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Dynamics of spectral components of heart rate variability during changes in autonomic balance

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Højgaard, Michael V., Niels-Henrik Holstein-Rathlou, Erik Agner, and Jørgen K. Kanters. Dynamics of spectral components of heart rate variability during changes in autonomic balance. *Am. J. Physiol.* 275 (*Heart Circ. Physiol.* 44): H213–H219, 1998.—Frequency domain analysis of heart rate variability (HRV) has been proposed as a semiquantitative method for assessing activities in the autonomic nervous system. We examined whether absolute powers, normalized powers, and the low frequency-to-high frequency ratio (LF/HF) derived from the HRV power spectrum could detect shifts in autonomic balance in a setting with low sympathetic nervous tone. Healthy subjects were examined for 3 h in the supine position during 1) control conditions ($n = 12$), 2) acute β -blockade ($n = 11$), and 3) chronic β -blockade ($n = 10$). Heart rate fell during the first 40 min of the control session (72 ± 2 to 64 ± 2 beats/min; $P < 0.005$) and was even lower during acute and chronic β -blockade (56 ± 2 beats/min; $P < 0.005$). The powers of all spectral areas rose during the first 60 min in all three settings, more so with β -blockade ($P < 0.05$). LF/HF was found to contain the same information as powers expressed in normalized units. LF/HF detected the shift in autonomic balance induced by β -blockade but not the change induced by supine position. In conclusion, none of the investigated measures derived from power spectral analysis comprehensively and consistently described the changes in autonomic balance.

sympathetic and parasympathetic nervous activity; low frequency-to-high frequency ratio and normalized units; β -blockade; posture; human subjects

HEART RATE VARIABILITY (HRV) is a measure of the variations in the interbeat intervals (R-R intervals) and is one of the most powerful prognostic factors of mortality following myocardial infarction (16). For the most part, HRV is caused by variations in input to the sinus node from the autonomic nervous system. Multiple mechanisms cause the variation in autonomic activity, including respiration, baroreceptor reflexes, and inputs from higher cerebral centers. Removal of direct autonomic nervous influence to the sinus node nearly abolishes HRV, as seen after cardiac transplantation (3, 23) or after combined β -adrenergic and parasympathetic blockade (1, 21). The result is a highly regular (“metronome-like”) heart rate. The magnitude of HRV is probably more dependent on the modulation of the firing frequency in the nerves than on the mean firing frequency (“nervous tone”), as suggested by Hedman et al. (12). Heart rate depends on the sinus node’s intrinsic rate and the integration of sympathetic and parasympathetic nervous tone. The nature of this integration is not completely understood, but heart rate and HRV are often inversely related, indicating a

relationship between nervous tone and the modulation of the firing frequency.

The variations in heart rate can be separated into different components by use of spectral analysis. A high-frequency component (HF; 0.15–0.4 Hz) is generated by respiratory modulation of vagal nerve activity (1, 17, 21). Slower fluctuations between 0.04 and 0.15 Hz constitute the low-frequency component (LF), which is, to some extent, generated by baroreceptor modulation of sympathetic and vagal nervous tone; LF is believed by some investigators to be a marker of sympathetic nervous tone (17). Variations at frequencies below 0.04 Hz constitute the very low frequency (VLF) and ultralow frequency (ULF) bands. The physiological origin of these very slow fluctuations is still unsettled, but it has been suggested that variations in the activity of the renin-angiotensin system (1) and thermoregulation (15) are of importance. In addition, changes in physical activity may be responsible for some of the HRV in the VLF and ULF bands (4).

It is controversial whether spectral analysis of HRV can be used to determine either changes in the sympathovagal balance or the absolute values of sympathetic and parasympathetic nervous modulation. Most investigators agree that the power of the HF band reflects vagal modulation, when expressed both in absolute power and in normalized units. This view is supported by a study from Berger et al. (2), which showed that modulation of sympathetic firing frequency in the HF area is not reflected in the heart rate power spectrum because the sympathetic receptors in the sinus node act as a low-pass filter with a corner frequency of ~ 0.15 Hz. The controversial issue is whether the LF band reflects sympathetic modulation and whether this modulation is quantifiable by the amount of power in the band. In this connection, the question of normalized units is central. When expressed in normalized units, the LF band appears to reflect sympathetic modulation in several settings, mainly involving sympathetic activation; this is not the case when it is expressed in units of absolute power (18, 20). Whether the low frequency-to-high frequency ratio (LF/HF) is a comprehensive measure of autonomic balance is another controversial question, and the present paper examines this measure in settings with low sympathetic nervous tone.

