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Sympathovagal balance is major determinant of short-term blood pressure variability in healthy subjects

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GHISLAINE GEELEN,[3] AND ESKO LÄNSIMIES[1]
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Laitinen, Tomi, Juha Hartikainen, Leo Niskanen, Ghislaine Geelen, and Esko Länsimies. Sympathovagal balance is major determinant of short-term blood pressure variability in healthy subjects. *Am. J. Physiol.* 276 (*Heart Circ. Physiol.* 45): H1245–H1252, 1999.—Short-term blood pressure variability (BPV) has been suggested to provide important information about cardiovascular regulation. However, the background of BPV, its determinants, and physiological correlates have remained obscure. The aim of this study was to characterize physiological correlates of BPV and to investigate associations between BPV and neural and hormonal regulatory systems at rest in healthy subjects. We studied 117 healthy, normal-weight, nonsmoking male and female subjects aged 23–77 yr. Spectral analysis of BPV and heart rate variability (HRV) was performed from 5-min blood pressure (Finapres) and electrocardiogram recordings during controlled breathing. Baroreflex sensitivity (BRS) was measured using the phenylephrine method. In addition, plasma concentrations of norepinephrine, epinephrine, and arginine vasopressin and plasma renin activity were measured. We found that the ratio between the low- and high-frequency components of HRV, an index of cardiac sympathovagal balance, correlated positively with total power and very low- and low-frequency components of systolic and diastolic BPV and inversely with high-frequency components of systolic and diastolic BPV. BRS, predominantly a measure of cardiac vagal regulation, correlated inversely with BPV. Furthermore, age, gender, body mass index, and systolic blood pressure contributed to BPV. Vasoactive hormones were not significant correlates of BPV. We conclude that sympathovagal balance of cardiovascular regulation is the major determinant of BPV. Other factors associated with BPV are age, gender, body mass index, blood pressure, and BRS.

autonomic nervous system; baroreflex sensitivity; heart rate variability

RHYTHMIC, SHORT-TERM blood pressure variability (BPV) is an intriguing phenomenon most likely yielding important but hitherto largely neglected information about cardiovascular regulation. BPV consists of rhythmic and nonrhythmic blood pressure changes. In particular, the rhythmic component of BPV has been thought to provide information about central neural regulation, whereas physical and mental activities are thought to be responsible for the nonrhythmic component of BPV (23). The sympathetic control of the microcirculation has been suggested to play an important role in the origin of BPV (3, 14). In addition, blood pressure is regulated by baroreflex, a buffering mechanism that opposes increases or decreases of blood pressure by changes in heart rate, myocardial contractility, and peripheral resistance (9). In this respect, baroreflex has been thought to be the link between BPV and heart rate variability (HRV) (22, 37). On the other hand, there is some evidence that heart rate changes related to respiration may contribute to BPV, suggesting fundamental relationships between BPV and HRV (35).

The knowledge about determinants and physiological correlates of BPV is limited and refers mainly to the time-domain analysis of BPV. Some studies have investigated the association between rhythmic, short-term BPV and HRV (12, 29, 35–37). Only a few studies have investigated the association between rhythmic, short-term BPV and blood pressure (4, 31, 34), and there are no reports dealing with the relation between frequency-domain measures of BPV and baroreflex sensitivity (BRS). This study was designed to characterize determinants and physiological correlates of BPV and to investigate the association between BPV and neural and hormonal regulatory systems at rest in healthy subjects.

METHODS

Subjects. The study population consisted of 117 healthy subjects, 59 men and 58 women, with an age range of 23–77 yr (47.5 ± 1.5 yr, mean ± SE; Table 1). The subjects were evaluated with a detailed history, physical examination, routine laboratory tests, and a clinical exercise test. All subjects were free of hypertension and other systemic diseases. None of them was taking any cardiovascular medication or smoking. All subjects were requested to abstain from beverages with caffeine for 12 h before the experiment. The subjects came to the laboratory between 0730 and 0900, 45 min after ingestion of a light breakfast. Subjects gave fully informed consent, and the study protocol was approved by the Ethics Committee of Kuopio University Hospital.

