood pressure (BP) and heart rate (HR) tracings can be divided into oscillatory components arising from various sources: one is linked to respiration (at approximately 0.25 Hz), another is assumed to be caused by baroreceptor activity (at approximately 0.1 Hz), and the third is believed to originate in the system responsible for regulating body temperature (in the range of 0.08 to 0.04 Hz). The slowest component also has been held to be due to local adjustments of resistance in individual vascular beds matching blood flow to local metabolic demand. It is not known exactly how these fluctuations enter into the cardiovascular control loops, but they are generally considered to be mediated principally by the autonomic nervous system. Respiratory coupled variability, especially in HR, is a marker of vagal activity, whereas the lower frequencies are jointly mediated by parasympathetic and sympathetic outflows. Accordingly, spectral analysis is a method that can provide important information about autonomic nervous control of the cardiovascular system in various pathophysiological conditions such as arterial hypertension.

We undertook the present study to establish whether short-term variability of BP and HR in subjects with slightly elevated BP differs from that in normotensive subjects. Hypertensive individuals may have abnormalities in cardiovascular control systems, and their identification may lead to ways of predicting individuals at increased risk of cardiovascular morbidity. Parameters of prognostic relevance are especially important in the diagnostic phase of arterial hypertension, because most subjects with borderline hypertension will not progress to permanent, established hypertension and are not likely to benefit from antihypertensive drug treatment. Spectral analysis of HR variability has suggested enhanced sympathetic and depressed vagal activity in mildly hypertensive individuals, but the differences in power spectra compared with normotensive subjects appeared to be so small that a search for characteristic values for each individual to be able to classify them as having a disease or no disease is unrealistic. Relatively little work has been done on spectral analysis of BP, and so far no differences between spectral estimates of normotensive and hypertensive subjects have been observed. In principle, however, the beat-to-beat analysis of BP variability could be a more sensitive method than that of HR in detecting the overfunction of the sympathetic nervous system, whereas HR, which is presumably influenced more by parasympathetic activity, could provide more information on the underlying vagal disorder.

In this study, we compared the effects of postural changes (supine, sitting, standing) and sleep or wake-
fulness on spectral components of both HR and intraarterial BP tracings in normotensive (NT), borderline hypertensive (BHT), and hypertensive (HT) subjects. Controlled steady-state conditions were selected to variably activate the autonomic nervous system.

Methods

Study Population

The subjects in this study were male volunteers chosen from a routine health check carried out on all 35-, 40-, and 45-year-old people in the city of Tampere, Finland. They gave informed consent to the study, which had been approved by the Ethics Committee of Tampere University Hospital. Before intraarterial BP measurement, BP was recorded by the standard cuff method at least twice on three occasions over a period of 1 to 2 months. According to the guidelines of the World Health Organization (WHO),12 the subjects were classified as NT (systolic BP [SBP] ≤140 and diastolic BP [DBP] ≤90 mm Hg), BHT (SBP <160 and DBP <95 mm Hg but SBP between 141 and 159 mm Hg and/or DBP between 91 and 94 mm Hg), or HT (SBP ≥160 or DBP ≥95 mm Hg). The average (SD) clinic BP levels of the different groups were as follows: NT, 130/82 (7/5) mm Hg; BHT, 143/90 (5/5) mm Hg; and HT, 152/99 (5/5) mm Hg, suggesting a mild degree of hypertension. The distribution of the study subjects was as follows (age groups of 35, 40, and 45 years are separated by slashes, respectively): NT, 12/10/11; BHT, 9/9/11; and HT, 8/14/12. None had been treated for hypertension before the study. Some of the NT subjects had been included previously in smaller studies reported from our laboratory.13,14