The effect of sympathetic blockade with β -blockers has also led to conflicting results. In healthy subjects oral atenolol increased both HRV and the powers in the LF and HF bands (6). In postinfarction patients both atenolol and metoprolol increased HRV and the power in the HF band (25). In contrast, oral acebutolol was reported to decrease fluctuations in the LF band (10).



The purpose of the present study was to investigate the dynamic power spectral changes of HRV in two settings with progressive lowering of sympathetic nervous tone and rise in parasympathetic nervous tone. The shift in autonomic balance was achieved by the supine position and by β -blockade with metoprolol. This was motivated by the fact that most of the prior studies have been done in settings in which the sympathetic nervous system was activated. If a comprehensive measure of autonomic balance exists, it should also be useful in settings with vagal predominance.

METHODS

Twelve health care workers (6 male and 6 female), aged between 25 and 38 yr, were included in the study. All of the subjects were healthy as determined by their history and a brief examination; none received any medication. The subjects gave informed consent, and the study was approved by the local ethical review board.

All examinations took place in the morning in quiet surroundings. After the subjects arrived, they sat quietly for 5 min and electrocardiogram (ECG) electrodes were placed. They stood up, walked a few steps to a couch, and assumed the supine position, and the ECG recording was started immediately. The subjects were instructed to stay awake during the entire recording period, and the supine position was maintained for a minimum of 2½ h. The subjects were examined during three conditions: 1) a control period ($n = 12$); 2) acute β -blockade [$n = 11$; metoprolol (Seloken; Astra) was given at a rate of 0.9 mg/min iv for the first 20 min and then at 0.18 mg/min for the remainder of the period, ensuring a fairly constant plasma concentration (22)]; and 3) chronic β -blockade [$n = 10$; metoprolol (SeloZok; Astra) was given for 3 wk in a dose of 100 mg once a day].

Initially, there was the same number of subjects in each group, but because of a computer malfunction some data were lost. This did not represent a selection of subjects and did not influence the result of the study.

ECG recording. Continuous ECG was recorded digitally using a Custo Med R6 ambulatory ECG recorder (Custo Med) with a sampling rate of 500 Hz. After on-line determination of the R-R intervals with an autocorrelation technique for R wave detection, the segment of the digitally recorded ECG was compressed and stored together with the R-R interval for later retrieval. All R-R intervals were manually reviewed, and premature beats and artifacts were removed. In the case of premature beats, the R-R intervals both before and after

the beat were removed. No subject had >10 premature beats/h.

Spectral analysis. For power spectral analysis, a fast Fourier transform with a square window was used. The power spectrum was calculated as the square of the absolute values of the corresponding Fourier transform. For assessments of dynamic changes in HRV, power spectra were computed on series of 512 beats. Consecutive series were initiated at 10-min intervals, so the starting points of the series were 0 min, 10 min, 20 min, and so forth. The power spectra were corrected with respect to the mean heart rate in the actual window, resulting in a frequency unit of equivalent Hertz (eqHz). Because of the relatively short time series, no ULF band was calculated and the VLF band was defined as the power at frequencies <0.04 Hz (24).

Spectral powers were calculated as absolute powers, normalized units, and the coefficient of component variance (CCV; Ref. 11). Normalized units were calculated for the LF and the HF bands as

$$\text{Power}_{nu} = (100 \times \text{absolute power}) / (\text{total power} - \text{VLF})$$

CCV was calculated as the square root of power divided by the mean R-R interval and expressed as a percentage. CCV reduces the correlation between the mean R-R interval and spectral power that can be seen under some circumstances (24).

Statistical analysis. The distributions of spectral powers were skewed to the right and, therefore, were transformed to their logarithms (\log_{10}). For statistical comparisons, heart rate and spectral powers were averaged over the nine 512-beat series in the interval from 60 min to 140 min. The resulting values were compared with the values at 0 min using a two-tailed, paired t -test. In addition, the values at 0 min and the mean from 60 min to 140 min were compared with corresponding values between sessions, also using a two-tailed, paired t -test. A P value of <0.05 was considered statistically significant. Regression and correlation analyses were done using the regression module in Statistica (StatSoft, Tulsa, OK). Values are presented as means \pm SE.