Recordings of electrocardiogram and continuous blood pressure. A Viggo-Spectramed (Helsingborg, Sweden) Teflon catheter was inserted into a large antecubital vein of the left arm at least 1 h before the recordings. Electrodes were placed for the electrocardiogram (ECG) recording. Continuous blood pressure recording was performed on the middle finger of the right hand with a Finapres digital plethysmograph (Ohmeda, Englewood, CO). Self-adjustment of the Finapres device was performed just before the recordings, and it was disconnected during the recordings. ECG and blood pressure signals were recorded and simultaneously analog-to-digital converted with

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Table 1. *Clinical, neural, and hormonal characteristics of study population*

Age, yr	47.5 ± 1.5
Body mass index, kg/m^2	23.6 ± 0.2
Heart rate, beats/min	72 ± 1
Systolic blood pressure, mmHg	140 ± 2
Diastolic blood pressure, mmHg	82 ± 1
Baroreflex sensitivity, ms/mmHg	12.7 ± 0.9
Total power of HRV, ms^2	2,408 ± 296
VLF of HRV, ms^2	800 ± 72
LF of HRV, ms^2	376 ± 62
HF of HRV, ms^2	1,197 ± 196
LF/HF of HRV	0.50 ± 0.05
Plasma norepinephrine, nmol/l	1.37 ± 0.07
Plasma epinephrine, nmol/l	0.21 ± 0.04
Plasma arginine vasopressin, pmol/l	0.54 ± 0.13
Plasma renin activity, $\mu g \cdot l^{-1} \cdot h^{-1}$	1.37 ± 0.09

Values are means ± SE; n = 117 subjects. HRV, heart rate variability; VLF, very low frequency power; LF, low frequency power; HF, high frequency power.

temporal resolution of 200 Hz/channel and amplitude resolution of 12 bits (33). All data acquisition, QRS detection (accuracy <2 ms), and analyses were performed with an IBM-PC-compatible microcomputer with CAFTS software (Medikro, Kuopio, Finland).

Study protocol. The subjects performed a series of tests. After 1-h rest in the supine position, a controlled breathing test was performed. This was followed by withdrawal of blood samples, and, finally, BRS was assessed by the phenylephrine method.

Assessment of blood pressure and HRV. Blood pressure and HRV were determined from 5-min blood pressure and ECG recordings. Subjects were asked to breathe following a metronome at a 0.2-Hz frequency for 5 min. Spectral estimates of systolic and diastolic blood pressures and R-R intervals were obtained from stationary regions free of ectopic beats and technical artifacts. After detrending (1st degree), a modified covariance autoregressive model (fixed model order 14) was used to obtain power spectral estimates of BPV and HRV. Total power (TP, variance) in the frequency range from 0 to 0.5 Hz times frequency equal to mean R-R interval (Hz) was divided into three frequency bands: very low frequency (VLF; 0–0.07 Hz), low frequency (LF; 0.07–0.15 Hz), and high frequency (HF; 0.15–0.40 Hz). The signal power of each band was calculated as an integral under the respective power spectral density function and expressed in absolute units (mmHg^2 or ms^2).

Hormone measurements. Blood samples (total of 25 ml) for the assessment of plasma catecholamine, insulin, and arginine vasopressin concentrations and plasma renin activity were taken just after the controlled breathing test. Within 45 min after withdrawal, the blood samples were centrifuged at +4°C for 15 min and plasma samples were aliquoted for different hormone measurements and stored immediately either at −20°C (renin activity) or −80°C (catecholamines and arginine vasopressin). Plasma catecholamine concentrations were measured by HPLC with electrochemical detection (18). Plasma arginine vasopressin concentrations and plasma renin activity were measured by RIA, arginine vasopressin using the RIA set in the laboratory with synthetic arginine vasopressin (375 IU/mg; gift from Ferring, Malmö, Sweden) and antiserum K9-IV (gift from Dr. L. C. Keil, NASA Ames Research Ctr., Moffett Field, CA) (7) and renin activity using a commercial kit (Medix Angiotensin I test; Oy Medix Ab, Helsinki, Finland).

Assessment of BRS. Five minutes after the withdrawal of the blood samples, subjects were given a 150-μg bolus injection of phenylephrine intravenously flushed with 10 ml of saline. The phenylephrine test was repeated up to five times to get three acceptable measurements. BRS was assessed with a modification of the method described by Smyth et al. (32). In brief, the slope of the linear relationship between the R-R intervals (ms) and systolic blood pressure (mmHg) of the preceding cardiac cycle was calculated from a user-defined time window using linear least-mean-squares fitting. The analysis window included the time from the beat that started the sustained rise of systolic blood pressure to the beat of the peak blood pressure rise. Only regression lines with a correlation coefficient >0.80 or statistically significant ($P < 0.05$) were accepted. The average of the three measurements was used in further analyses.

