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Power Spectral Analysis of Short-Term RR Interval and Arterial Blood Pressure Oscillations in the Lizard, *Gallotia galloti*: Effects of Sympathetic Blockade

LUIS DE VERA* AND JULIÁN GONZÁLEZ
Department of Physiology, Faculty of Medicine, University of La Laguna, 38320 Tenerife, Canary Islands, Spain

ABSTRACT The role of the sympathetic limb of the autonomic nervous system (ANS) in the mediation of oscillations in consecutive beat-to-beat RR interval (RRI) and systolic blood pressure (SBP) values of lizards, *Gallotia galloti*, was investigated using spectral analysis and measuring effects of autonomic blockers. α-Adrenergic blockade decreased the power spectral density (PSD) of both RRI and SBP very low frequency (VLF: 0.007–0.055 Hz) and low frequency (LF: 0.055–0.150 Hz) bands, whereas β-adrenergic blockade increased the PSD of both RRI- and SBP-VLF and RRI- and SBP-LF bands. These findings suggest that in lizards 1) the VLF and LF peaks of RRI and SBP power spectra are α-adrenergic mediated, and that 2) the β-adrenergic activity of the sympathetic system may act buffering all RRI and SBP oscillations below 0.150 Hz. These results, when analyzed jointly with the ones obtained from a previous study (De Vera and González. 1997. Comp Biochem Physiol 85A:389–394) on the effects of parasympathetic blockade on lizards' RRI and SBP oscillations, demonstrate that these reptiles, like mammals, exhibit spontaneous short-term oscillations in their HR and SBP which are mediated by the ANS. However, unlike mammals, the RRI and ABP low-frequency oscillations in *Gallotia* seem to be similarly affected by the ANS and appear to be powered by α-adrenergic and parasympathetic activities and buffered by β-adrenergic activity. *J. Exp. Zool. 283:113–120, 1999.*

Heart rate (HR) and arterial blood pressure (ABP) in vertebrates do not remain constant but fluctuate about a mean value in consequence of the action and interaction of complex mechanisms which act through neural, mechanical, vascular, and humoral factors as well as others (Cerutti et al., '93). Cardiovascular neural regulation has been described as the integrated response to a continuous interaction of inhibitory and excitatory reflexes, one of its special features being that there is a dynamic closed-loop interaction of these reflexes with rhythmic hemodynamic oscillations (Malliani et al., '86; Pagani et al., '97).

Frequency domain analysis—by FFT or by autoregressive modeling—of beat-to-beat HR and ABP short-term oscillations (in the seconds-to-minutes range) is increasingly used to obtain information on the mechanisms responsible for cardiovascular regulation. In mammals, a number of animal (Akselrod et al., '81, '85, '87; Japundzic et al., '90; Cerutti et al., '91; Murphy et al., '91; González et al., '95) and human (Pomeranz et al., '85; Pagani et al., '86; Saul et al., '90; Malliani et al., '91; Jokkel et al., '95; Parati et al., '95) studies have shown vagal activity to include mainly high-frequency (synchronous with respiration) oscillations and both sympathetic and vagal activity to include mainly low-frequency (0.02–0.6 Hz, most animal studies; 0.01–0.15 Hz, most human studies) fluctuations.

As occurs in mammals, central control neural mechanisms affecting the cardiovascular functions are also present in reptiles (White, '76). In this context, it has been demonstrated that the reptilian heart is innervated by vagal parasympathetic cholinergic inhibitory nerves that facilitate negative inotropic and chronotropic responses on electrical nerve stimulation, as well as by sympathetic adrenergic excitatory nerves that facilitate, when electrically stimulated, positive inotropic and chronotropic responses via β-adrenoceptors present in the pacemaker region, atria and ventricle (Berger,

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*Correspondence to: Dr. Luis De Vera, B.Sc. Ph.D., Department of Physiology, Faculty of Medicine, University of La Laguna, 38320 Tenerife, Canary Islands, Spain. E-mail: lvera@ull.es
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'71; Lillywhite and Seymour, '78; Nilsson, '83; Donald et al., '90; Lillywhite and Donald, '94). Thus it could be that in reptiles, as in mammals, the HR and ABP are affected by the activity of physiological control systems which regulate circulation through the ANS. In fact, a previous work from our laboratory (González and De Vera, '88) showed that the Fourier spectra of the lizard HR variability signal have two main peaks in the 0.008–0.150 Hz range, which are temperature dependent and possibly associated, as in mammals, with cutaneous vasomotor thermoregulatory and endogenous pressure vasomotor activities. More recently, we have described (De Vera and González, '97) short-term oscillations in the RRI and SBP values of lizards associated with the activity of the parasympathetic limb of ANS. This study indicated that vagal blockade with atropine decreased all RRI oscillations and low frequency (LF: 0.03–0.100 Hz) and high frequency (HF, synchronous with respiration) oscillations of SBP. As very low frequency (VLF: 0.008–0.030 Hz) and LF oscillations of RRI and SBP were still present after vagal blockade, it was suggested that other neural or humoral systems different from parasympathetic could also mediate low frequency RRI and SBP variabilities. Therefore, as we had not yet studied the possible sympathetic mediation of these fluctuations, we undertook the present study with the aim of investigating the role of the sympathetic limb of the ANS in the mediation of low frequency RRI and SBP variabilities in order to have a more complete view of the activity of physiological control systems that regulate circulation in reptiles through the ANS, as well as to demonstrate that the dynamic of cardiovascular control in reptiles produces, as in mammals, oscillations in the HR and ABP mediated by the ANS.