RESULTS

Normalization. As demonstrated in Fig. 1, nearly perfect linear relations exist between the normalized HF power (HF_{nu}) and $1/(1 - \text{LF}/\text{HF})$ and the normalized LF power (LF_{nu}) and $1/(1 - \text{HF}/\text{LF})$, respectively. The regression lines are not significantly different from the line of identity, and the correlation coefficients are

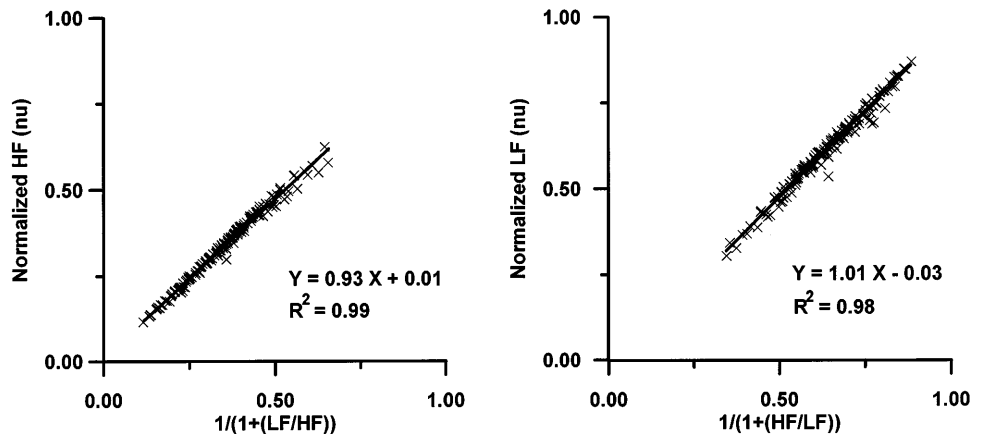


Fig. 1. Relationship between transformed low frequency (LF)/high frequency (HF) index and normalized HF area and normalized LF area. nu, Normalized units.

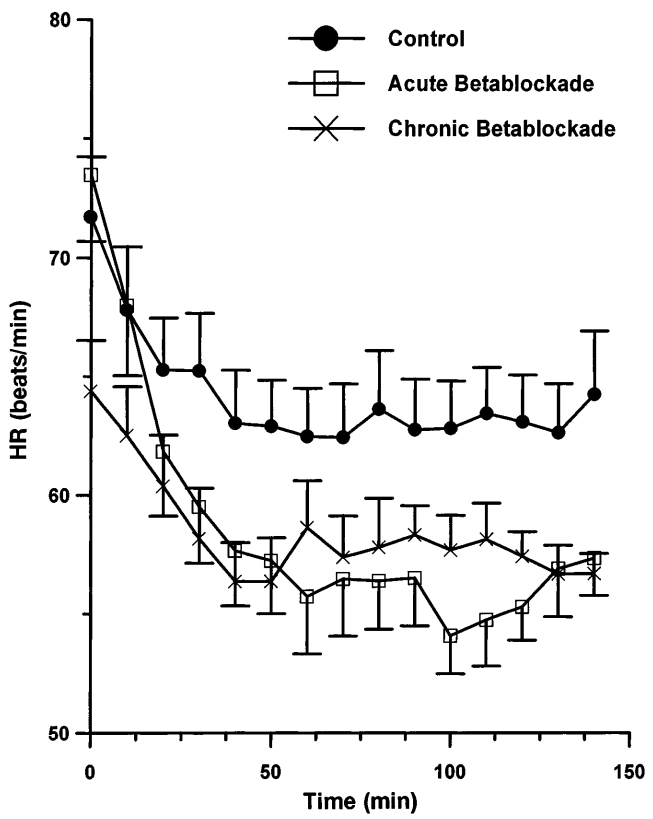


Fig. 2. Mean heart rate during the 3 recording days. Error bars indicate 1 SE.

close to 1 ($r^2 = 0.99$ and 0.98 , respectively). This supports the theoretical arguments, shown in the APPENDIX, that lead to the conclusion that LF/HF, LF_{nu}, and HF_{nu} all contain the same information. Because of this close relationship, only LF/HF will be presented.