Statistical analyses. Systolic blood pressure, BPV, HRV, BRS, and hormonal data were analyzed after logarithmic transformation. Student's t-test was used to test the significance of differences between men and women. One-way analysis of variance with Duncan's multiple-range test was used to test the significance of differences among the age groups for BPV and among the groups formed by the division of the study population into tertiles of BRS and tertiles of the LF-to-HF ratio of HRV. Univariate correlations were calculated using Pearson's correlation analysis. In addition, stepwise multiple-regression analysis was used to evaluate the independent determinants of BPV. A P value <0.05 was considered statistically significant. All values are presented as means ± SE. Statistical analyses were performed using a statistical program, SPSS for Windows (version 7.5.1; SPSS, Chicago, IL).

RESULTS

Clinical, neural, and hormonal characteristics. Table 1 shows the characteristics of the study population. The clinical, neural, and hormonal characteristics in relation to age and gender have been published previously (13).

BPV and HRV. All components of systolic and diastolic BPV correlated significantly with the LF-to-HF ratio of HRV in both univariate and multivariate analyses (Tables 2 and 3). The correlation was positive

Table 2. *Univariate correlations (correlation coefficients) between HRV and systolic and diastolic blood pressure variability in study population*

	HRV				
	TP	VLF	LF	HF	LF/HF
			SBPV		
TP	−0.11	−0.05	0.01	−0.21	0.33‡
VLF	−0.06	0.02	0.01	−0.20*	0.35‡
LF	0.03	0.06	0.24*	−0.06	0.49‡
HF	−0.22*	−0.30†	−0.27†	−0.14	−0.20*
LF/HF	0.19*	0.28†	0.38‡	0.05	0.51‡
			DBPV		
TP	0.04	0.09	0.12	−0.10	0.34†
VLF	0.02	0.16	0.07	−0.17	0.39‡
LF	0.18	0.20*	0.36‡	0.10	0.40‡
HF	0.08	−0.06	−0.01	−0.10	−0.20*
LF/HF	0.04	0.14	0.21*	−0.02	0.37‡

TP, total power; SBPV, systolic blood pressure variability; DBPV, diastolic blood pressure variability. *$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$.

for TP, VLF power, and LF power of systolic and diastolic BPV but negative for the HF power of BPV. To further illustrate the relationship between BPV and HRV, the subjects were divided into tertiles of the LF-to-HF ratio of HRV (lowest: <0.21, middle: 0.22–0.46, highest: >0.46). Subjects in the highest tertile had the highest TP and VLF and LF power and the lowest HF power of systolic BPV (Fig. 1).

BPV and BRS. In the univariate analysis, BRS correlated inversely with TP of both systolic and diastolic BPV ($r = -0.33$, $P < 0.001$ and $r = -0.27$, $P < 0.01$, respectively). Correspondingly, when the subjects were divided into BRS tertiles (lowest: <6.7, middle: 6.8–15.0, highest: >15.1 ms/mmHg), subjects in the lowest BRS tertile had the highest systolic and diastolic blood pressures as well as the highest BPV (Fig. 2). According to univariate and multivariate analysis, BRS was associated with the HF component of systolic BPV and the VLF component and the LF-to-HF ratio of diastolic BPV (Table 3). In addition, we found that the blood pressure component of BRS, i.e., the systolic blood pressure response rather than the heart rate response during the phenylephrine test, was responsible for the BRS-BPV association.

BPV and blood pressure. In the univariate analysis, both systolic and diastolic blood pressures correlated significantly with TP ($r = 0.31$, $P < 0.001$ and $r = 0.27$, $P < 0.01$, respectively; Fig. 3), VLF power ($r = 0.29$, $P < 0.01$ and $r = 0.26$, $P < 0.01$, respectively), and HF power of systolic BPV ($r = 0.22$, $P < 0.05$ and $r = 0.22$, $P < 0.05$, respectively). In addition, they correlated significantly with the VLF power of diastolic BPV ($r = 0.19$, $P < 0.05$ and $r = 0.20$, $P < 0.05$, respectively). However, in the multivariate analysis only systolic blood pressure was a significant correlate of TP and the VLF power of systolic BPV (Table 3).

BPV and age. There was a significant correlation between age and systolic ($r = 0.50$, $P < 0.001$) and diastolic ($r = 0.45$, $P < 0.001$) blood pressures. Age was not a significant determinant of TP of systolic and diastolic BPV in the univariate or multivariate analyses (Table 3 and Fig. 4). In the univariate analysis, age correlated significantly with HF power ($r = 0.26$, $P < 0.01$) and LF-to-HF ratio of systolic BPV ($r = -0.28$, $P < 0.01$) and LF power ($r = -0.35$, $P < 0.001$) and LF-to-HF ratio of diastolic BPV ($r = -0.26$, $P < 0.01$). In the multivariate analysis, after we adjusted for BRS and the LF-to-HF ratio of HRV, age was an independent determinant of the LF-to-HF ratio of systolic BPV as well as VLF power, LF power, and the LF-to-HF ratio of diastolic BPV (Table 3). In addition, we found that old subjects (60–77 yr) had a higher HF power of systolic BPV but a lower LF power of diastolic BPV and a lower LF-to-HF ratio of both systolic and diastolic BPV than young (23–39 yr) and middle-aged (40–59 yr) subjects (Table 4).