MATERIALS AND METHODS

Animals

Thirteen lizards of the species *Gallotia galloti*, 11.3–13.8 cm in length (snout-vent), and 66.7–79.2 g (mean 71.7 ± 4.0) body mass from the island of Tenerife (Canary Islands, Spain) were used. The animals were captured in their natural habitat and kept in terraria under a 12:12 hr light-dark cycle, for at least 1 week before experiments. A temperature of 23 ± 1°C was maintained during the light phase, and 20 ± 1°C during the dark. The relative humidity ranged from 56–68%. Food and water was available ad libitum. All lizards used in this study were deprived of food for at least 24 hr before experiments. The study protocol was approved by the Ethical Committee of the Faculty of Medicine of the University of La Laguna.

Surgery

Surgical procedures, under ether anesthesia, consisted of non-occluding cannulation of the dorsal aorta for continuous ABP recording and implantation of electrodes to record continuous electrocardiogram (ECG). The arterial catheter (ID 0.28 mm, OD 0.61 mm polyethylene tubing, Portex), filled with sodium heparin (100 IU/ml in 0.9% NaCl) was inserted into the dorsal aorta at the level of the pre-lumbar region and secured with a suture. It was flushed twice daily with heparinised saline to prevent clot formation. The bipolar ECG was recorded by two 6-mm length stainless steel wire electrodes implanted subcutaneously, one near the nuchal region and the other near the sacral region. After surgery, each lizard was housed individually in a temperature controlled terrarium (22 ± 1°C) and allowed to recover for 1 day before recording began. No postoperative drugs were used.

Experimental protocol

ABP and ECG recordings were carried out in the morning, in a room far from any visual and acoustic disturbance, keeping the lizard in a thermostatically-controlled (23 ± 0.5°C) chamber (45 × 30 × 25 cm). The animal was kept within the chamber for a 3-hr habituation period, after which measurements were made when it was at rest. Core body temperatures were monitored by an electric thermometer (Nihon-Kohden MGA III-219) provided with a thermistor probe that was inserted into the lizard's cloaca ~2 cm deep. The experiment consisted of a 50- to 70-min baseline recording session of ABP and ECG of the lizard at rest and a 40- to 100-min recording session after sympathetic nervous system blockade (SSB). The autonomic nervous system blocking agents used were prazosin (α-adrenergic blocker; 3 mg/kg) and propranolol (β-adrenergic blocker; 4 mg/kg). All drugs (purchased from Sigma Chemical Co., St. Louis, MO) were dissolved in saline and administrated by means of a 0.08-ml bolus injected via the dorsal aortic catheter. The blocker doses used in this study were empirically determined to be twice the minimum doses necessary to remove sympathetic effects from the resting HR (approximately 30 min after the bolus injection). Once the acute effects of these sole doses of blockers were

achieved, they always lasted the 140 min duration, if not more, of a typical recording session.

Measurements

ABP and ECG were continuously and simultaneously measured by a recording system Nihon Kohden RM-85. ECG electrodes were connected to the biopotential amplifier channels of the recording system and ABP was measured from the arterial catheter by means of a blood pressure transducer (4-327-I Physiological Pressure Transducer, Telos Medical, Upland, CA) connected to a transducer amplifier channel of the recording system. The analogue signals from the recording system were led to a 14-bit A/D converter card controlled by a 486-DX2 PC for on-line processing by means of a computer assembler program, developed in our laboratory, which sampled ECG and ABP signals simultaneously at a frequency of 1 kHz and calculated the series of RR consecutive intervals and SBP values within each RRI.

Spectral analysis

Spectral analysis of RRI and SBP values was performed from the time series by linearly interpolating (Rompelman, '80) the consecutive values of each variable every 500 ms. Several 256-sec duration (sets of 512 data points sampled at 2 Hz) RRI and SBP variability signal segments were spectrally analyzed using a fast Fourier transform (FFT) algorithm to obtain their power spectral density function (PSD). In order to perform this analysis according to time-series analysis standards, all signal segments were first linear-trend removed by means of the least-squares method and cosine tapered over the first and last 10% of the samples, to reduce leakage in the spectrum (Bendat and Piersol, '71). A total of 255 spectral coefficients with a frequency resolution of 0.0039 Hz were obtained from each spectrum.