Power spectral analysis. Figure 2 shows the time course of heart rate after the subjects were placed in the supine position during all three experimental conditions. The initial heart rate ($t = 0$ min) was significantly lower during chronic β -blockade compared with both control and acute β -blockade (Table 1). Heart rate fell significantly in all three cases and reached a stable value after ~ 40 min. Acute and chronic β -blockade resulted in similar heart rates after 20 min, indicating that, at this point, the full β -blocking effect was reached

during intravenous administration. In both cases, the heart rate was significantly reduced compared with the control condition (Fig. 2 and Table 1).

Chronic β -blockade increased initial heart rate variability as evidenced by an increase in the total power at $t = 0$ min compared with the control condition (Table 1 and Fig. 3). Although the initial powers tended to be higher in all frequency domains (VLF, LF, and HF) during chronic β -blockade, it was only the value for the HF power that was statistically significant (Table 1). This was also true when the powers were expressed as CCV.

Lying down caused an increase in total power and in the powers of all frequency domains (VLF, LF, and HF) in all three conditions. As seen in Fig. 3, a steady state was reached after ~ 60 min, i.e., somewhat later than for the heart rate. The steady-state levels were significantly higher during chronic β -blockade compared with the control condition, but when corrected for the lower heart rate, this only reached statistical significance for the HF power. The same pattern was evident during acute β -blockade, and there were no significant differences between the steady-state values of the various powers during acute and chronic β -blockade.

The supine position was not associated with a redistribution of the initial powers in the HF and the LF bands, as evidenced by an unchanged LF/HF (Table 1). In contrast, β -blockade was associated with a significant decrease in LF/HF during acute β -blockade and a persistence of the lower LF/HF during chronic β -blockade (Fig. 4 and Table 1).

All frequency spectra at 0 and 60 min were manually reviewed to determine the center frequency of the HF peak. In two cases, one during acute β -blockade and one during chronic β -blockade, a distinct HF peak could not be detected. The center frequencies (in Hz) of the HF peak were 0.27 ± 0.01 at 0 min and 0.25 ± 0.01 at 60 min during the control experiment ($n = 12$), 0.27 ± 0.02 at 0 min and 0.25 ± 0.02 at 60 min during acute β -blockade ($n = 10$), and 0.26 ± 0.01 at 0 min and 0.24 ± 0.01 at 60 min during chronic β -blockade ($n = 9$). Pooling the three days revealed averaged center frequencies (in Hz) of 0.27 ± 0.01 at 0 min and 0.25 ± 0.01 at 60 min ($P < 0.01$).

Table 1. Heart rate and spectral parameters for the 3 recording days

	Control (0 min)	Control (60–140 min)	Acute β -Blockade (0 min)	Acute β -Blockade (60–140 min)	Chronic β -Blockade (0 min)	Chronic β -Blockade (60–140 min)
HR, beats/min	72 ± 3	64 ± 2§	73 ± 3	56 ± 2†§	64 ± 2*	58 ± 1†§
log TP, log ms ²	3.25 ± 0.12	3.74 ± 0.07§	3.34 ± 0.13	3.89 ± 0.06*†	3.52 ± 0.12*	3.93 ± 0.05*†
log VLF, log ms ²	2.70 ± 0.10	3.17 ± 0.08§	2.80 ± 0.13	3.33 ± 0.06§	2.88 ± 0.16	3.36 ± 0.07*†
log LF, log ms ²	2.62 ± 0.14	3.09 ± 0.07†	2.72 ± 0.11	3.18 ± 0.09§	2.93 ± 0.14	3.23 ± 0.07*†
log HF, log ms ²	2.47 ± 0.12	2.81 ± 0.06†	2.33 ± 0.14	3.07 ± 0.08†§	2.88 ± 0.13*	3.08 ± 0.07†
CCV _{VLF} , %	4.13 ± 0.52	6.62 ± 0.54§	4.95 ± 0.75	6.65 ± 0.33†	4.48 ± 0.47	7.20 ± 0.36§
CCV _{LF} , %	2.79 ± 0.44	4.00 ± 0.26§	3.00 ± 0.39	3.89 ± 0.32†	3.36 ± 0.41	4.15 ± 0.33†
CCV _{HF} , %	1.84 ± 0.19	2.74 ± 0.19§	2.15 ± 0.32	3.34 ± 0.25*§	2.93 ± 0.39*	3.47 ± 0.27*
LF/HF	2.24 ± 0.33	2.45 ± 0.44	2.17 ± 0.43	1.55 ± 0.24*	1.49 ± 0.37*	1.76 ± 0.38*†

