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Heart Rate Power Spectrum and Plasma Catecholamine Levels after Postural Change and Cold Pressor Test

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ABSTRACT

During stress, low-frequency (0.01–0.15 Hz) heart rate power and plasma catecholamine levels increase in response to increased sympathetic efferent activity. To test the hypothesis that low-frequency heart rate power, a measure of sympathetic control of heart rate, directly correlates with plasma catecholamine concentrations during periods of increased sympathetic tone, we compared heart rate power spectral measures with antecubital vein norepinephrine, epinephrine, and dopamine concentrations during postural change and after cold pressor testing. We analyzed absolute levels and changes in mean heart rate, respiratory rate, blood pressure, heart rate power spectra, and concentration of norepinephrine, epinephrine, and dopamine in 14 healthy volunteers (seven female/seven male) after postural change and in six (three female/three male) during cold pressor testing. Postural change from supine to standing position resulted in increased heart rate [61 ± 8 *versus* 83 ± 11 (SD) bpm, $p < 0.05$], diastolic (68 ± 7 *versus* 77 ± 6 mm Hg, $p < 0.05$) and mean blood pressures (84 ± 6 *versus* 91 ± 9 mm Hg, $p < 0.05$), norepinephrine concentration (2.09 ± 1.11 *versus* 3.23 ± 1.62 nmol/L, $p < 0.05$), and low-frequency heart rate power (7.55 ± 5.63 *versus* 33.79 ± 23.55 bpm^2, $p < 0.05$). High-frequency heart rate power, a measure of parasympathetic control of heart rate, decreased with standing (5.38 ± 4.22 *versus* 2.94 ± 2.69 bpm^2, $p < 0.05$). Diastolic (66 ± 7 *versus* 81 ± 9 mm Hg, $p < 0.05$) and mean (83 ± 9 *versus* 97 ± 11 mm Hg, $p < 0.05$) blood pressures and norepinephrine concentration (1.21 ± 0.40 *versus* 1.77 ± 0.79 nmol/L, $p < 0.05$) increased with cold pressor testing. We found no correlation between absolute levels or changes in low-frequency heart rate power and norepinephrine, epinephrine, or dopamine concentration. Thus, we conclude that low-frequency heart rate power and plasma catecholamines are significantly affected by physiologic changes but are likely regulated by different areas within the sympathetic nervous system. ***(Pediatr Res* 36: 358–363, 1994)**

Abbreviations

DA, dopamine
[DA], dopamine concentration
E, epinephrine
[E], epinephrine concentration
FFT, fast Fourier transform
NE, norepinephrine
[NE], norepinephrine concentration

Power spectral analysis of heart rate variability is a noninvasive measure of both sympathetic and parasympathetic control of heart rate (1–8) and has been used to assess normal physiologic and pathophysiologic changes in autonomic control of the cardiovascular system (1–12). Low-frequency heart rate power (0.01–0.15 Hz) measures combined sympathetic and parasympathetic modulation of heart rate at rest with sympathetic modulation predominating during stressful conditions (5). Plasma levels of NE, E, and DA are reported to correlate with mean sympathetic activity (13–15). We hypothesized that under physiologic conditions in humans that produce increased sympathetic tone, such as those induced by postural change to the standing position or the cold pressor test, there would be a direct correlation between low-frequency heart rate power and plasma catecholamine concentrations. To investigate this hypothesis, we compared heart rate power spectral measures with plasma levels of NE, E, and DA in 14 healthy volunteers. Our results showed no correlation between low-frequency heart rate power and plasma catecholamine levels during stress, suggesting that postural change and cold pressor testing result in differential activation of the sympathetic nervous system.

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METHODS

Subjects. Fourteen healthy volunteers (seven female/seven male) (mean age ± SD = 26.1 ± 2.9 y; range = 20–30 y) were studied after postural change from supine to standing positions. Six volunteers (three female/three male) (mean age = 25.7 ± 3.8 y, range = 21–30 y) were also studied during the cold pressor test.