After processing all signal segments of a recording session from a particular lizard, an average power spectrum of each of the two signals (RRI variability signal and SBP variability signal) was obtained; in this way, four average spectra (two spectra from each signal, one in control conditions and the other under SSB) were obtained for each experimental animal. The average spectra from the RRI and SBP variability signals were analyzed calculating their total PSD and the cumulative PSD in two different spectral bands: a very low frequency (VLF) band from 0.007 to 0.055 Hz and a low frequency (LF) band from 0.055 to 0.150 Hz.

Statistical analysis

Data are expressed as mean ± SE. Statistical analyses of differences between control condition and after SSB were performed using a nonparametric Wilcoxon's signed rank test for paired data. Comparisons between percentages of variation in total PSD or in cumulative PSD of the different spectral bands before and after SSB were carried out by means of a Kruskal-Wallis one-way nonparametric AOV test. A P value less than 0.05 was considered to be statistically significant.

RESULTS

An example of simultaneous ECG and ABP signals recorded in control conditions in a 73.2 g lizard is shown in Figure 1. The mean baseline RRI and SBP values obtained in resting conditions (n = 13) were 1363.2 ± 66.0 ms (HR: 46.1 ± 2.3 beats/min) and 31.1 ± 1.0 mmHg, respectively.

Mean RRI and SBP values in control conditions and after SSB are shown in Table 1. Prazosin decreased (–22.7 ± 4.7%) the RRI as well as the SBP (–44.6 ± 6.1%) whereas propranolol increased (50.7 ± 8.6%) the RRI and did not change the SBP.

Examples of 256-sec beat-to-beat RRI variability signals in control (top), after α-adrenoceptor blockade (middle) and after β-adrenergic blockade (bottom) conditions are shown in Figure 2 on the left. The corresponding PSD of these signals is shown in Figure 2 on the right. In control condi-

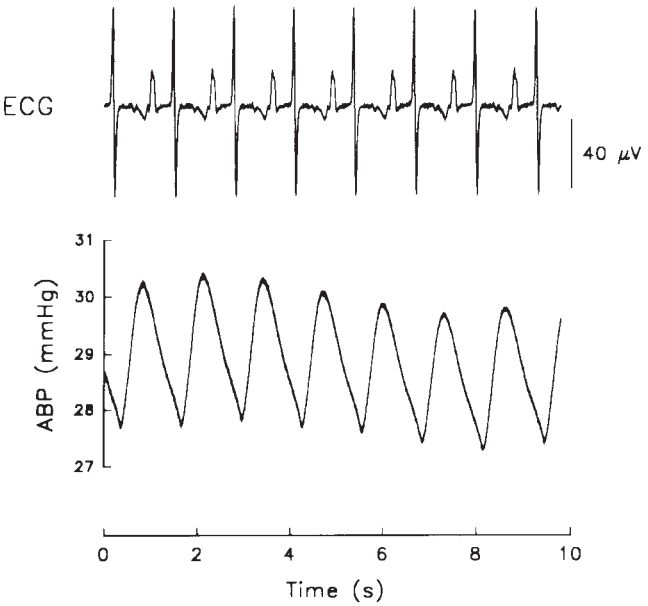

Fig. 1. An example of simultaneous digitized recordings of cardiovascular variables from a 73.2-g male lizard in control conditions at 23°C.

TABLE 1. RRI and SBP mean values calculated in control and SSB lizards[1]

	RRI[2]	SBP[2]
Control	1335.4 ± 104.9	30.1 ± 1.6
Prazosin	1028.8 ± 106.3**	20.4 ± 1.4**
N = 7		
Control	1454.4 ± 118.3	31.6 ± 1.4
Propranolol	2161.2 ± 135.4*	29.8 ± 1.2
N = 6		

[1]N, number of lizards.
[2]Values are mean ± SE. RRI in ms and SBP in mmHg.
*P = 0.0156 blockade vs. controls; **P = 0.0078 blockade vs. controls.

tions, the RRI power spectra exhibits two major spectral peaks: a very low-frequency peak corresponding to oscillations in the VLF band, and a low-frequency peak in the LF band. Prazosin decreased the power of the RRI-VLF and RRI-LF spectral bands (–59.6 ± 4.5%) as well as the total power of the spectra, whereas propranolol increased the power of the RRI-VLF (381.2 ± 117.0%) and RRI-LF (1485.8 ± 578.9%) bands as well as the total power of the spectra. Under propranolol, the increase of power in the RRI-LF band was greater (P = 0.0325) than the increase of