Values are means ± SE; 60–140 min, calculated mean based on the 7 time series of 512 beats from 60 min to 140 min; HR, heart rate; TP, total power; VLF, very low frequency; LF, low frequency; HF, high frequency; CCV, coefficient of component variance. * $P < 0.05$ vs. control; † $P < 0.005$ vs. control; ‡ $P < 0.05$ vs. time 0 min; § $P < 0.005$ vs. time 0 min.

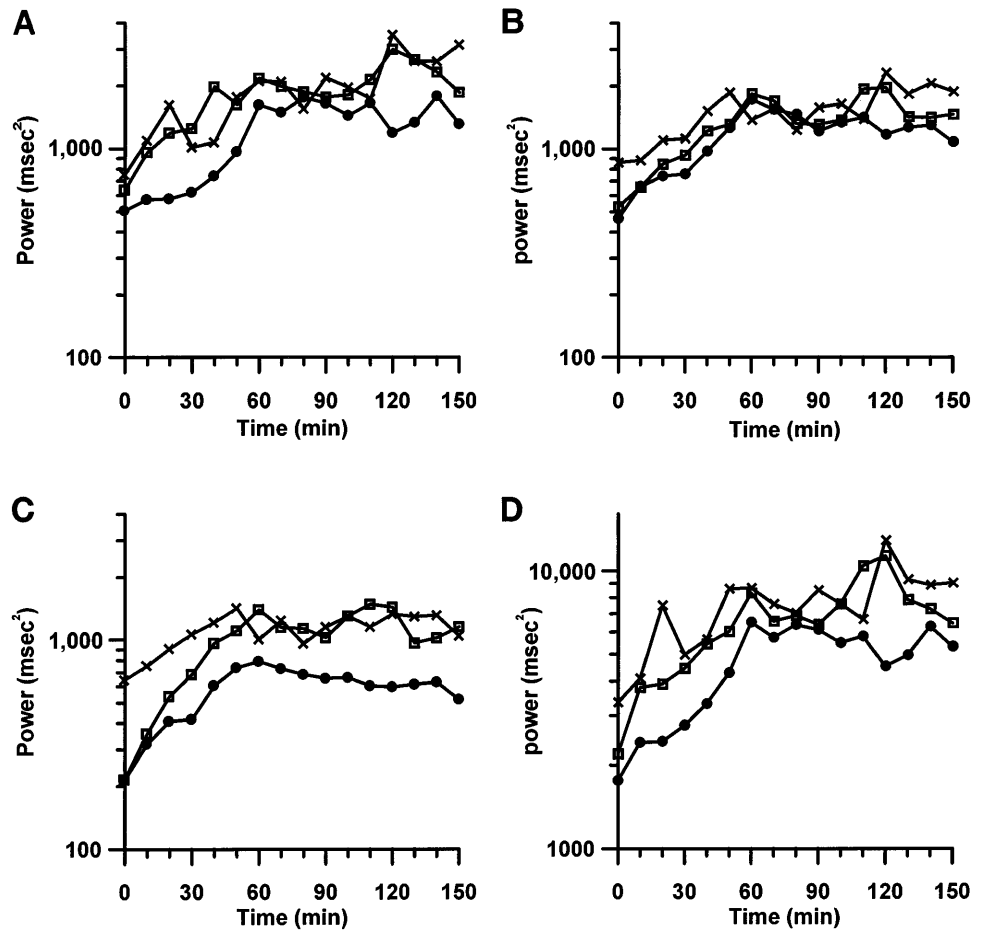


Fig. 3. Dynamic power distribution over time for very low frequency (VLF) area (A), LF area (B), HF area (C) and total power (D) during the 3 recording days. ●, Control; □, acute β -blockade; ×, chronic β -blockade.