BPV and gender. Gender was a significant determinant of LF power and the LF-to-HF ratio of systolic

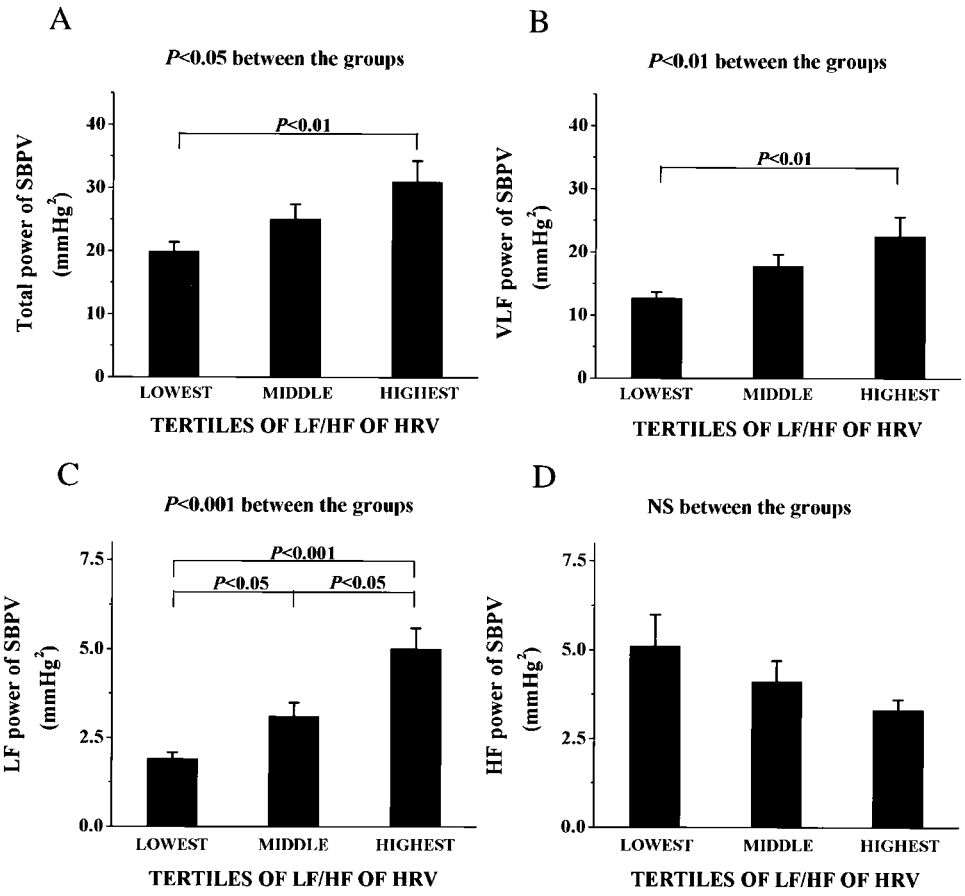

Fig. 1. Relationship between blood pressure variability (BPV) and low frequency (LF)-to-high frequency (HF) ratio of heart rate variability (HRV). Total power (*A*) and very low frequency (VLF; *B*), LF (*C*), and HF (*D*) powers of systolic BPV (SBPV) in subjects divided into tertiles of LF-to-HF ratio of HRV. NS, not significant.

