Effects of systemic hypoxia on R-R interval and blood pressure variabilities in conscious rats

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Murasato, Yoshinobu, Haruhisa Hirakawa, Yuji Harada, Tadashi Nakamura, and Yoshiaki Hayashida. Effects of systemic hypoxia on R-R interval and blood pressure variabilities in conscious rats. Am. J. Physiol. 275 (Heart Circ. Physiol. 44): H797–H804, 1998.—The effects of systemic hypoxia with different levels of CO₂ on R-R interval (RRI) and systolic blood pressure (SBP) variabilities were investigated in conscious rats. Wistar rats chronically instrumented for the measurement of blood pressure, electrocardiogram, and renal sympathetic nerve activity (RSNA) were exposed to hypocapnic (Hypo), isocapnic (Iso), and hypercapnic (Hyper) hypoxia. On another day, the rats were treated with atropine and exposed to the same type of hypoxia. Sinoaortic denervation (SAD)-treated rats were exposed to Iso and Hyper, and RRI and SBP variabilities before and during hypoxia were analyzed using the maximum-entropy method with high resolution. With regard to RRI variability, very low frequency (VLF), low frequency (LF), and high frequency (HF) powers all decreased during Hyper, increased during Hyper, and did not change during Iso in intact rats. Changes during Hypo were attenuated by atropine, and those during Hyper were abolished by either atropine or SAD. The ratio of LF power to HF power decreased independently of increases in RSNA during each type of hypoxia. On the other hand, there were no changes in VLF, LF, or HF power in SBP variability during each type of hypoxia in intact rats. In atropine-treated rats, LF power increased during Iso and Hyper and HF power increased during each type of hypoxia. There was no difference in respiratory frequency among the three kinds of hypoxia in both intact and atropine-treated rats. The results suggest that arterial PCO₂ level rather than respiration frequency produces changes in powers of RRI variability through changes in parasympathetic nerve activity and that with regard to SBP variability, parasympathetic nerve activity masks changes in LF power that reflect an increase in RSNA and those in HF power that reflect a mechanical consequence of respiration.

maximum-entropy method; parasympathetic nerve; sympathetic nerve; peripheral chemoreceptor; carbon dioxide

Because cardiovascular function is regulated by the autonomic nervous system to produce periodic oscillations in heart rate (HR or R-R interval (RRI)) and blood pressure (BP), the frequency components of these oscillations have been investigated by various methods to correlate their power(s) with respective autonomic nervous activities. Variabilities in HR and BP consist of high-frequency (HF), low-frequency (LF), and very low-frequency (VLF) components. It is also generally accepted that the HF component of HR variability is mediated by cardiac parasympathetic tone, which depends on respiration, whereas the LF component is mediated by both cardiac sympathetic and parasympathetic tone. Hence, the ratio of LF power to HF power (LF/HF) is an index of cardiac sympathetic tone. On the other hand, the HF component of BP variability has been regarded as a mechanical consequence of respiration and the LF component has been reported to parallel sympathetic nerve activity. The VLF component of both HR and BP variabilities has been suggested to be related to impairment of the baroreflex.

It is an important issue whether each component power responds to the changes in respective autonomic nervous activities during various stresses. There are many reports on the cardiovascular responses to systemic hypoxia with different levels of PaCO₂ in conscious rats using the maximum-entropy method with high resolution. Renal sympathetic nerve activity (RSNA) was monitored in each experiment to investigate the correlation of each component change with actual sympathetic nerve activity. In our previous study, we showed that there are differences in the cardiovascular responses to systemic hypoxia that depend on different activation of the autonomic nervous system in response to arterial Pco₂ (PaCO₂). In short, sympathetic nerve activity is excited via peripheral chemoreceptors and parasympathetic nerve activity is changed in parallel with PaCO₂.

Therefore, we analyzed RRI and systolic blood pressure (SBP) variabilities during systemic hypoxia with different levels of PaCO₂ in conscious rats using the maximum-entropy method with high resolution. Renal sympathetic nerve activity (RSNA) was monitored in each experiment to investigate the correlation of each component change with actual sympathetic nerve activity. The same analysis was performed in rats that were pretreated with muscarinic receptor blocker to clarify the contribution of parasympathetic nerve activity and also in sinoaortic denervation (SAD)-treated rats to clarify the contribution of chemo- and baroreflex.