DISCUSSION

Previous studies have indicated that a shift in the autonomic balance was associated with a redistribution of the power between the LF and the HF bands, and it was suggested that normalized power units (LF_{nu} and HF_{nu}) or LF/HF were efficient means for detecting shifts in autonomic balance (17, 24). This could not be confirmed in the present study. When the subjects were placed in the supine position, autonomic balance was progressively altered, as evidenced by the marked reduction in heart rate, which can only be explained by a shift in the autonomic balance in favor of the parasympathetic nervous system. Both during the control recording and during the chronic β -blockade recording, HRV and LF and HF powers all increased but LF/HF did not change significantly. This demonstrates that the LF/HF index is not a consistent measure of autonomic balance under these conditions.

Previous studies that observed a shift in LF and HF powers used experimental protocols that led to increased sympathetic nervous tone and decreased vagal tone (17, 18, 20). In these studies heart rate increased, and this was associated with a large reduction in HF power and no or only a small decrease in LF power and thus a rise in the LF/HF index. These observations led to the suggestion that the LF/HF index could be used as an indicator of autonomic balance. In the present study, we failed to find any change in the LF/HF index when

the experimental subjects were placed in the supine position. There could be several reasons for this discrepancy. The LF/HF index during the control recording is of the same magnitude as the LF/HF index during rest in the earlier studies (18, 20). It could therefore be argued that the assumption of the supine position, before the start of the recordings, had already caused a change in the autonomic balance compared with the standing position. Although this may have occurred to some extent, it is clear that a significant change in autonomic balance takes place during the recording period, as evidenced by the considerable decrease in heart rate.

A second possibility is that assumption of the supine position caused a decrease in the respiratory frequency. If the respiratory frequency decreases to values below ~ 9 breaths/min, part of this activity moves into the LF area. This would increase LF power and decrease HF power and thus could mask the change in LF/HF caused by changes in autonomic balance. Although we did not measure respiratory frequency in the experimental subjects, this possibility seems unlikely because the change in the center frequency of the HF area was small. Previous studies have shown that the center frequency of the HF area correlates closely with the respiratory frequency (19). Thus it appears unlikely that there should have been a major decrease in the respiratory frequency in the supine position.

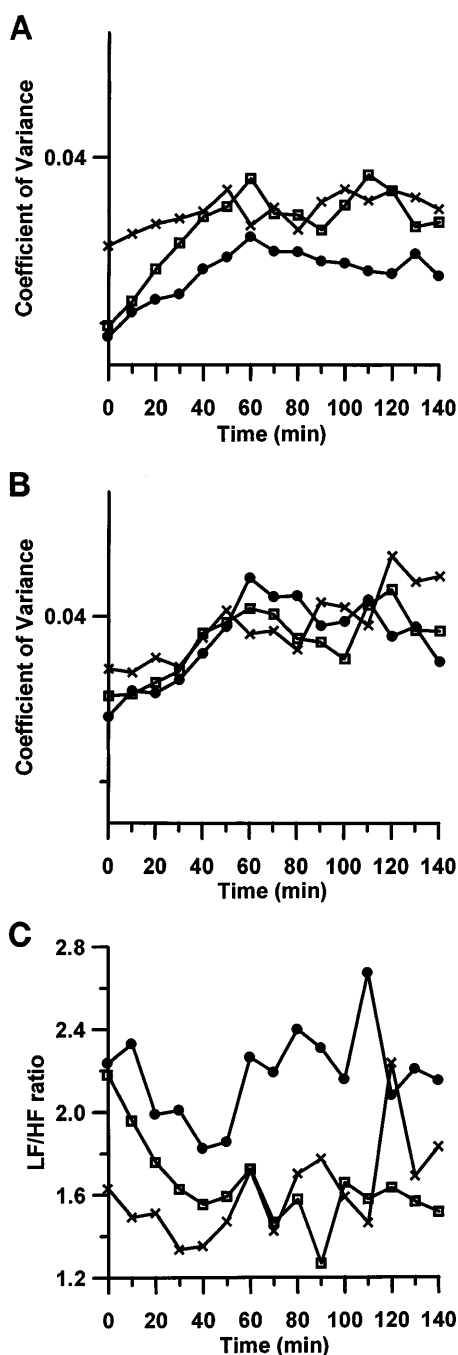


Fig. 4. Development in coefficient of component variance for HF area (CCV_{HF}; A) and LF area (CCV_{LF}; B) and LF/HF index (C) during the 3 recording days. ●, Control; □, acute β -blockade; ×, chronic β -blockade.