This study was approved by the University of Rochester Research Subjects Review Board, and informed consent was obtained from all subjects studied. Exclusion criteria included subjects with a history of smoking, with cardiovascular disease, or who were taking medications containing atropine or atropine-like drugs, phosphodiesterase inhibitors, or β-adrenergic antagonists.

Procedure. On admission to the University of Rochester's Clinical Research Center after an overnight fast, the subject was placed in the supine position in a quiet and darkened room. A 20-gauge i.v. catheter (Insyte 3875201, Becton Dickinson, Sandy, UT) was placed in the antecubital vein of each subject and flushed with a 1:1000 heparin solution. Limb lead electrodes (Medtronic 1700-001, Haverhill, MA) were placed in standard fashion for lead II ECG recording. After a 30-min period to allow for hemodynamic equilibration, basal heart rate, respiratory rate, blood pressure, and lead II ECG were recorded. ECG and the impedance respiratory signal were recorded with Hewlett-Packard monitors (models 78213C and 78212D, Hewlett-Packard, Palo Alto, CA) and digitally sampled at 1 kHz. Systolic, diastolic, and mean blood pressure were recorded with a Dinamap Vital Signs Monitor (model 845XT, Critikon, Inc., Tampa, FL). Power spectral data were derived offline from a 256-s lead II ECG recording. Three mL of blood for plasma catecholamine levels were drawn through the i.v. catheter at the conclusion of the ECG recording. Subjects then stood quietly for 15 min to allow for equilibration and ECG and respiratory signals were recorded and blood samples obtained as above. The subjects resumed the supine position, and after another 15-min equilibration period, cold pressor testing was performed.

Cold pressor testing. Cold pressor testing was accomplished by immersion of one hand in ice water for 4 min. Vital signs and power spectral data were recorded for the last 128 s of immersion. Blood for plasma catecholamines was obtained at the conclusion of the cold pressor test.

Heart rate power spectral analysis. We modified the methodology described by Akselrod *et al.* (1) for determining power spectral density of heart rate variability. The ECG and impedance respiratory signals were recorded for 256 s from a standard lead II ECG using Hewlett-Packard monitors models 78213C and 78212D with a low-pass filter at 100 Hz. Data were collected and analyzed using an AST Premium 386/25 PC in conjunction with a Data Translation 2801A data acquisition board. Sampling rate for data collection was done at 1 kHz. Separate spectral analysis of the raw ECG wave forms using a Hewlett-Packard 3563A Systems Analyzer revealed no frequency content > 480 Hz; thus, sampling at 1 kHz was sufficient to meet Nyquist sampling criteria. ECG R waves were detected and a nominal 128-s instantaneous heart rate series was constructed that was artifact-free and exhibited no linear drift in mean heart rate by visual inspection (*i.e.* no mean heart rate ramp effect). A heart rate variability data set was obtained by subtracting the average heart rate from the instantaneous heart rate tachogram followed by linear interpolation to 4096 equispaced data points suitable for FFT. A Hanning window was applied to each 4096-point time series. The time series was multiplied by a factor of 2 to compensate for signal attenuation due to the Hanning window. FFT was performed and the magnitude of the resultant FFT was squared to obtain the absolute power spectrum. Data acquisition and analysis algorithms were coded in ASYST data acquisition and analysis software (Keithly-Asyst, Inc., Rochester, NY).

The absolute power spectrum is equivalent to the (normalized power spectrum) × (mean heart rate)^2, with recognition that the normalized direct current value is unity. All power spectral data in this study are presented as absolute power spectral values with the direct-current component removed to focus on the alternating-current attributes of the signal.