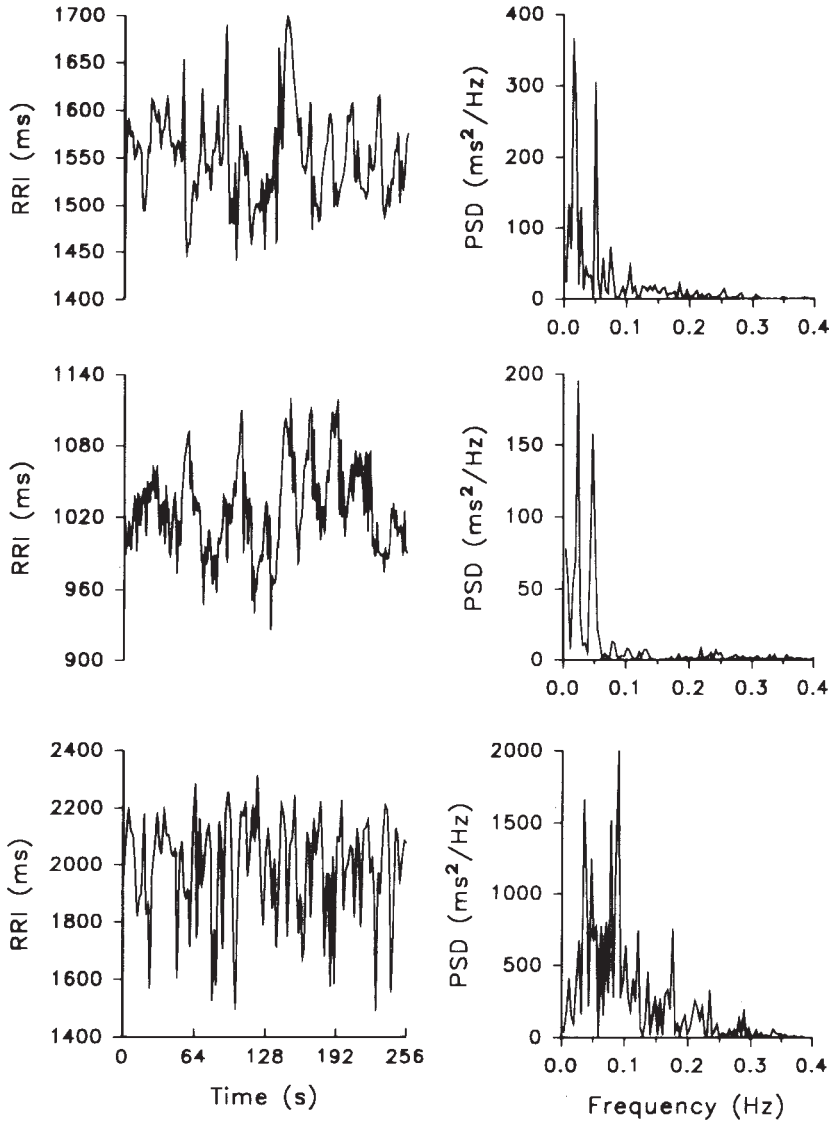

Fig. 2. An example of RRI 256-sec beat-to-beat time series (**left**) and their corresponding power spectra (**right**) obtained from a 68.3-g male lizard in control conditions (**top**), after α-adrenergic blockade with prazosin (**middle**) and after β-adrenergic blockade with propranolol (**bottom**). Time series are linear-trend removed. In order to improve visual resolution, power spectra have been cut down to the 0.4-Hz level where most of the power is concentrated.

power in the RRI-VLF band. These results are summarized in Table 2.

The left side of Figure 3 depicts examples of 256-sec beat-to-beat SBP variability signals in control (top), under α-adrenergic blockade (middle) and under β-adrenergic blockade (bottom) conditions. The corresponding PSD of these signals is shown in Figure 3 on the right. The PSD of SBP variability signal also showed, in control conditions, two major spectral peaks falling in the same frequency ranges as that of the RRI-VLF and RRI-LF spectral bands. Prazosin decreased the power of the SBP-VLF and SBP-LF spectral bands (–80.6 ± 0.9%) as well as the total power of the spectra, whereas propranolol increased the power of the SBP-VLF and SBP-LF bands (894.1 ± 149.0%) as well as the total power of the spectra. These results are also summarized in Table 2.

DISCUSSION

The control resting RRI value reported for *Gallotia galloti* in the present investigation follows the trend of the values obtained by the authors in previous studies (De Vera and González, '86, '97; González and De Vera, '88). The SBP value reported for our lizards in control conditions falls within the range (15–90 mmHg) previously reported for different reptilian species (Lillywhite and Seymour, '78; Stinner and Ely, '93) and it is also in accordance with the value reported by the authors in a previous paper (De Vera and González, '97).