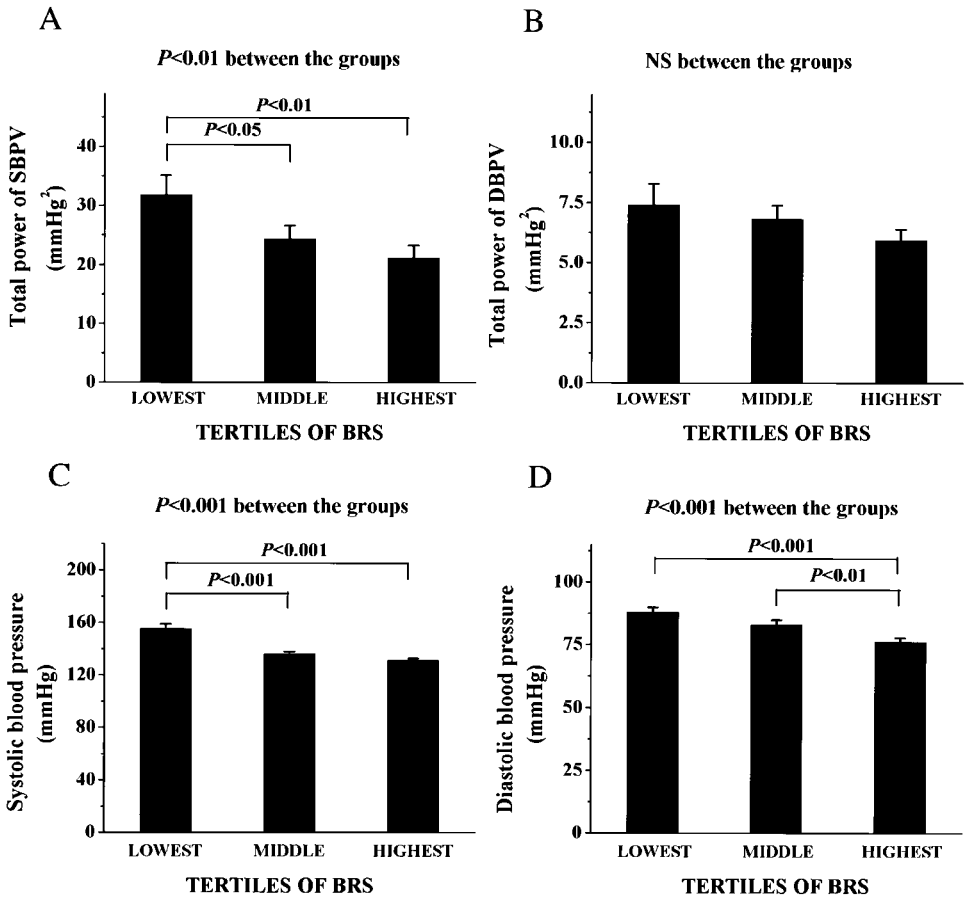

Fig. 2. Relationship between BPV and baroreflex sensitivity (BRS). Total power of SBPV (*A*) and diastolic BPV (DBPV; *B*) and systolic (*C*) and diastolic (*D*) blood pressures in subjects divided into tertiles of BRS.

BPV and LF power of diastolic BPV in multivariate analysis (Table 3). Women had significantly higher LF components of both systolic BPV and diastolic BPV compared with men ($P < 0.05$; Fig. 5). In addition, TP and VLF components of systolic and diastolic BPV also tended to be higher (but nonsignificantly) in women compared with men (Fig. 5).

BPV and body mass index. In the univariate analysis, body mass index correlated inversely with TP of systolic and diastolic BPV ($r = -0.20$, $P < 0.05$ and $r = -0.24$, $P < 0.01$, respectively) and with the LF component of diastolic BPV ($r = -0.21$, $P < 0.05$). In the multivariate analysis, body mass index remained as an independent correlate of TP of both systolic and diastolic BPV and also of the VLF component of systolic BPV (Table 3).

BPV and vasoactive hormones. We found no significant correlations among plasma concentrations of norepinephrine, epinephrine, arginine vasopressin, or insulin or plasma renin activity and any components of either systolic or diastolic BPV ($r = -0.13$ to $r = 0.17$, not significant).

Table 3. *Multivariate correlations (β-values) for SBPV and DBPV and selected variables*

	Age	Gender	Body Mass Index	Systolic Blood Pressure	Baroreflex Sensitivity	LF/HF of HRV	r^2
				SBPV			
TP	−0.12	−0.04	−0.22†	0.25*	−0.17	0.29‡	0.27
VLF	−0.02	−0.01	−0.21*	0.32‡	−0.13	0.32‡	0.24
LF	−0.15	0.28‡	−0.01	0.05	0.02	0.48‡	0.30
HF	0.13	−0.05	−0.03	0.12	−0.28†	−0.25†	0.13
LF/HF	−0.30‡	0.17*	0.07	0.02	0.04	0.55‡	0.22
				DBPV			
TP	−0.01	−0.05	−0.25†	0.16	−0.16	0.34‡	0.18
VLF	−0.28*	−0.06	−0.11	0.17	−0.40‡	0.37‡	0.24
LF	−0.37‡	0.25†	−0.05	0.15	−0.12	0.42‡	0.36
HF	0.16	−0.01	−0.03	0.08	−0.03	−0.19*	0.03
LF/HF	−0.44‡	0.05	0.05	0.01	−0.36‡	0.56‡	0.25

*$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$.