Low-frequency heart rate oscillations are mediated by combined sympathetic and parasympathetic activity at rest (2, 3, 5–7), whereas sympathetic activity predominates during stressful conditions (5–7). The total power from 0.01 to 0.15 Hz was used to quantify sympathetically mediated heart rate oscillations (low-frequency power) during stress (5–7). The low-frequency bandwidth can be further divided into very low-frequency (0.01–0.04 Hz) and mid low-frequency (0.04–0.07 Hz) bandwidths. Heart rate power at 0.01–0.04 Hz correlates with changes in blood pressure, blood volume, and sympathetic nerve activity, whereas changes in respiratory, phrenic nerve, and sympathetic nerve activity correlate with heart rate power between 0.04 and 0.07 Hz (5, 7, 8). Integrated total power (area) at high frequencies (0.15–0.50 Hz) was used as a measure of parasympathetically mediated respiratory sinus arrhythmia (high-frequency power) (1, 2, 5). High-frequency oscillations are solely under parasympathetic control (1, 5, 7). The location of the respiratory frequency was confirmed by spectral analysis of the impedance respiratory signal obtained from the electrocardiographic leads (Fig. 1).

Plasma catecholamine levels. Samples for plasma catecholamines were drawn from indwelling venous catheters. Samples were stored at −80°C until assayed by a modification of the radioenzymatic technique of Peuler and Johnson (16). The corresponding intraassay and interassay coefficients of variation were, respectively, 7.7 and 16.8% for [NE], 6.9 and 14.4% for [E], and 6.8 and 13.0% for [DA]. Normal values for our laboratory are [NE] < 2.64 nmol/L, [E] < 390 pmol/L, and [DA] < 0.59 nmol/L (17).