Blood Pressure and Electrocardiogram Recording

All study periods were extracted from long-term ambulatory recordings. Direct arterial BP and one bipolar electrocardiogram (ECG) lead were recorded over a 30-hour period with a Medilog 20 FM recorder (Oxford Medical Systems Ltd, Abingdon, Oxford, UK). A cannula was positioned in the brachial artery of the nondominant arm and linked by a pressure transducer to a recorder worn by the subject on a belt. The transducer was placed at the level of the fourth rib on the chest. A pump system slowly infused heparinized saline solution into the catheter to keep it patent. The pressure transducer was calibrated with constant steps of 0, 50, 100, 150, 200, and 275 mm Hg three times during the recording. The frequency response of the BP channel in the entire recording and replay system has been shown to be 0 to 15 Hz.15

Data Acquisition and Signal Processing

A tape recording was played back at 60 times real time. During the playback the BP signal was pretreated with a 1800-Hz low-pass filter and the ECG signal with a 3000-Hz low-pass filter. Thus, the real-time cutoff frequencies were 30 and 50 Hz, respectively. Analog-to-digital conversion of the two signals was accomplished by means of a PDP 11/34 computer (Digital Equipment Corp, Maynard, Mass) at 6000-Hz sampling frequency per channel to achieve a real-time A/D conversion of 100 Hz. The amplitude resolution was 12 bits. Automatic detection of cardiac cycle fiducial points on the BP signal and pulse classification into normal, abnormal, and artificial were carried out on the same computer.16 The HR was derived from R waves of the ECG. The signals were then transformed to time series of instantaneous SBP (mm Hg), DBP (mm Hg), and HR (beats per minute [bpm]) values. After linear interpolation of the consecutive heartbeats by the method of Luceak and Lurie,17 the sampling rate of these time series was equalized to correspond to one sample per second.

Power Spectrum Analysis

The following steady-state situations were selected for the spectral analysis: a sleeping period at about 2 AM in the nighttime and supine, sitting, and standing periods in the daytime. The posture manipulations were included as part of a series of physical tests in the laboratory. Graphic displays of the time series were viewed to select 5-minute data segments in which no evident nonstationarities were present. A stationery time series, by definition, does not change its statistical parameters, ie, mean value, probability distribution, or frequency content, with time.18 One HT subject had a strongly nonstationary time series in the sitting position and another in the standing position. These data segments were excluded from the study. Two subjects from the same group, 40-year-old HT subjects, had no nighttime recordings for technical reasons. The power spectrum analysis was carried out with a Sparstation IPC (Sun Microsystems, Inc). The analysis program was based on MATLAB subroutines (The Mathworks, Inc).

Before the analysis, any trends and zero frequency were removed from the time series by estimating the trend with 90-second moving median-filtered time series and subtracting the trend estimate from the original sequence. Equidistant sampling allowed direct spectral analysis using an autoregressive modeling method.19 Power spectrum density (PSD) estimates were calculated by first fitting the 30-order autoregressive model by the least-squares method to each data segment and then calculating the PSD estimates from the models. The PSD estimates were then quantified by integrating the area under bandwidths of 0.02 to 0.075 Hz (low-frequency, LF), 0.075 to 0.15 Hz (midfrequency, MF), and 0.15 to 0.35 Hz (high-frequency, HF) in the spectral curve. The PSD estimates were calculated in absolute (bpm^2/Hz or [mm Hg]^2/Hz) and normalized units. Normalized data were calculated by subtracting the mean of the data segment from each value of the time series and dividing the difference by its standard deviation. Thus, the mean of each normalized data segment was 0 and its variance 1.

Statistics

The absolute values of PSD estimates are represented as box and whisker plots, because the data are skewed to the right. The PSD estimates after normalization and logarithmic transformation, which produced distributions that were nearly normal and also stabilized the variances, are expressed as means and SD. Data were analyzed using BMDP statistical software (Los Angeles, Calif). Pairwise comparisons were performed by two-way ANOVA using a priori contrasts.