TABLE 2. PSD of RRI and SBP variability signals calculated in VLF and LF bands in control and SSB lizards[1]

	VLF[2] (0.007–0.055 Hz)	LF[2] (0.055–0.150 Hz)
RRI (ms^2)		
Control	1739.7 ± 239.0	883.3 ± 171.0
Prazosin N = 7	688.9 ± 179.9**	296.1 ± 120.1**
Control	1950.8 ± 311.0	961.9 ± 186.9
Propranolol N = 6	8303.4 ± 1422.3*	11880.1 ± 3179.4*
SBP ($mmHg^2 \times 10^{-2}$)		
Control	2.84 ± 1.18	0.37 ± 0.10
Prazosin N = 7	0.43 ± 0.17**	0.05 ± 0.02**
Control	0.97 ± 0.48	0.24 ± 0.16
Propranolol N = 6	11.08 ± 6.82*	1.73 ± 1.01*

[1]N, number of lizards.
[2]Values are means ± SE.
*P = 0.0156 blockade vs. controls; **P = 0.0078 blockade vs. controls.

α-Adrenergic blockade with prazosin decreased SBP and increased HR. The decreasing of ABP after prazosin has been explained in mammals as the result of a fall in peripheral vascular resistance and in venous return as a consequence of the blockade of the α-adrenoceptors of arterioles and veins (Hoffman and Lefkowitz, '91). The parallel tachycardia after prazosin found in *Gallotia galloti* has also been described in snakes after α-adrenoceptor blockade with phentolamine (Lillywhite and Seymour, '78) and in mammals after α-adrenergic blockade with prazosin (Japundzic et al., '90; González et al., '95), and it has been explained as a 'circulatory effort' to compensate for the lowered central pressure incurred by vasodilation of the resistance vessels (Lillywhite and Seymour, '78). Propranolol is a competitive β-receptor antagonist that blocks adrenergic cardiac stimulation (Lillywhite and Seymour, '78) therefore, the bradycardic response, together with the lack of change in SBP after β-adrenergic blockade with propranolol obtained in the present study, constitute findings that are in accordance with previous observations in different groups of reptiles (Hohnke, '75; Lillywhite and Seymour, '78; Cipolle et al., '86; Singh et al., '91) and mammals (Akselrod et al., '85; González et al., '95).

As was shown in our previous studies (González and De Vera, '88; De Vera and González, '97), and as was also the case in the present investigation, both the beat-to-beat RRI and SBP spectra of *Gallotia galloti* in control conditions at 23°C exhibited two main spectral peaks below 0.150 Hz. A high frequency spectral peak in the same frequency band as that of the respiration was also observed in some of the RRI and/or SBP spectra when the lizards exhibited a pronounced regular respiratory pattern. The behavior of this high frequency spectral peak has been discussed in a previous report (De Vera and González, '97) and due to precisely the sporadic character of this peak, its study was not included in the present work. Two peaks below the respiratory frequency in the HR and ABP spectra have also been described in rats and humans (Pagani et al., '86; Japundzic et al., '90; Cerutti et al., '91; González et al., '95): a very low-frequency (VLF: 0.02–0.2 Hz in rats; 0.01–0.06 in humans) peak and a low frequency (LF: 0.2–0.6 Hz in rats; 0.08–0.12 Hz in humans) peak, whereas in mice and dogs it has been described (Akselrod et al., '85; Ishii et al., '96) only a low-frequency (LF: 0.1–1.0 Hz in mice; 0.02–0.09 Hz in dogs) spectral peak.