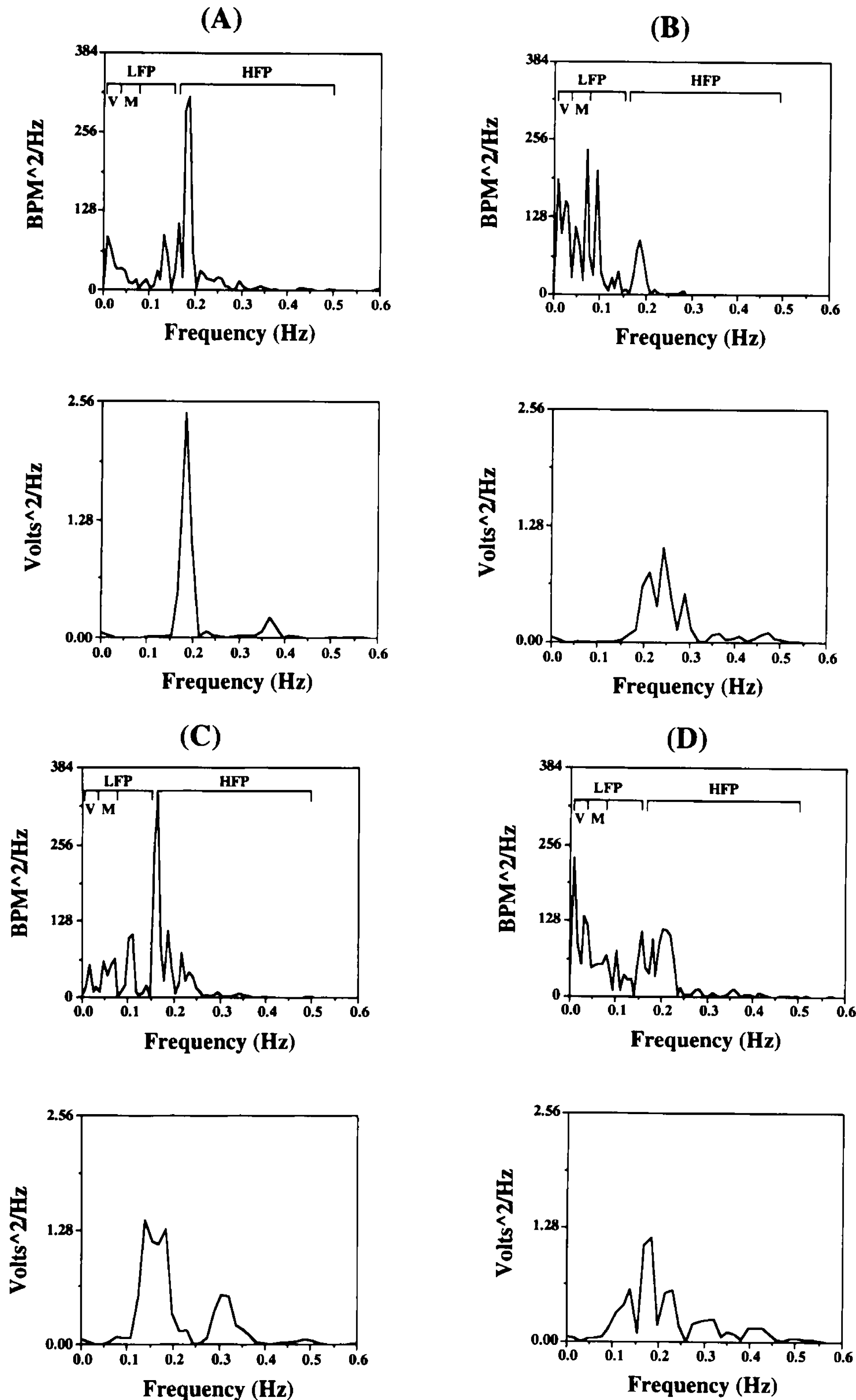

Figure 1. Heart rate and respiratory rate power spectra for a subject in the supine (*A*) and standing (*B*) positions, after resumption of the supine position (*C*), and during cold pressor testing (*D*). All spectra contain a peak at the respiratory frequency (~0.2 Hz). Very low-frequency (0.01–0.04 Hz), mid low-frequency (0.04–0.07 Hz), and low-frequency (0.01–0.15) peaks may be distinguished in some spectra (*C*). Note the decrease in high-frequency power associated with standing (*B*) and the increase in low-frequency power after standing and cold pressor testing (*B* and *D*). Power at the respiratory bandwidth is broadened with both standing and cold pressor testing (*V*, very low-frequency heart rate power; *M*, mid low-frequency heart rate power; *LFP*, low-frequency heart rate power; *HFP*, high-frequency heart rate power).

Statistical analysis. Data were analyzed using mean, standard deviation, standard error, paired t tests, and signed-rank test. When appropriate, data were logarithmically transformed because of the 10 000-fold range of values and resultant abnormal distributions. Differences were considered significant at $p \leq 0.05$.

RESULTS

The mean heart rate, respiratory rate, blood pressure, heart rate power spectra data, and plasma catecholamine levels in the supine and standing positions; after resumption of the supine position; and during cold pressor testing are shown in Table 1.

Heart rate and diastolic and mean blood pressure increased after postural change. Cold pressor testing increased diastolic and mean blood pressure, whereas heart rate and systolic pressure tended to increase but the differences did not reach statistical significance. Respiratory rate did not significantly change during the study.

There was an increase in mean heart rate power at all low-frequency bandwidths with postural change that was not seen with cold pressor testing. There was a trend toward an increase in very low-, low-, and high-frequency power with cold pressor testing (Table 1, Fig. 1).

Plasma [NE] increased but neither [E] nor [DA] increased significantly after both stimuli (Table 1). Mean [E] levels doubled upon standing, but this difference did not reach statistical significance due to the large variance.

Heart rate and mid low-frequency and low-frequency heart rate power were greater after standing than during cold pressor testing (Table 1, Fig. 2). High-frequency heart rate power significantly decreased with standing but tended to increase with cold pressor testing, although this difference again did not reach statistical significance (Table 1, Fig. 2). The increase in [NE] tended to be greater after postural change (Δ [NE] postural change = 1.15 ± 0.55 SEM *versus* Δ [NE] cold pressor = 0.57 ± 0.33 nmol/L, $p = 0.058$).

We found no correlation between either absolute levels or changes in heart rate power at any low-frequency bandwidth and plasma [NE, E, or DA] at rest, after postural change, or during the cold pressor test. There was also no correlation between heart rate, respiratory rate, blood pressure, heart rate power spectra, or plasma catecholamine levels with gender or age.