DISCUSSION

The fluctuations in blood pressure and heart rate have been suggested to reflect the dynamic interplay of diverse physiological processes (2). Therefore, rhythmic, short-term BPV may represent important information about cardiovascular regulation. However, before BPV can be used in clinical and experimental studies, its physiological correlates and its behavior in a healthy population must be clarified. In this study we demonstrate that rhythmic BPV yields important information about cardiovascular regulation, particularly the status of the sympathetic nervous system or sympathovagal balance. In addition, we demonstrate that physiological variables such as age, gender, body mass index, blood pressure, and BRS are independent physiological correlates of BPV.

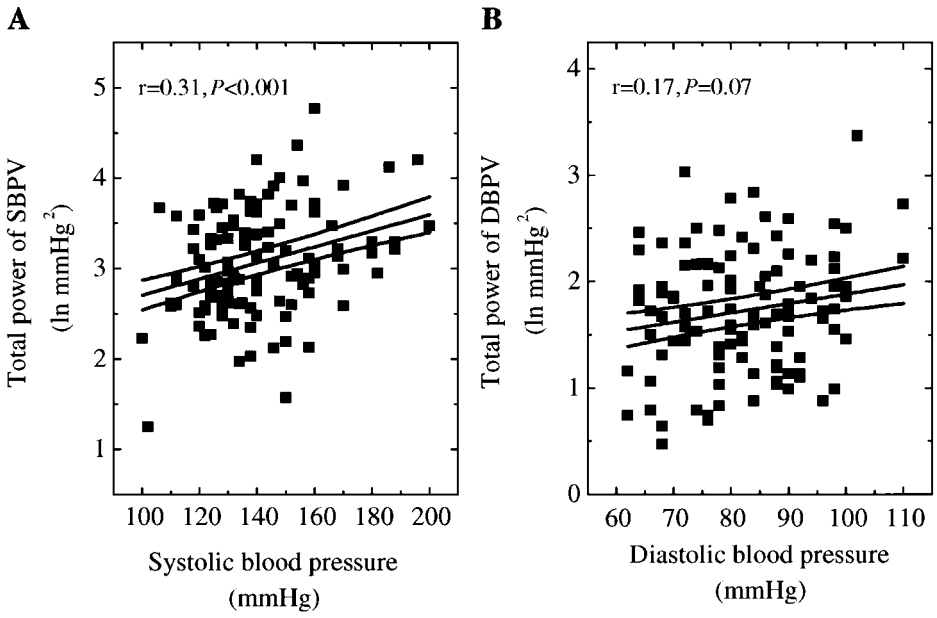

Fig. 3. Relationships between systolic blood pressure and total power of SBPV (*A*) and diastolic blood pressure and total power of DBPV (*B*). Regression slopes and 95% confidence intervals are shown. BPV is expressed after logarithmic transformation.

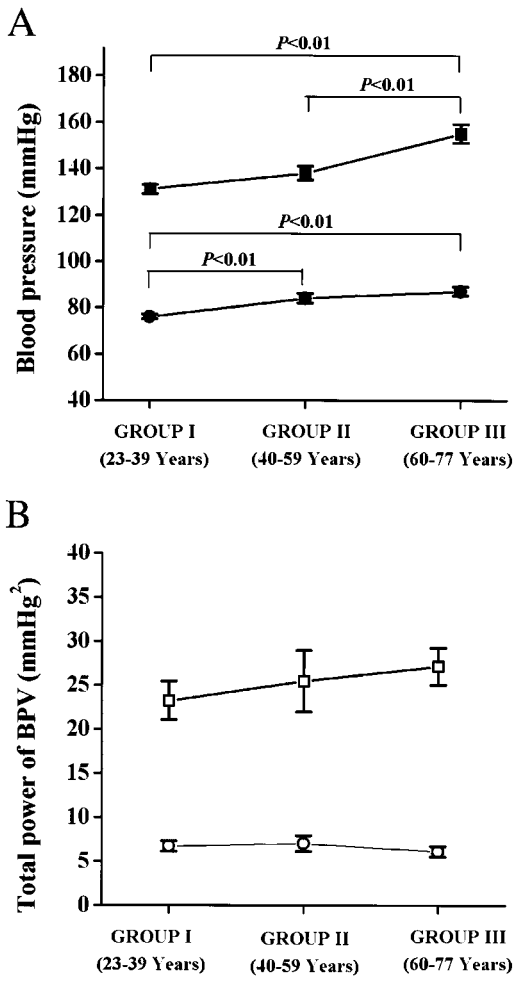

Fig. 4. Effect of age on systolic (■) and diastolic (●) blood pressure (*A*) and total power of systolic (□) and diastolic (○) BPV (*B*). Values are means ± SE.