In relation to the decrease of the lizard's RRI-

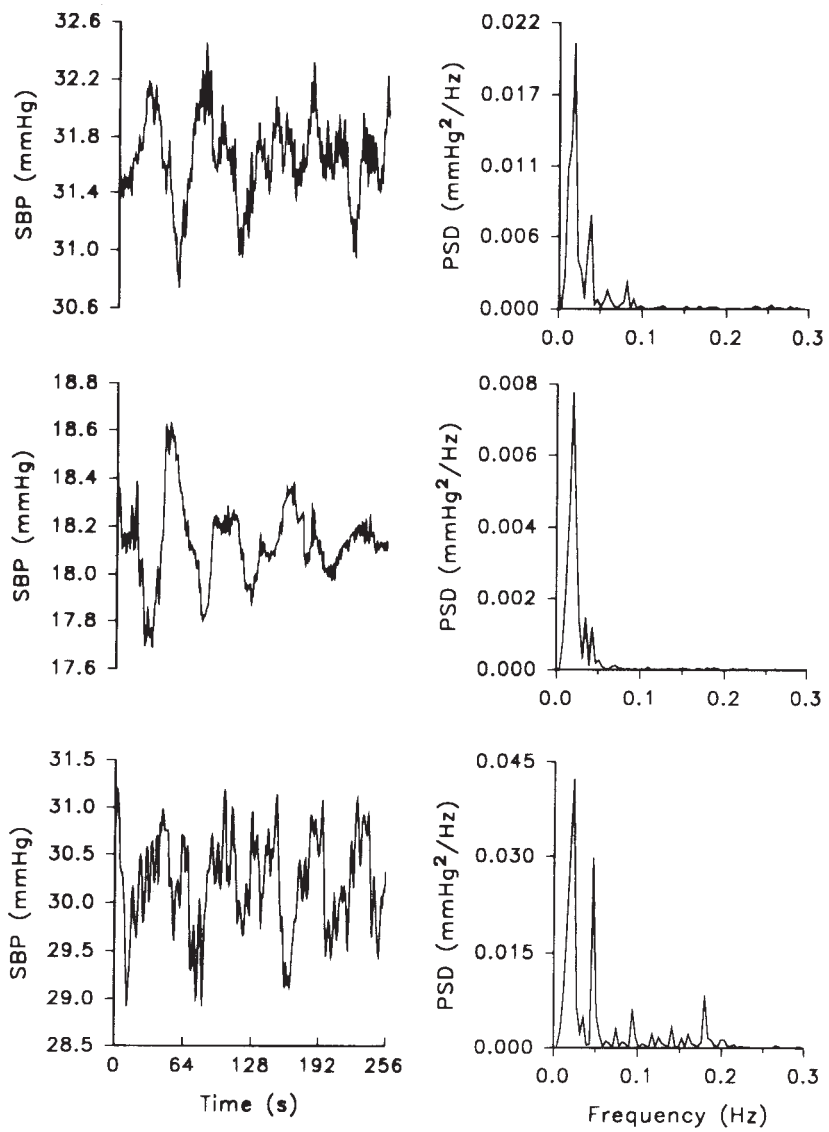

Fig. 3. An example of SBP 256-sec beat-to-beat time series (**left**) and their corresponding power spectra (**right**) obtained from the same lizard as Fig. 1 in control conditions (**top**), after α-adrenergic blockade with prazosin (**middle**) and after β-adrenergic blockade with propranolol (**bottom**). Time series are linear-trend removed. In order to improve visual resolution, power spectra have been cut down to the 0.3-Hz level where most of the power is concentrated.

VLF and RRI-LF oscillations after α-adrenergic blockade, our results are in agreement with some findings reported in rats (Murphy et al., '91; González et al., '95), but they are in disagreement with other results also described in rats (Japundzic et al., '90; Cerutti et al., '91), where no alteration of HR spectra has been found, and, in dogs (Akselrod et al., '85), where an increase in power in the LF band has been reported. According to the results of the present investigation, the activity of both RRI-VLF and RRI-LF oscillatory components seems to be α-adrenergic mediated, because their power decreased after blockade with prazosin; however, it has been demonstrated (De Vera and González, '97) that atropine also decreased the power of these fluctuations in approximately the same amount (~60%) and, therefore, it can be suggested that both RRI-VLF and RRI-LF spectral components of *Gallotia galloti* may be jointly α-adrenergic and parasympathetic mediated. But it must be taken into account that the decrease of RRI oscillations could also be attributed to a vagal inhibition in response to the decrease in ABP induced by the α-adrenergic blockade, as it has been suggested by Cerutti et al. ('91) to explain the decrease of the respiratory RRI oscillations of rats.

With regard to the increase of the lizard's RRI-

VLF and RRI-LF oscillations after β-adrenergic blockade, our findings are in agreement with some results reported for the dogs' and humans' LF HR oscillations (Akselrod et al., '85; Jokkel et al., '95), but they disagree with most findings in rats and mice, where a decrease in these oscillations has been reported (Japundzic et al., '90; Murphy et al., '91; González et al., '95; Ishii et al., '96). According to our results, it can be suggested that, in lizards, the β-adrenergic activity of the sympathetic system may act buffering the RRI low frequency oscillations, mainly those in the RRI-LF range. The physiology of the lizards we have studied in relation to the sympathetic-vagal modulation of RRI oscillations seems to be similar to that of humans and dogs, where the parasympathetic system could act strengthening these fluctuations and the β-sympathetic system acting instead as a buffer of them. However, in rats, whose HR is considerably greater than that of lizards at 23°C, humans, and dogs, it seems that there is not any sympathetic-vagal balance in the modulation of the RRI low frequency oscillations, because, in these mammals, the β-adrenergic blockade does not produce any buffer effect (Murphy et al., '91; González et al., '95).