Most likely, the sympathetic activity is quite low at the start of the recording period in the present study. A third reason for the apparent discrepancy between this and the previous studies could therefore be that “noise” and the increased low-frequency modulation caused by increased vagal activity mask the expected decrease in the LF band. However, despite these considerations, it is clear from the present results that it is possible to have a marked change in the balance between the tones of the sympathetic and parasympathetic nervous sys-

tems without a change in the ratio or the distribution of the powers in the LF and HF bands.

It was only when the positional change was combined with acute β -blockade that the expected shift in LF/HF was observed in the present study. However, a closer inspection of the data reveals that this was not caused by a change in the LF band but rather by changes in the HF band. During acute β -blockade, the power in the LF band followed virtually the same course as during the control experiment, whereas the power in the HF band showed a greater increase. The increased HF power was also present after chronic β -blockade with no significant change in LF power. If sympathetic activity contributes to the power in the LF band, but not in the HF band, it is difficult to explain these findings as the result of sympathetic withdrawal. Rather, it appears that β -blockade with metoprolol enhances vagal modulation, preferentially at the respiratory frequency (the HF band). This could be through a central effect, because metoprolol is lipid soluble and therefore readily passes through the blood-brain barrier. However, a similar observation was made with the water-soluble drug atenolol (6), which scarcely passes through the blood-brain barrier (9). This observation makes it less likely that the effect is caused by a central action of the drug. Because the sinus node, at least to some extent, acts as a low-pass filter of vagal modulation (2), a second possibility is that sympathetic activity influences the filtering characteristics of the sinus node. Thus we speculate that acute β -blockade may lower the time constant of the sinus node and thereby enhance vagally mediated frequencies >0.15 Hz. Clearly, this speculation requires experimental verification.

Interestingly, the complete effect of β -blockade on heart rate appears to be achieved after only 20–30 min. During both acute and chronic β -blockade, heart rate was similar at this point in time.

As is the case with many other indirect physiological measures, it seems that there are limitations to the use of LF/HF as a marker of autonomic balance. This is not surprising, considering that the rationale for using LF/HF is that LF fluctuations are caused by the combined action of the sympathetic and the vagal nerves, whereas HF fluctuations are caused only by vagal activity. A priori, one would therefore expect that LF/HF was of little use in detecting decreases in sympathetic activity in settings in which the sympathetic tone is already low. The present study confirms this expectation. Also, in settings with high sympathetic tone but without specific enhancements of LF fluctuations in the sympathetic nerves, one would not expect LF/HF to be useful in assessing autonomic balance. This has recently been confirmed in a study by Hoshikawa and Yamamoto (13). On the other hand, it is well known that LF/HF can detect large, oppositely directed, and preferably baroreceptor-mediated shifts in sympathetic and vagal tone (20). However, as this and other studies have shown, there are limitations for its general use as a marker of sympathetic-parasympathetic balance. Because it is impossible to predict whether LF/HF will work as a marker for autonomic



balance in a given experimental or clinical situation, it is advisable always to test its usefulness under the appropriate experimental or clinical conditions.

The results of the present study show that it takes almost an hour to achieve stationarity in the heart rate and HRV after a change from the standing to the supine position. This is much longer than the equilibration period normally used in studies that examine the effect of positional changes on HRV. Because stationarity is a requirement in spectral analysis, the slow rise in HRV calls for caution when examining power spectra from short-term HRV analysis. It also stresses the importance of stating the conditions (time from lying down, length of time series, etc.) under which an R-R time series has been obtained so that results can be compared between laboratories.

Because it was outside the scope of the present study to investigate the physiological mechanisms that underlie the changes in heart rate and HRV, no hemodynamic variables besides heart rate were measured. There can be little doubt, however, that the changes in hemodynamics associated with achieving the supine position involve a movement of blood to the central veins, leading to an increased venous return to the heart. This triggers low-pressure baroreceptors. Distension of the ventricles also leads to an increased cardiac output by the Frank-Starling mechanism, which in turn triggers arterial baroreceptors. Both mechanisms will lead to lower sympathetic activity and cause vagal activation.