Perhaps the most important finding of the present study was that all spectral components of BPV correlated significantly with the LF-to-HF ratio of HRV, an index of cardiac sympathovagal balance (1). The correlation was positive for TP and VLF and LF components, suggesting that these provide information about the sympathetic status of cardiovascular regulation. This is further supported by our finding that TP of systolic BPV correlated negatively with the HF component of HRV, a measure of cardiac parasympathetic regulation (1). These observations are in line with previous studies suggesting a close connection between the sympathetic nervous system and BPV. Rimoldi et al. (26) observed an increase in the LF component of BPV during nitroglycerin infusion, transient coronary occlusion, and exercise, maneuvers known to increase sympathetic tone. Correspondingly, Pagani et al. (19) found in healthy subjects a predominance of LF oscillations of blood pressure, R-R interval, and muscle sympathetic nerve activity during sympathetic activation, whereas during sympathetic inhibition, the HF component of cardiovascular variability predominated. In an experimental study by Hedman et al. (10), blocking sympathetic nerve transmission by spinal anesthesia decreased all components of BPV, whereas vagotomy decreased HRV but did not influence BPV.

The arterial baroreflex serves as a pressure buffer system against increases and decreases in arterial pressure (9). Thus one might expect that baroreflex function contributes to BPV. In experimental studies, baroreceptor denervation has resulted in increased BPV and decreased HRV (30). BRS has been shown to correlate inversely with both 2-h and 24-h standard deviations of intra-arterial blood pressure recordings (16). However, to our knowledge, no previous studies have evaluated the association between BRS and frequency-domain measures of BPV, particularly during standardized conditions. In our study, BRS correlated

Table 4. *Effect of age on blood pressure variability*

	Group I (23–39 yr) *n* = 44	*Group II* (40–59 yr) *n* = 38	*Group III* (60–77 yr) *n* = 35	ANOVA	Group Comparisons		
					I–II	*I–III*	*II–III*
			SBPV				
TP, mmHg^2	24.2 ± 2.4	25.4 ± 3.5	27.1 ± 2.1	NS	NS	NS	NS
VLF, mmHg^2	17.1 ± 2.0	18.0 ± 3.1	18.3 ± 1.6	NS	NS	NS	NS
LF, mmHg^2	3.5 ± 0.4	4.0 ± 0.7	2.5 ± 0.3	NS	NS	NS	NS
HF, mmHg^2	3.5 ± 0.4	3.3 ± 0.3	6.0 ± 1.0	*	NS	*	*
LF/HF	1.45 ± 0.23	1.66 ± 0.25	0.78 ± 0.13	†	NS	†	†
			DBPV				
TP, mmHg^2	6.7 ± 0.6	7.0 ± 0.9	6.1 ± 0.6	NS	NS	NS	NS
VLF, mmHg^2	4.2 ± 0.4	4.8 ± 0.8	4.0 ± 0.4	NS	NS	NS	NS
LF, mmHg^2	1.5 ± 0.2	1.5 ± 0.2	0.7 ± 0.1	‡	NS	†	†
HF, mmHg^2	1.0 ± 0.2	0.6 ± 0.1	1.3 ± 0.4	NS	NS	NS	NS
LF/HF	3.31 ± 0.53	3.26 ± 0.49	1.33 ± 0.22	†	NS	†	†

Values are means ± SE; *n*, no. of subjects. *$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$; NS, not significant.

inversely with BPV. In addition, subjects in the lowest BRS tertile had the highest BPV and blood pressure values. BRS is considered to represent predominantly the efficacy of cardiac parasympathetic regulation. Thus our finding of the inverse relationship between BPV and BRS provides indirect evidence supporting the concept that BPV bears information about sympathetic regulation. Interestingly, the magnitude of the systolic blood pressure rise to phenylephrine was the dominating component of the BRS-BPV relationship. This infers that high BPV, indicative of a less potent buffering capacity against blood pressure changes, results in higher blood pressure response (rather than heart rate response) during phenylephrine administration.