With regard to the effects of the α-adrenergic blockade on SBP-VLF and SBP-LF oscillations, our results are in agreement with most findings reported in rats (Akselrod et al., '87; Japundzic et al., '90; Cerutti et al., '91; Persson et al., '92; González et al., '95). As prazosin decreased the power of both SBP-VLF and SBP-LF spectral bands by about the same amount (~80%), it seems that these ABP oscillatory components are α-adrenergic mediated; however, as occurred in the RRI spectra, it has been demonstrated (De Vera and González, '97) that atropine also decreased the power of SBP-LF oscillations of *Gallotia galloti* in almost the same quantity and it can therefore be suggested that while the SBP-VLF oscillatory component is solely α-adrenergic mediated, the SBP-LF component may be jointly α-adrenergic and parasympathetic mediated.

The β-adrenergic blockade increased the ABP very low- and low-frequency oscillations, in agreement with some findings described in dogs (Akselrod et al., '85), but in disagreement with most results obtained in rats, where no spectral alteration (Japundzic et al., '90; Murphy et al., '91) or decrease in the LF band (Cerutti et al., '91; González et al., '95) has been found. As propranolol increased the power of both SBP-VLF and SBP-LF components, it can be suggested that, in lizards, the β-adrenergic system may also act buffering these SBP oscillations.

In summary, lizards, like mammals, exhibit spontaneous short-term oscillations in their HR and SBP which are mediated by the ANS, a circumstance that is particularized in the following items: (1) the RRI-VLF, RRI-LF and SBP-LF oscillations could be both β-adrenergic and parasympathetic (De Vera and González., '97) mediated; (2) the SBP-VLF oscillation could solely be α-adrenergic mediated; (3) the α-adrenergic activity of the sympathetic system may act buffering all RRI and SBP oscillations; and (4) this buffer effect seems to be maximum at the RRI-LF band level, then at the SBP-VLF and SBP-LF bands level and, finally, at the RRI-VLF band level.

Finally, the dynamics of the ABP oscillations in *Gallotia galloti* seems to be mediated by the ANS in a similar way than that of RRI, where the parasympathetic and α-sympathetic activities appear to act driving these oscillations and the β-sympathetic buffering them. This simple and characteristic behavior of the lizards studied is different from that of occurring in mammals, in which more complicated feedforward and feedback interactions may exist between HR and ABP. In addition, it has been emphasized (Burnstock, '69; Cipolle et al., '86) that when analyzing components of autonomic receptors in nonmammalian species, drugs that have a specially defined action in mammals may not demonstrate the same action in other animal groups.

LITERATURE CITED

Akselrod S, Gordon D, Ubel FA, Shannon DC, Barger AC, Cohen RJ. 1981. Power spectrum analysis of heart rate fluctuations: a quantitative probe of beat-to-beat cardiovascular control. Science 213:220–222.

Akselrod S, Gordon D, Madwed JB, Snidman NC, Shannon DC, Cohen JR. 1985. Hemodynamic regulation: investigation by spectral analysis. Am J Physiol 249:H867–H875.

Akselrod S, Eliash S, Oz O, Cohen S. 1987. Hemodynamic regulation in SHR: investigation by spectral analysis. Am J Physiol 253:H176–H183.

Bendat JS, Piersol AG. 1971. Random data: analysis and measurements procedures. New York: Wiley-Inter-Science.

Berger PJ. 1971. The vagal and sympathetic innervation of the heart of the lizard, *Tiliqua rugosa*. Aust J Exp Biol Med Sci 49:397–404.

Burnstock G. 1969. Evolution of the autonomic innervation of visceral and cardiovascular systems in vertebrates. Pharmacol Rev 21:247–324.

Cerutti C, Gustin MP, Paultre CZ, Lo M, Julien C, Vincent M, Sassard J. 1991. Autonomic nervous system and cardiovascular variability in rats: a spectral analysis approach. Am J Physiol 261:H1292–H1299.

Cerutti S, Baselli G, Bianchi A, Signorini MG. 1993. Spectral techniques of analysis for blood pressure and heart rate signals. In: Di Rienzo M, Mancia G, Parati G, Pedotti A, Zanchetti A, editors. Blood pressure and heart rate variability. Amsterdam: IOS Press. p 39–55.

Cipolle MD, Zehr JE, Reinhart GA. 1986. Effects of autonomic agents on renin release in the turtle, *Pseudemys scripta*. Am J Physiol 251:R1103–R1108.

De Vera L, González J. 1986. Cardiac responses to temperature in the lizard *Gallotia galloti*. Comp Biochem Physiol 85A:389–394.

De Vera L, González J. 1997. Power spectral analysis of short-term RR interval and arterial blood pressure oscillations in lizard (*Gallotia galloti*): effects of parasympathetic blockade. Comp Biochem Physiol 118A:671–678.

Donald JA, O'Shea JE, Lillywhite HB. 1990. Somatostatin and the innervation of the heart of the ratsnake, *Elaphe obsoleta*. Am J Physiol 258:R1001–R1007.