On longer timescales possible mechanisms that could influence the HRV would be blood pressure-regulating systems working on relevant timescales (<1 h), e.g., the renin-angiotensin system, vasopressin, or the capillary fluid shift mechanism. Vasopressin modifies the baroreceptor response, but there are no studies investigating its effect on HRV (8). The effect of the renin-angiotensin system on HRV is not well understood, either. In dogs, angiotensin-converting enzyme inhibition (ACE-I) increased VLF power (1); in humans, the results with ACE-I have been conflicting. Enalapril had no influence on HRV in healthy subjects (14), but HRV increased during treatment of patients with congestive heart failure (26). In patients with congestive heart failure, zofenopril increased total power with 50% and HF power with 200%, suggesting a major role of the renin-angiotensin system in regulating HRV (5). On timescales of minutes to one hour, these hormones could influence HRV through modulation of the baroreceptor response. The fluid shift mechanism causes a net distribution of fluid into the vessels in the supine position, and the effect on HRV would be mediated through baroreceptor responses.

Several normalization procedures have been suggested in earlier studies (11, 20). Normalization of spectral powers by the method of coefficient of variance is done to minimize the dependence on heart rate. In the present study, the result from this procedure was no different from what was seen by examining absolute powers. Normalized units were implemented as a means of explaining power spectral changes in relation to

known physiological changes. As seen in the APPENDIX, theoretically, the normalized powers contain no additional information compared with LF/HF. This is also confirmed experimentally, because an almost perfect correlation exists between the transformed LF/HF and the normalized powers. The only parameter that could confound the transformation of LF/HF to normalized units is the power above 0.4 Hz. Thus, in conditions with very low HRV, there could be a decreased correlation between the normalized powers and the transformed LF/HF because the noise in the area above 0.4 Hz will have relatively more weight in the power spectrum. However, the power above 0.4 Hz has no known relation to changes in the sympathovagal balance.

The present study demonstrates that LF/HF is not a comprehensive and consistent measure of autonomic balance. In a setting with low sympathetic tone and weak modulation by the sympathetic nervous system, LF/HF fails to detect shifts in autonomic balance. Normalized powers describe a balance rather than a modulation from individual limbs of the autonomic nervous system, and they reveal no additional information compared with the LF/HF index. Lying down leads, in itself, to a progressive increase in total power and the powers of the VLF, LF and HF bands, an increase that continues for 1 h before a plateau phase is reached. Metoprolol increases total power and HF power and enhances vagal modulation at the respiratory frequency.

APPENDIX

Because TP is the total power from 0 to 0.5 Hz, it can be seen that

$$TP - VLF = LF + HF + P_{>0.4\text{Hz}} \quad (A1)$$

where $P_{>0.4\text{Hz}}$ is the power in the area above 0.4 Hz. Because HRV resembles fractal Brownian motion (a $1/f$ process), the amount of power above 0.4 Hz is negligible. Thus, $P_{>0.4\text{Hz}} \approx 0$

$$LF_{\text{nu}} = \frac{LF}{TP - VLF} \approx \frac{LF}{LF + HF} \quad (A2)$$

$$HF_{\text{nu}} = \frac{HF}{TP - VLF} \approx \frac{HF}{LF + HF} \quad (A3)$$

By dividing LF into the nominator and denominator in Eq. A2 and dividing HF into the nominator and denominator in Eq. A3 we get

$$LF_{\text{nu}} \approx \frac{1}{1 + \frac{HF}{LF}} \quad HF_{\text{nu}} \approx \frac{1}{1 + \frac{LF}{HF}} \quad (A4)$$

which leads to the conclusion that normalized power is just a simple monotone transformation of LF/HF and contains no additional information compared with LF/HF. Setting $f(x) = 1/(1 + x)$, we have

$$LF_{\text{nu}} \approx f\left(\frac{HF}{LF}\right) \quad HF_{\text{nu}} \approx f\left(\frac{LF}{HF}\right) \quad (A5)$$



Because $f(x)$ is a monotonically decreasing function of x (for $x > 0$), it follows that if LF_{nu} increases there is a decrease in HF_{nu} and vice versa. Thus the use of normalized units could be replaced by LF/HF .

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