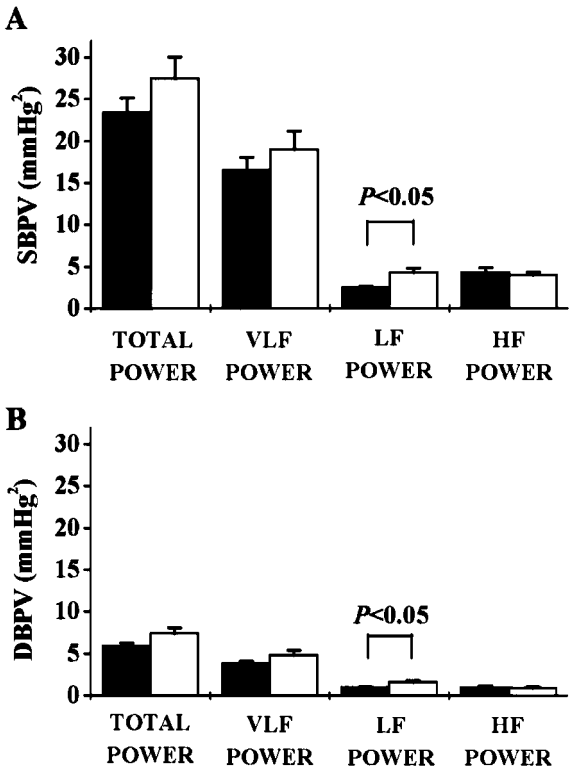

Fig. 5. Effect of gender on systolic (*A*) and diastolic (*B*) BPV. Values are means ± SE. ■, Men; □, women.

In our study with normotensive subjects, we found a positive correlation between blood pressure and rhythmic BPV in healthy subjects. This is in line with Duprez et al. (4), who reported that subjects with borderline hypertension had higher LF and HF components of systolic and diastolic BPV than control subjects. Correspondingly, in another study, hypertensive patients had higher VLF and LF components of systolic BPV and a higher LF component of diastolic BPV than normotensive controls (31). In addition, in the latter study left ventricular hypertrophy correlated negatively with BPV, and hypertensive patients with left ventricular hypertrophy did not have increased BPV compared with normotensive subjects. This suggests that not only blood pressure alone but also adaptive processes such as development of compensatory left ventricular hypertrophy contribute to BPV.

Aging is known to be associated with increased sympathetic activity (6, 17), decreased baroreflex function (5, 8, 13), and reduced vessel wall compliance (25), all of which may influence BPV. Studies using time-domain analyses of ambulatory blood pressure recordings have shown that BPV increases with aging (15, 16). However, there are few data about rhythmic, short-term BPV and aging. In a study based on a large population of healthy subjects, Veerman et al. (38) reported that aging had no significant effect on overall BPV expressed as within-subject coefficient of variation. However, when BPV was assessed by the frequency-domain method, they found that aging was associated with a decrease in midfrequency (0.08–0.12 Hz) components of systolic and diastolic BPV and an increase in the HF component of systolic BPV. In line with that group, in our study the VLF and LF components of diastolic BPV decreased, whereas the HF component of systolic BPV increased with aging. We found a slight but nonsignificant increase in the VLF component of systolic BPV with aging, which agrees with Parati et al. (21). Finally, using multivariate analysis for adjusting the effect of the LF-to-HF ratio of

HRV and BRS, total BPV was not influenced by aging, whereas age turned out to be an independent determinant of the VLF and LF components of diastolic BPV as well as the LF-to-HF ratio of both systolic and diastolic BPV.

In our study both systolic and diastolic BPV, and particularly their LF components, were higher in women than in men. Gender-related differences were described earlier in terms of HRV (11, 27), muscle nerve sympathetic activity (17), and BRS (11, 13), whereas gender-related differences in rhythmic BPV have not been previously reported. Sex hormone-related changes in autonomic regulatory function, body composition, and fluid balance might influence peripheral vasomotor function and thus BPV. However, these mechanisms are poorly understood and need further investigation. We found that body mass index also has an impact on BPV, but the mechanism linking body composition and BPV is not known.

Under nonstimulated conditions in the supine position, we were not able to demonstrate any association between neurohormones and short-term BPV. In fact, this was not surprising, because vasoactive hormones are known to participate in long-term control rather than in short-term control of blood pressure regulation.

In this study, the study population was large, equally distributed between sexes, and with a wide age range. The subjects were well characterized and carefully evaluated for the exclusion of diseases and conditions known to influence cardiovascular regulation. In our study, all recordings were performed under controlled conditions in the supine position. Because respiratory rate has a significant effect on HRV and BPV, breathing frequency was controlled during the recordings as has been recommended (28). The assessment of rhythmic, short-term BPV requires continuous blood pressure recording, which has been the most important limiting factor for the use of this method. In recent years, a method based on continuous monitoring of blood pressure through a finger cuff has offered a noninvasive alternative to the intra-arterial approach (24). In the present study, finger blood pressure was measured beat to beat noninvasively using a Finapres device, which has been found to provide accurate estimates for both means and variabilities for arterial blood pressure (20). This study incorporated many variables into the univariate and multivariate analyses, making it possible to investigate statistically independent associations between BPV and its physiological correlates.

In conclusion, our study demonstrates that BPV, particularly TP and VLF and LF components, represents predominantly sympathetic modulation of cardiovascular regulation. In addition, we found that age, gender, body mass index, blood pressure, and BRS are significant independent determinants of BPV.

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