Results

Blood Pressure and Heart Rate

The BHT and HT groups displayed only slightly elevated BP levels (Table 1), suggesting that these subjects indeed had initial stages of primary hypertension. The intergroup differences in SBP and DBP were statistically highly significant almost without exception (data not shown), but in HR there were marked differences only between the BHT and HT groups. The HT group showed somewhat higher HR than the BHT group in supine, sitting, and standing positions (P values of .03, .03, and .02, respectively).

Frequency Domain Analyses

Wakefulness and change from supine position to sitting and standing positions caused more or less parallel changes in the variability of SBP, DBP, and HR over the whole frequency range (Figure): Activation of the sympathetic nervous system is clearly seen in the MF band and to a lesser extent also in the LF band of
TABLE 1. Average Systolic and Diastolic Blood Pressures and Heart Rate in Different Test Situations

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Sleep</th>
<th>Supine</th>
<th>Sitting</th>
<th>Standing</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP, mm Hg</td>
<td>NT</td>
<td>94 (10)</td>
<td>116 (10)</td>
<td>126 (10)</td>
<td>123 (9)</td>
</tr>
<tr>
<td></td>
<td>BHT</td>
<td>107 (11)</td>
<td>132 (10)</td>
<td>138 (9)</td>
<td>136 (8)</td>
</tr>
<tr>
<td></td>
<td>HT</td>
<td>114 (13)</td>
<td>137 (12)</td>
<td>143 (14)</td>
<td>141 (13)</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>NT</td>
<td>58 (9)</td>
<td>64 (8)</td>
<td>77 (7)</td>
<td>79 (8)</td>
</tr>
<tr>
<td></td>
<td>BHT</td>
<td>64 (7)</td>
<td>73 (7)</td>
<td>83 (6)</td>
<td>86 (7)</td>
</tr>
<tr>
<td></td>
<td>HT</td>
<td>69 (10)</td>
<td>78 (6)</td>
<td>89 (8)</td>
<td>93 (9)</td>
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<tr>
<td>HR, bpm</td>
<td>NT</td>
<td>60 (8)</td>
<td>59 (8)</td>
<td>67 (11)</td>
<td>79 (11)</td>
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<tr>
<td></td>
<td>BHT</td>
<td>59 (7)</td>
<td>58 (10)</td>
<td>65 (11)</td>
<td>78 (13)</td>
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<tr>
<td></td>
<td>HT</td>
<td>61 (8)</td>
<td>63 (9)</td>
<td>70 (9)</td>
<td>84 (14)</td>
</tr>
</tbody>
</table>

SBP indicates systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; bpm, beats per minute; NT, normotensive subjects (n=33); BHT, borderline hypertensive subjects (n=29); and HT, hypertensive subjects (n=32 to 34). Data are mean (SD).

Within-Group Differences of Power Spectra

The BHT group exhibited attenuated changes between different circumstances in the LF band (Table 4). Another notable finding was that HR variability and DBP variability were subject to greater changes than SBP variability in the HF band. However, no evident differences were seen between the groups.

Discussion

To our knowledge, this is the first study to demonstrate that there are qualitative differences in the short-term oscillations in BP between NT, BHT, and HT individuals. The PSD estimates for DBP provided the best and those for HR the poorest statistical separation between the groups. In the following discussion we therefore concentrate on the PSD estimates for DBP. There were two important observations. First, the LF variability was frequently higher or the MF variability lower in the BHT group compared with the other groups. Second, the responses between nighttime and daytime values of LF
variability were blunted in the BHT group. In addition, we found some markers of diminished HF variability for DBP in the BHT group. Only some hypotheses can be proposed as to the mechanisms underlying the findings. It should be noted that the actual origin of these oscillations, especially in the LF band, is a matter of debate.