METHODS

This study was performed in accordance with the “Guiding Principles for the Care and Use of Animals in the Fields of Physiological Sciences” published by the Physiological Society of Japan.
Animal Preparation

Thirty male Wistar rats, weighing 350–450 g, were divided into two groups: 18 intact and 12 SAD rats. The procedure has been described previously (13). Briefly, rats were anesthetized with pentobarbital sodium (50 mg/kg ip). Stainless steel hooked bipolar electrodes were placed around a left renal sympathetic nerve under a dissecting microscope. A pair of stainless steel wires were implanted at midchest to record the electrocardiogram (ECG). Polyethylene catheters were inserted via the femoral artery into the inferior vena cava for drug injection. After the operation, the rat was kept in a home cage at room temperature (25°C) and received Ringer solution and antibiotics.

Sinoaortic Denervation

The afferent pathways of peripheral chemo- and baroreceptors were denervated according to the procedure described by Krieger (18) >2 wk before implantation of the electrodes and catheters. To evaluate the completeness of SAD, phenylephrine (2 µg/kg) was injected intravenously. Mean BP increased by 56 ± 4 mmHg in response to phenylephrine, whereas the decrease in HR was small (13 ± 3 beats/min) compared with that previously reported in intact rats (64 ± 5 beats/min) (30).

Experimental Protocol

The details have been described previously (13). At least 48 h after implantation of the electrodes and catheters, an unrestrained rat in its home cage was placed inside an air-tight acrylic chamber (40 liters). Air, N₂, and CO₂ were mixed in a multiflowmeter, and the gas mixture flowed into the chamber at 20 l/min. After a control period of 30 min, air was replaced by one of the following hypoxic gas mixtures for 30 min: 1) hypoxic hypoxia (Hypo, 10% O₂ in N₂), 2) isocapnic hypoxia (Iso, 10% O₂ and 3.0–4.0% CO₂ in N₂), and 3) hypercapnic hypoxia (Hyper, 10% O₂ and 6.5–7.0% CO₂ in N₂). Changes in blood pH, arterial Po₂, and Paco₂ produced by this system were confirmed in a previous report (13). The 18 intact rats were divided equally between Hypo, Iso, and Hyper (6 rats per type of hypoxia). The 12 SAD rats were divided equally between Iso and Hyper because of the absence of a peripheral chemoreceptor drive to expel CO₂.

Administration of atropine sulfate. The identical hypoxic exposure was carried out on the rats that had served as intact animals after atropine treatment on the next day. Atropine sulfate was administered before hypoxia in a primary intravenous dose of 5 mg/kg, and this was followed by intravenous doses of 0.2 mg every 10 min. Phenylephrine (4 µg/kg iv) was injected after the experiment to confirm blockade of the parasympathetic effect. Mean BP increased by 46 ± 3 mmHg, whereas the rapid decrease in HR was abolished.

Data recording. The original RSNA was amplified and filtered (50–1,000 Hz) by a low-noise amplifier (AVB11, Nihon Kohden), and the rectified signal with a time constant of 0.1 s was then integrated (E1601G, Nihon Kohden). To quantify the RSNA response, percent changes in the integrated RSNA during hypoxia were calculated, taking the mean value before hypoxia as 100%. The fast peaks of R waves on the ECG were detected, and RRI was measured (AT601G, Nihon Kohden). BP and the ECG were also amplified and filtered (AP621G and AB621G, Nihon Kohden), and analog outputs of BP, RRI, ECG, and RSNA were recorded on a digital tape recorder (PC116, Sony).

Statistical Analysis

All values are given as means ± SE. Comparisons between groups and within groups were analyzed using a two-way ANOVA. Individual comparisons were performed using Fisher’s protected least significant difference when the F value was significant (P < 0.05). Significance was established at P < 0.05.

RESULTS

Effect of Hypoxia on RRI, SBP, RSNA, and Respiratory Frequency

Means and SE of RRI, SBP, RSNA, and respiratory frequency of 280-s sections selected before and during hypoxia are shown in Table 1. In intact rats, RRI and SBP both decreased during Hypo. Neither RRI nor SBP changed significantly during Iso. RRI and SBP both increased during Hyper. RSNA increased during Iso and Hyper and tended to increase during Hypo (P = 0.065). The increase was in proportion to the increase in Paco₂. The respiratory frequency increased during each type of hypoxia, and there was no significant difference in the level of the increase.