DISCUSSION

We found no association between low-frequency heart rate power and plasma catecholamine concentrations at rest in healthy human volunteers. These findings are consistent with previous reports (5, 6) that low-frequency heart rate power does not solely reflect modulation of sympathetic efferent cardiac activity at rest but rather combined parasympathetic and sympathetic activity. Saul *et al.* (5) reported no association between heart rate variability and muscle sympathetic activity, plasma [NE], mean heart rate, or blood pressure at rest. They suggested a model in which low-frequency heart rate oscillations are dependent on both sympathetic and parasympathetic efferent input to the heart (5, 6) and concluded that low-frequency heart rate oscillations do not accurately reflect mean cardiac sympathetic activity (5). Using a different study paradigm, our results support this conclusion.

Previous studies suggest that an increase in low-frequency power from a resting to a stressed state represents an increase in sympathetic modulation of heart rate as long as there is no change in respiratory rate and high-frequency power remains unchanged or decreases (6, 18–20). Therefore, both plasma catecholamine and low-frequency heart rate spectral values represent quantitative sympathetic responses to stress. Saul *et al.* (5) showed a direct correlation between the fraction of heart rate power at low frequencies and plasma [NE] during increased muscle sympathetic activity in adult males. Although we found no association between absolute levels or changes in low-frequency power and plasma catecholamine concentrations during postural change or cold pressor testing, we speculate that this is due to differential activation of the sympathetic nervous system in response to different stressful stimuli. It seems clear from our results that the type of stressful stimulus influences the nature of the sympathetic response in both a quantitative and qualitative fashion.

Table 1. *Cardiorespiratory, heart rate power spectral, and catecholamine changes during postural changes and after cold pressor testing**

	Supine	Standing	Supine 2	Cold pressor
n	14	14	6	6
HR (bpm)	61 ± 8	83 ± 11†	58 ± 6	64 ± 7
RR (per min)	16 ± 3	17 ± 4	13 ± 3	16 ± 9
Blood pressure (mm Hg)				
Systolic	114 ± 6	116 ± 10	118 ± 14	130 ± 13
Diastolic	68 ± 7	77 ± 6†	66 ± 7	81 ± 9‡
Mean	84 ± 6	91 ± 9†	83 ± 9	97 ± 11‡
Very low-frequency heart rate power (0.01–0.04 Hz) (bpm^2)	2.30 ± 1.66	13.95 ± 14.34†	4.48 ± 5.38	5.76 ± 4.99
Mid low-frequency heart rate power (0.04–0.07 Hz) (bpm^2)	1.15 ± 1.15	4.61 ± 4.99†	2.18 ± 2.05	1.54 ± 1.15
Low-frequency heart rate power (0.01–0.15 Hz) (bpm^2)	7.55 ± 5.63	33.79 ± 4.33†	11.90 ± 0.64	20.86 ± 18.94
High-frequency heart rate power (0.15–0.5 Hz) (bpm^2)	5.38 ± 4.22	2.94 ± 2.69†	4.86 ± 3.58	8.58 ± 8.32
Norepinephrine (nmol/L)	2.09 ± 1.11	3.23 ± 1.62†	1.21 ± 0.40	1.77 ± 0.79‡
Epinephrine (pmol/L)	150 ± 90	300 ± 280	90 ± 60	100 ± 80
Dopamine (nmol/L)	0.22 ± 0.16	0.26 ± 0.22	0.13 ± 0	0.13 ± 0

* Data are expressed as mean± SD. HR, heart rate; RR, respiratory rate.
† $p < 0.05$ compared with supine values.
‡ $p < 0.05$ compared with supine 2 values.

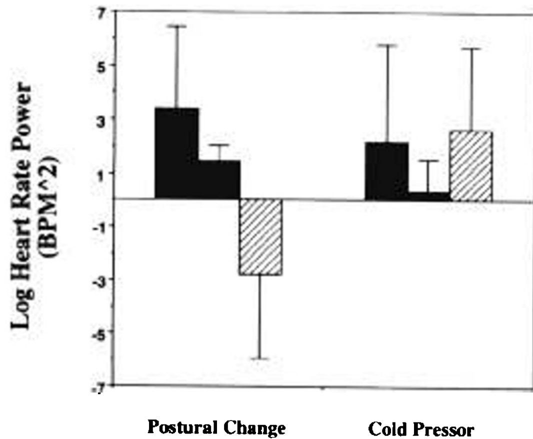

Figure 2. Comparison of changes in heart rate power at mid low-frequency (■), low-frequency (▧), and high-frequency (▨) bandwidth after postural change and during cold pressor testing ($p < 0.05$).