Many authors have by different experimental methods documented increased sympathetic tone in borderline hypertension. For example, Anderson et al observed an increased rate of bursts of activity in the sympathetic fibers of the peroneal nerves in direct microneurographic recordings in BHT subjects. Alteration in the function of the sympathetic nervous system may also explain our main result. Pomeranz et al demonstrated that in the supine position fluctuations in HR below 0.12 Hz are largely mediated by the parasympathetic nervous system, whereas in the standing posture there is a strong sympathetic influence on these oscillations. The increased sympathetic tone induced by postural change was seen in the power spectrum of the HR as a shift from HF to LF. Thus, in our study population the frequency shift in the oscillations of BP and HR may have been greater in BHT subjects than in the other groups in each body position as a consequence of elevated sympathetic tone. The increased sympathetic activity may in turn be central in origin, or it may result from altered peripheral mechanisms (e.g., increased vascular reactivity to norepinephrine or change in regional release or reuptake of norepinephrine). An increased central sympathetic outflow, for example, may be due to a variety of behavioral factors because there were no intergroup differences in the nighttime. On the other hand, it may be conjectured that, in particular, the baroreceptor oscillations are shifted to lower frequencies, a phenomenon that has previously been linked to dysfunction of the autonomic nervous system in diabetes mellitus and sudden infant death syndrome. In that case, the reason could lie in the afferent, central, or efferent part of the baroreceptor reflex arc.

It is more difficult to explain the blunted night-day pattern for the LF band observed in the BHT group. In fact, the question perhaps is why the NT and HT groups compared with the BHT group have so much lower
TABLE 3. Comparisons of Power Spectrum Density Estimates Between Blood Pressure Groups in Each Test Situation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Comparison</th>
<th>LF</th>
<th>MF</th>
<th>HF</th>
<th>LF</th>
<th>MF</th>
<th>HF</th>
<th>LF</th>
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<th>HF</th>
<th>LF</th>
<th>MF</th>
<th>HF</th>
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<tbody>
<tr>
<td>SBP</td>
<td>NT vs BHT</td>
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<td>NT vs HT</td>
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<td>BHT vs HT</td>
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<tr>
<td>HR</td>
<td>NT vs BHT</td>
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<td>NT vs HT</td>
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<td>BHT vs HT</td>
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</table>

LF indicates low frequency; MF, midfrequency; HF, high frequency; SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; NT, normotensive subjects (n=33); BHT, borderline hypertensive subjects (n=29); and HT, hypertensive subjects (n=32 to 34).

Logical operators < and > are used to define the relation between means. < and > indicate P<.05; << and >>, P<.01; <<< and >>>, P<.001; and -, not significant.

Normalized powers for the LF band in the daytime than in the night. The mechanisms underlying these findings apparently cannot be attributed solely to changes in sympathetic balance. Di Rienzo et al27 have previously described a tendency of the LF power for SBP and DBP, and even more clearly for pulse interval, to increase during sleep in mildly hypertensive subjects.

The circadian pattern in the MF power for SBP and HR in turn seems to parallel sympathetic activity.28-30 Guzzetti et al30 have reported blunted circadian changes in the MF component for HR (approximately 0.1 Hz in their study) in mild hypertension.

Guzzetti et al30 also have shown normalized HF variability for HR, a proposed measure of parasympathetic

TABLE 4. Comparisons of Power Spectrum Density Estimates Between Different Test Situations in Each Blood Pressure Group

<table>
<thead>
<tr>
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<th>MF</th>
<th>HF</th>
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</thead>
<tbody>
<tr>
<td>SBP</td>
<td>SL vs SU</td>
<td>&gt;&gt;&gt;</td>
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<td>SL vs SI</td>
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<td>SL vs ST</td>
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<td>SU vs ST</td>
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<td>SI vs ST</td>
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<td>DBP</td>
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<td>SI vs ST</td>
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<td>SL vs SU</td>
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<td>SL vs SI</td>
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LF indicates low frequency; MF, midfrequency; HF, high frequency; NT, normotensive subjects (n=33); BHT, borderline hypertensive subjects (n=29); HT, hypertensive subjects (n=32 to 34); SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; SL, sleep; SU, supine; SI, sitting; and ST, standing.