In atropine-treated rats, RRI decreased and there was no significant change in SBP during Hypo. There was no significant change in RRI during Iso or Hyper. RSNA was increased as in intact rats in proportion to the increase in
PaCO₂. The respiratory frequency increased during each type of hypoxia as in intact rats.

In SAD rats, there were no significant changes in these parameters or respiratory frequency during I0. RRI decreased and RSNA increased during Hyper, but there was no significant change in SBP. The respiratory frequency increased, but the level of this increase was less than that in intact rats.

Effect of Atropine and SAD on Spectra of RRI and SBP Variabilities

Figure 1 shows means and SE of each power in RRI variability before hypoxia in intact, atropine-treated, and SAD rats. In atropine-treated rats all of the powers were decreased compared with those in intact rats. In SAD rats, VLF and LF powers were decreased compared with intact rats, but there was no significant difference in HF power. In atropine-treated and SAD rats LF/HF was decreased compared with that in intact rats (see Fig. 5A).

Figure 2 shows means and SE of each power in SBP variability before hypoxia. In atropine-treated rats LF power was decreased, whereas there were no significant differences in VLF and HF powers. In SAD rats VLF power was increased, whereas LF power was decreased. There was no significant difference in HF power.

Change in RRI Variability During Hypoxia

Typical changes in the spectra of RRI during the three different types of hypoxia in intact rats are shown in Fig. 3A. Each graph consists of 12 consecutive spectra that show the relations between frequencies ranging from 0.25 to 3.3 Hz and power spectral densities calculated from 60-s data sets. There are discrete LF and HF components, and each component was

Table 1. RRI, SBP, RSNA, and respiratory frequency before and during hypoxia

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>RRI, ms</th>
<th>SBP, mmHg</th>
<th>RSNA, %</th>
<th>f, breaths/min</th>
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<tr>
<td><strong>Intact</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Before hypoxia</td>
<td>6</td>
<td>160.6 ± 5.8</td>
<td>140.0 ± 5.5</td>
<td>100</td>
<td>87.0 ± 5.5</td>
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<tr>
<td>Hypocapnic hypoxia</td>
<td>6</td>
<td>127.7 ± 3.0*</td>
<td>120.1 ± 5.4*</td>
<td>125.4 ± 10.8</td>
<td>130.0 ± 3.5*</td>
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<tr>
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<td>6</td>
<td>166.9 ± 2.8</td>
<td>127.0 ± 6.3</td>
<td>100</td>
<td>80.0 ± 4.6</td>
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<td>6</td>
<td>166.6 ± 12.0</td>
<td>124.6 ± 4.7</td>
<td>161.5 ± 16.3*</td>
<td>139.0 ± 3.2*</td>
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<tr>
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<td>6</td>
<td>172.7 ± 4.1</td>
<td>127.3 ± 5.7</td>
<td>100</td>
<td>87.5 ± 7.2</td>
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<td>213.8 ± 5.1*</td>
<td>132.8 ± 5.2*</td>
<td>242.4 ± 20.7*</td>
<td>142.5 ± 4.4*</td>
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<td>Before hypoxia</td>
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<td>148.1 ± 6.2</td>
<td>122.5 ± 5.3</td>
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<td>89.0 ± 6.1</td>
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<td>118.0 ± 5.7</td>
<td>169.1 ± 9.8*</td>
<td>141.5 ± 3.5*</td>
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<tr>
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<td>6</td>
<td>148.4 ± 3.5</td>
<td>118.1 ± 5.7</td>
<td>100</td>
<td>82.5 ± 3.8</td>
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<td>148.4 ± 3.4</td>
<td>131.0 ± 4.7*</td>
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<td>132.0 ± 5.3*</td>
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<tr>
<td>Before hypoxia</td>
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<td>163.4 ± 8.3</td>
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Values are means ± SE of R-R interval (RRI), systolic blood pressure (SBP), renal sympathetic nerve activity (RSNA), and respiratory frequency (f) of selected sections before and during hypoxia in intact, atropine-treated, and sinoaortic denervation (SAD)-treated rats; n, no. of rats. *P < 0.05, significant change compared with each value before hypoxia.