Differential activation of the sympathetic nervous system in response to stress has been reported in various pathophysiologic states (21–23). Measurement of low-frequency heart rate power reflects second-to-second control of heart rate. The anatomic pathways include the entire sympathetic feedback loop between the heart and CNS. Plasma catecholamines are secreted from several anatomic sites and may alter heart rate based on longer time constants. Epinephrine is released into venous blood primarily from the adrenal gland, whereas plasma NE (>90%) reflects spillover from synaptic clefts throughout the body (16, 24, 25). Recent evidence suggests that DA participates in the general sympathetic response to stress only when the adrenal component is maximally stimulated, such as in severe brain injury (26). Therefore, if heart rate power spectral measurements and plasma catecholamine levels reflect activation of different anatomic pathways within the sympathetic nervous system, it is not surprising we found no direct correlation between them. Figure 3 depicts a model explaining the factors involved in autonomic control of heart rate. Postural change from supine to standing position results in activation of both sympathetic input to the heart and

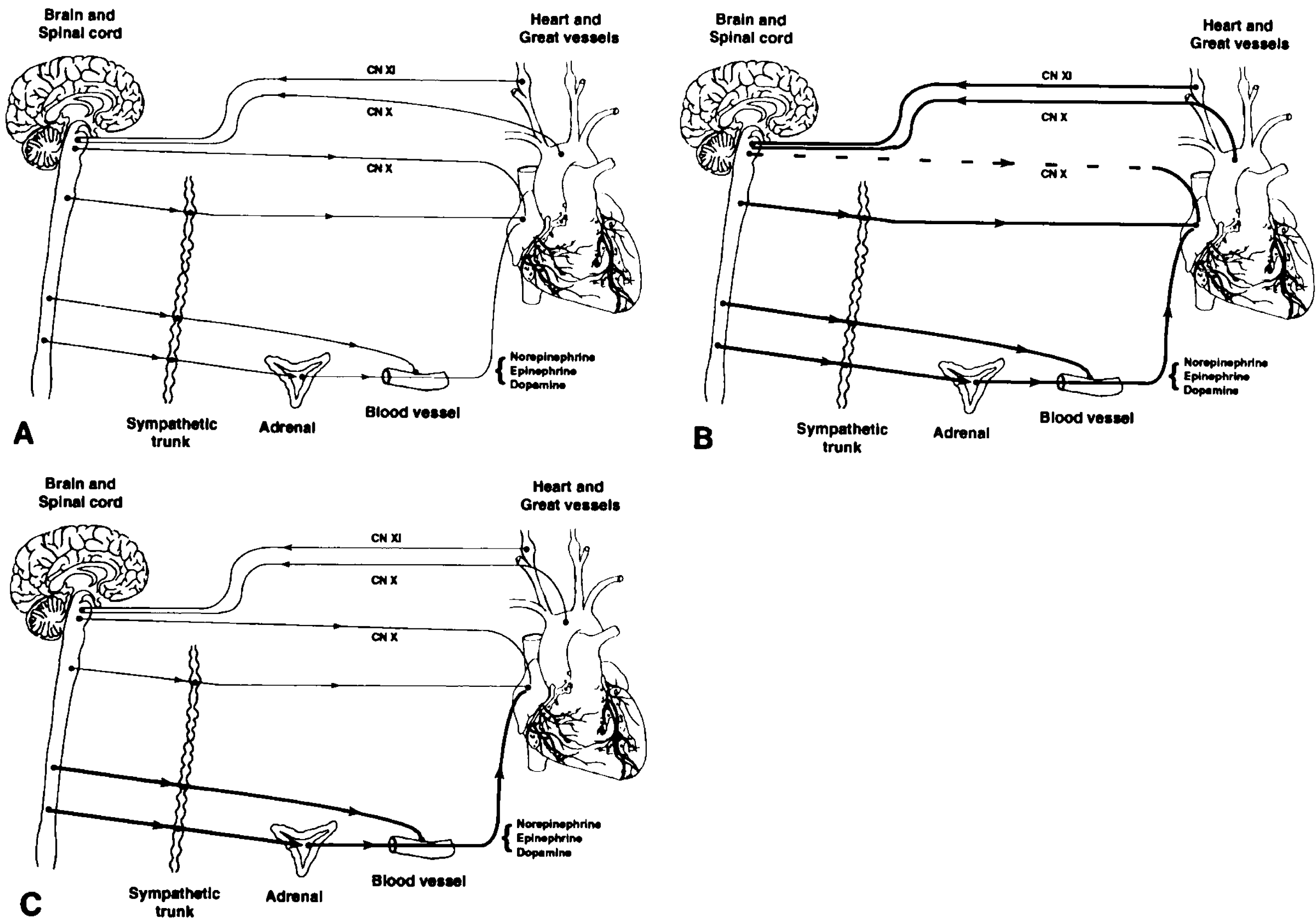

Figure 3. Schematic diagram of autonomic control of heart rate. *A*, Normal resting conditions. Sympathetic efferent (→) and afferent (←) nerve fibers travel to and from the CNS and the heart, respectively. Parasympathetic efferent cardiac fibers go mainly to the sinoatrial and atrioventricular nodes, whereas parasympathetic afferent fibers travel from baroreceptors in the aortic arch via cranial nerve X and the carotid bodies via cranial nerve XI. Catecholamines are released from the adrenal medulla and arterial blood vessels as a result of sympathetic efferent stimulation. Epinephrine is released into venous blood primarily from the adrenal gland and norepinephrine primarily as spillover from the synaptic cleft between sympathetic nerve fibers and arterioles. *B*, Postural change from supine to standing position results in activation of both sympathetic efferent input to the heart and adrenal (*bold line*) as well as inhibition of parasympathetic efferent input to the heart (*dashed line*). *C*, Cold pressor testing results in an increase in sympathetic efferent input to the adrenal (*bold line*) with no significant effect on autonomic efferent cardiac signals.