Logical operators < and > are used to define the relation between means. < and > indicate P<.05; << and >>, P<.01; <<< and >>>, P<.001; and -, not significant.
nervous system function, to be lower in hypertensive than in normotensive individuals. We failed to detect significant between-group differences in HF variability for HR, although absolute powers in the HF band appeared to be somewhat lower in the BHT group than in the others. Our data suggest that DBP variability in respiratory frequency can also mirror vagal activity. Changes in interbeat interval (IBI, which is the inverse of HR) are perhaps better reflected in DBP than in SBP, despite the fast baroreceptor reflex, because a decreased IBI will interrupt the diastolic decline at a higher point on the curve and raise the pressure, whereas an increased IBI will lead to a longer decline and lower level of DBP. The IBI is not a major determinant for SBP as it is for DBP. Reduced HF variability for DBP in BHT subjects should have been associated with a significant reduction in HF variability for HR also, but our hypothesis is supported by the fact that HF variability for DBP and HR was subject to greater changes between different test situations than that for SBP.

Different methods for assessing sympathovagal balance do not necessarily correlate52 and may represent different aspects of circulatory control. In our study the HR was in most situations fastest in the HT group. This is in disagreement with the explanation that the sympathetic tone is increased and parasympathetic tone decreased expressly in the BHT group.

Parati et al33 observe no differences between mean normalized power values of SBP, DBP, or pulse interval in ambulant normotensive and mildly hypertensive subjects. There can be several reasons for the discrepancy between the results of Parati et al and ours. First, in our opinion, the classification of subjects into three groups according to the WHO criteria is of great importance. The alterations in the PSD estimates of SBP and DBP do not appear to change parallel with severity of disease, and it is the BHT group that is clinically problematic—how to treat them. We emphasize that the grouping of our study subjects was done in a prospective fashion, during a period of 1 to 2 months.

Second, in ambulatory subjects in daily life the signals are to a considerable extent strongly nonstationary, which makes for problems in spectral analysis. Our preliminary data indicate that the between-group differences disappear, owing to the great uncertainty of the PSD estimates, when our present techniques are applied to nonstationary time series. Thus, special techniques are needed for these recordings to find accurately the rhythmicity hidden behind the signals. It should be noted that spectral analysis can be used to every existing signal without any assumptions of stationarity. However, when a signal to be analyzed contains large peaklike or steplike variations, the power of these variations will coat most of the power of other oscillatory variations. In the present study, to maximize the resolution of the analysis concerning the oscillations, especially in the lower (0.02 to 0.15 Hz) frequency range, we excluded by visual inspection any sequence containing nonoscillating disturbances or artifacts.

Third, a critical question in this type of research is whether the duration of the data segment is sufficient to obtain reliable results. Typically, 5 minutes of stationary data is used to compute the PSD estimates in the band from 0.02 to 1.0 Hz,4 which gives 6 to 22.5 periods of the fundamental frequencies in the LF band. The use of a fast Fourier transform algorithm of 256 beats causes variation in the length of the data segment. The higher the HR, the shorter the data segment, and consequently, the more unreliable the estimate for the LF band. Moreover, the autoregressive modeling method is considered more suitable for computing PSD estimates from relatively short-length data by providing better frequency resolution than fast Fourier transform.

Finally, our viewpoint is that components with a frequency slower than respiration should, as in Parati et al,33 be divided into two bands, where the lower limit of the center frequency in the baroreceptor rhythm, that is, 0.075 Hz,34 is a logical dividing line.

The observation that there are three populations with different control of short-term BP variability might prove to be of clinical relevance. The spectral indexes may provide information of prognostic value. Most BHT individuals do not develop sustained hypertension, and it should be of paramount importance for both society and the individual patient to be able to identify those subjects who are at high risk of cardiovascular morbidity and hence need treatment. Further studies are needed to define subpopulations with abnormal control of BP and HR variability. Cross-spectral analysis35 may provide additional information to aid in the clinical diagnosis of hypertension.

Acknowledgments

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References


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