![Fig. 1. Means and SE of each power in R-R interval (RRI) variability before hypoxia in intact (n = 18), atropine-treated (n = 18), and sinoaortic denervation (SAD)-treated (n = 12) rats. VLF, very low frequency; LF, low frequency; HF, high frequency. Each power decreased in atropine-treated rats. VLF and LF powers decreased in SAD rats. Significantly different from values in intact rats: *P < 0.05, **P < 0.001.](image1)

![Fig. 2. Means and SE of each power in systolic blood pressure (SBP) variability before hypoxia in intact (n = 18), atropine-treated (n = 18), and SAD (n = 12) rats. VLF power increased in SAD rats. LF power decreased in atropine-treated and SAD rats. Significantly different from values in intact rats: *P < 0.05, **P < 0.001.](image2)
uniform with regard to central frequency and the magnitude of the power spectral density among the 12 consecutive spectra before hypoxia. The central frequency and power spectral density of each component changed with the application of hypoxia but remained constant during hypoxia. This characteristic was also observed before and during hypoxia in atropine-treated and SAD rats.

Figure 4 shows the percent changes in each power during hypoxia, which were calculated by taking the mean value before each hypoxia as 100%, in intact, atropine-treated, and SAD rats. In intact rats (Fig. 4A), each power was decreased during Hypo compared with that before hypoxia. There was no significant change in each power during Iso, whereas each power was markedly increased during Hyper. In atropine-treated rats (Fig. 4B) VLF and LF powers decreased, whereas there was no change in HF power, during Hypo. There were no significant changes in each power during Iso or Hyper. In SAD rats (Fig. 4C) VLF power decreased,
whereas LF and HF powers did not change during Iso. There was no significant change in any power during Hyper.

LF/HF before and during hypoxia are shown in Fig. 5, A and B. In intact rats LF/HF was decreased during each type of hypoxia compared with that before hypoxia. In atropine-treated rats LF/HF also tended to be decreased during each type of hypoxia. In SAD rats there was no significant change in LF/HF.

Change in SBP Variability During Hypoxia

Typical changes in the spectra of SBP variability during each type of hypoxia in intact rats are shown in Fig. 3 B. Each spectrum had discrete LF and HF components throughout 12 consecutive spectra before and during hypoxia. There are also discrete components in atropine-treated and SAD rats.

Figure 6 shows the percent changes in each power during hypoxia, which are expressed as in Fig. 4, in intact, atropine-treated, and SAD rats. In intact rats (Fig. 6A) there was no significant change in each power during each type of hypoxia. In atropine-treated rats (Fig. 6B) HF power increased, whereas VLF and LF powers did not change, during Hypo. In SAD rats (Fig. 6C) LF and HF powers did not change, whereas LF and HF powers did not change, during Iso. There was no significant change in each power during Hyper.

DISCUSSION

RRI Variability

Before hypoxia. Effect of atropine. The present results show that peripheral muscarinic receptor blockade decreased each power in RRI variability (Fig. 1). This result is consistent with other reports in conscious rats (15), dogs (28), and humans (2, 24). The effect on the central nervous system might also contribute to the decrease in VLF and LF powers, because it has been reported that atropine sulfate elicited an additional increase in HR during cold blockade of the vagi (27).

Effect of SAD. The present results (Fig. 1) suggest that the baroreflex mainly influenced VLF and LF powers. Our results are consistent with previous reports on VLF and LF powers in SAD dogs (28) and SAD cats (9) and on HF power in SAD dogs (28). Cerruti et al. (5) reported that the coherence between BP and HR variabilities was decreased in the VLF and LF bands but not in the HF band in SAD rats.

Response to hypoxia. The effect of the increased respiratory frequency during hypoxia should be taken into consideration in the present study. It has been reported that LF and HF powers decreased as respiratory frequency increased without a change in mean RRI in humans (4, 12). There are also reports that expansion of the lungs inhibits cardiac vagal motoneurons during hypoxia (7) and that parasympathetic nerve activity has an effect on each power (2, 24). Thus it is reasonable to assume that a decrease in power during hypoxia is caused by changes in the mode of respiration.

Response to Hypo. Hypo decreased all of the powers in intact rats (Fig. 4A) and decreased VLF and LF powers in atropine-treated rats (Fig. 4B). This decrease in HF power was abolished in atropine-treated rats (Fig. 4B), suggesting that parasympathetic nerve activity is inhibited because of changes in mode of respiration during Hypo in intact rats. Although the inhibition of parasympathetic nerve activity might influence VLF and LF powers to some extent in intact rats, it is also likely that the baroreflex involves these powers, because its sensitivity was reported to be decreased during Hypo (13).