adrenal as well as inhibition of cardiac parasympathetic efferent pathways. Cold pressor testing increases adrenal sympathetic stimulation with no significant effect on autonomic efferent cardiac signals. Thus, the model in Figure 3 adequately explains the cardiovascular changes observed in our subjects.

There may be alternative explanations for the lack of correlation between heart rate power spectral values and plasma catecholamine levels other than differential activation of anatomic pathways within the autonomic nervous systems. Modulation of sympathetic control of heart rate may not equal mean sympathetic activity (7) as reflected by plasma catecholamine concentration. Also, although low-frequency power during stress reflects sympathetic control of heart rate, changes in low-frequency power may also be due to decreases in parasympathetic modulation of heart rate. Finally, heart rate power spectra and plasma catecholamine concentrations are continuous functions, and sampling at discrete times may not adequately represent changes in these phenomena.

Limitations of the study. The major limitation of this study is the large variance in the data. However, because the variance in measuring heart rate power is indirectly proportional to the sampling interval, statistically significant changes at relatively short sampling periods (*i.e.* 128 s) must be valid, but the possibility of false-negatives remains. It is possible that small changes in heart rate control associated with cold pressor testing were not detected. Further examination of this relationship using other techniques or a larger study group to exclude type II errors may be warranted.

We conclude that during periods of increased sympathetic tone, there is no direct correlation between low-frequency heart rate power and plasma catecholamine concentrations. Our data suggest heart rate power spectral analysis may be used to quantitatively evaluate concurrent changes in sympathetic and parasympathetic control of heart rate during physiologic stress but should not be used as a substitute for catecholamine determination for evaluation of mean sympathetic activity.

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