González J, De Vera L. 1988. Spectral analysis of heart rate variability of lizard, *Gallotia galloti*. Am J Physiol 254: R242–R248.

González J, Cordero JJ, Feria M. 1995. Autonomic mediation of short-term cardiovascular oscillations after acute hemorrhage in conscious rats. J Auton Nerv Syst 55:123–130.

Hoffman BB, Lefkowitz RJ. 1991. Antagonistas de los receptores adrenérgicos. In: Gilman AG, Rall TW, Nies AS, Taylor P, editors. Las bases farmacológicas de la terapéutica de Goodman y Gilman. México D.F.: Editorial Médica Panamericana S.A. p 228–249.

Hohnke LA. 1975. Regulation of arterial blood pressure in the common green iguana. Am J Physiol 228:386–391.

Ishii K, Kuwahara M, Tsubone H, Sugano S. 1996. Autonomic nervous function in mice and voles (*Microtus arvalis*): investigation by power spectral analysis of heart rate variability. Lab Anim 30:359–364.

Japundzic N, Grichois ML, Zitoun P, Laude D, Elghozi JL. 1990. Spectral analysis of blood pressure and heart rate in conscious rats: effects of autonomic blockers. J Auton Nerv Syst 30:91–100.

Jokkel G, Bonyhay I, Kollai M. (1995) Heart rate variability after complete autonomic blockade in man. J Auton Nerv Syst 51:85–89.

Lillywhite HB, Seymour RS. 1978. Regulation of arterial blood pressure in australian tiger snakes. J Exp Biol 75:65–79.

Lillywhite HB, Donald JA. 1994. Neural regulation of arterial blood pressure in snakes. Physiol Zool 67:1260–1283.

Malliani A, Pagani M, Lombardi F. 1986. Positive feedback reflexes. In: Zanchetti A, Tarazi RC, editors. Handbook of hypertension: pathophysiology of hypertension. Amsterdam: Elsevier Science Publishing Co., Inc. p 69–81.

Malliani A, Pagani M, Lombardi F, Cerutti S. 1991. Cardiovascular neural regulation explored in the frequency domain. Circulation 84:482–492.

Murphy CA, Sloan RP, Myers MM. 1991. Pharmacologic responses and spectral analyses of spontaneous fluctuations in heart rate and blood pressure in SHR rats. J Auton Nerv Syst 36:237–250.

Nilsson S. 1983. Autonomic nerve function in vertebrates. In: Zoophysiology, vol. 13. Berlin: Springer.

Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, Sandrone G, Malfatto G, Dell'Orto S, Piccaluga E, Turiel M, Baselli G, Cerutti S, Malliani A. 1986. Power spectral analysis of heart rate and arterial blood pressure variabilities as a marker of sympathovagal interaction in man and conscious dog. Circ Res 59:178–193.

Pagani M, Montano N, Porta A, Malliani A, Abboud FM, Birkett C, Somers VK. 1997. Relationship between spectral components of cardiovascular variabilities and direct measures of muscle sympathetic nerve activity in humans. Circulation 95:1441–1448.

Parati G, Saul JP, Di Rienzo M, Mancia G. 1995. Spectral analysis of blood pressure and heart rate variability in evaluating cardiovascular regulation: a critical appraisal. Hypertension 25:1276–1286.

Persson PB, Stauss H, Chung O, Wittmann U, Unger T. 1992. Spectrum analysis of sympathetic nerve activity and blood pressure in conscious rats. Am J Physiol 263:H1348–H1355.

Pomeranz B, MaCauley RJB, Caudill MA, Kutz I, Adam D, Gordon D, Kilborn KM, Barger AC, Shannon DC, Cohen RJ, Benson H. 1985. Assessment of autonomic function in humans by heart rate spectral analysis. Am J Physiol 248:H151–H153.

Rompelman O. 1980. The assessment of fluctuations in heartrate. In: Kitney RI, Rompelman O, editors. The study of heart-rate variability. Oxford: Clarendon Press. p 59–77.

Saul JP, Rea RF, Eckberg DL, Berger RD, Cohen RJ. 1990. Heart rate and muscle sympathetic nerve variability during reflex changes of autonomic activity. Am J Physiol 258:H713–H721.

Singh CP, Sinha NK, Singh DK. 1991. Effect of propranolol on ECG of myna, *Acridotheres tristis*, and goh, *Varanus bengalensis*. Ind J Exp Biol 29:546–553.

Stinner JN, Ely DL. 1993. Blood pressure during routine activity, stress, and feeding in black racer snakes (*Coluber constrictor*). Am J Physiol 264:R79–R84.

White FN. 1976. Circulation. In: Gans AC, Dawson WR, editors. Biology of the reptilia, vol 5. Physiology. London: Academic Press. p 275–334.