Response to Hyper. Hyper increased all of the powers, and the level of the increase in HF power was greater than those in other powers in intact rats (Fig. 4A). These increases in all of the powers were abolished in atropine-treated rats (Fig. 4B). This suggests that all of the powers are increased because of the excitation of parasympathetic nerve and that HF power is most
sensitive to parasympathetic nerve activity during Hyper. In the present study, the increases in all of the powers were also abolished in SAD rats (Fig. 4C). Therefore, afferent impulses from peripheral chemoreceptors are needed to produce the oscillation of efferent impulses to the heart, which are mainly via parasympathetic nerve during Hyper. Fukuda et al. (10) concluded that hypercapnia produced bradycardic or no change in HR because of the local inhibitory effect of CO2 on the heart, despite cardiac sympathetic excitation and vagal inhibition. However, this is not likely in the present study, because intact rats showed increases in all of the powers and these increases were abolished in atropine-treated rats.

**Response to Iso.** Iso did not change any of the powers in intact and atropine-treated rats or either LF or HF power in SAD rats (Fig. 4). These results suggest that sympathetic nerve activity increased via peripheral chemoreceptors has little effect on RRI variability and also that parasympathetic nerve activity does not change significantly during Iso.

**Effect of \( P_{\text{ACO}_2} \).** It has been reported that controlled respiration (22), respiratory frequency (4, 12), and tidal volume (4) have important effects on RRI variability. However, the effect of hypoxia per se, which changed respiratory mode, on RRI variability was small, and those of Hypo and Hyper depended on the changes in the level of \( P_{\text{ACO}_2} \). In this study, i.e., a decrease during Hypo, no change during Iso, and an increase during Hyper in intact rats. Our results suggest that \( P_{\text{ACO}_2} \) rather than respiratory mode is an important factor for the regulation of RRI variability.

**Does LF/HF reflect sympathetic nerve activity?** RSNA increased in proportion to the increase in \( P_{\text{ACO}_2} \) (Table 1). It is generally accepted that LF power and LF/HF increase during sympathoexcitation (22, 23). However, LF power increased only during Hyper (Fig. 4A). Because the increase in LF power was abolished in atropine-treated rats (Fig. 4B), this increase was thought to be caused by the excitation of parasympathetic nerve. Our results are consistent with a previous report that LF power in HR variability was not correlated with muscle sympathetic nerve activity in humans, even when sympathoexcitation and vagal inhibition were induced by the injection of nitroprusside (29). LF/HF did not reflect the increase in RSNA in intact rats, either (Fig. 5B), because the change in LF power depended on parasympathetic nerve activity to a greater extent during each type of hypoxia. Furthermore, it has been reported that both sympathetic and parasympathetic nerves were coactivated during the stimulation of chemoreceptors (13, 17).

**SBP Variability**

Before hypoxia, \( E \)fect of atropine. The present results (Fig. 2) are not completely consistent with those of other authors; decreases in both LF and HF powers in humans (2, 24), no change in LF power and an increase in HF power in conscious dogs (28), no change in each power in conscious rats (15), and increase in both LF and HF powers in conscious rats (6) have been reported. In this study, the parasympathetic efferent loop of the baroreflex had been completely inhibited, because abolition of the decrease in HR in response to the increase in BP by phenylephrine was confirmed in each experiment. There are many reports that LF power is influenced by baroreflex control (5, 9, 14, 28).

Atropine had no effect on respiratory frequency (Table 1) or HF power (Fig. 2), regardless of the decrease in RRI oscillation. HF power in SBP variability has been reported to be a mechanical consequence of respiration (22). In another report, HF power was interpreted as a direct effect of centrally mediated HR oscillation (2). However, a change in relation to the decrease in RRI oscillation was not observed in this study.

**Effect of SAD.** The present result (Fig. 2) is consistent with other reports on VLF and LF powers (5, 9, 14, 28) and HF power (5, 9, 28). It suggests that baroreflex control involves VLF and LF powers but not HF power. In the absence of baroreflex control, a nonneural mechanism, for example, hormonal regulation (e.g., renin-angiotensin, kallikrein-kinin; Refs. 11, 25) and independent local regulation of regional hemodynamics (e.g., nitric oxide), may produce VLF power (16).

Response to hypoxia. Does LF power reflect sympathetic nerve activity? LF power did not change and did not reflect the excitation of sympathetic nerve during each type of hypoxia in intact rats (Fig. 6A). This result...
coincides with reports that rats with high sympathetic nerve activity did not always have high LF power (1, 31). However, it is generally accepted that LF power corresponds to the Meyer wave (28). LF power has been proposed as an indicator of vasosympathetic tone because of its close relation to RSNA spectra (3) and because it increases in response to vasodilatation (15, 22) or mental stress (23). In the present study LF power increased and reflected the increase in RSNA during Hyper, and this tendency was observed during Iso in atropine-treated rats (Fig. 6B). This suggests that the excitation of parasympathetic nerve might mask the increase in LF power produced by excitation of sympathetic nerve and might explain the differences between our results and those of other researchers (15, 22, 23).

There was no change in LF power during each type of hypoxia in SAD rats (Fig. 6C), because of the absence of afferent impulses from peripheral chemo- and baroreceptors.

RESPONSE OF VLF POWER. Peripheral blood vessels are reportedly dilated by a direct effect of hypoxia (10, 19) or hypercapnia (10) in anesthetized rats. The decrease in VLF power during Iso in SAD rats (Fig. 6C) may be caused by vasodilatation induced by hypoxia, which is compensated for by peripheral chemo- and baroreceptors in conscious intact rats. VLF power recovered during Hyper in SAD rats (Fig. 6C), which might produce vasoconstriction mediated via central chemostimulation.

RESPONSE OF HF POWER. In atropine-treated rats, HF power increased to 160–200% of control during each type of hypoxia, which paralleled the change in respiratory frequency and there was no difference in the level of increase (Fig. 6B). In intact rats, there was no change in HF power during each type of hypoxia (Fig. 6A) regardless of the increase in respiratory frequency. HF power was considered a mechanical consequence of respiration, which could act directly on intrathoracic vessels or indirectly through changes in stroke volume and HR (2, 22). Our results suggest that parasympathetic nerve activity may control the respiration-related changes in stroke volume and HR and regulate HF power secondarily, so that HF power might not increase in intact rats.

In summary, frequency components of RRI variability depended on the level of P_{ACO_2}, which changes efferent impulses of parasympathetic nerves via peripheral chemoreceptors, but were not affected by systemic hypoxia per se. Frequency components of SBP variability were not affected by either systemic hypoxia or P_{ACO_2} level, because of the difference in sensitivity to the change in parasympathetic nerve activity between the heart and blood vessels. No spectral component reflected sympathoexcitation caused by hypoxia and/or hypercapnia, but LF power in SBP variability did reflect sympathoexcitation after atropinization.

APPENDIX

In the program of MemCalc, a time series is assumed to be composed of underlying variation and fluctuating parts; the underlying variation is expressed as the function x_m(t), which can be given by a linear combination of sine and cosine functions

\[ x_m(t) = a_0 + \sum_{n=1}^{N_p} \left[ a_n \sin(2\pi f_n t) + b_n \cos(2\pi f_n t) \right] \]  \hspace{1cm} (A1)

where \( f_n \) is the frequency of the nth component, \( a_n \) and \( b_n \) are the amplitudes of the nth periodic component, \( N_p \) is the total number of components, and \( a_0 \) is a constant that indicates the mean value of the time series. The value of \( f_n \) is determined by the peaks in the power spectral density. Its estimate \( P(f) \) can be expressed as

\[ P(f) = \frac{\Delta P_m}{\sum_{k=-m}^{m} \gamma_{mk} \exp(-12\pi f k \Delta t)^2} \]  \hspace{1cm} (A2)

where \( P_m \) is the output power of the prediction error filter of the order \( m \), and \( \gamma_{mk} \) is the corresponding filter coefficient, \( m = 0, 1, 2, ..., M \) (\( M \) = optimum filter order). \( P_m \) and \( \gamma_{mk} \) are determined by Yule-Walker equations using Burg’s algorithm. Ohtomo and Tanaka (20) demonstrated that Eq. A1 gives a basis for determining the filter order, and the optimum order should be determined by the condition; the filter order is \( >1/f_{\min} \), where \( f_{\min} \) is the minimum among the central frequencies of the components.

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