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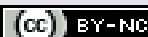
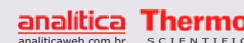
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# Time course of the hemodynamic responses to aortic depressor nerve stimulation in conscious spontaneously hypertensive rats

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## Abstract

The time to reach the maximum response of arterial pressure, heart rate and vascular resistance (hindquarter and mesenteric) was measured in conscious male spontaneously hypertensive (SHR) and normotensive control rats (NCR; Wistar; 18-22 weeks) subjected to electrical stimulation of the aortic depressor nerve (ADN) under thiopental anesthesia. The parameters of stimulation were 1 mA intensity and 2 ms pulse length applied for 5 s, using frequencies of 10, 30, and 90 Hz. The time to reach the hemodynamic responses at different frequencies of ADN stimulation was similar for SHR (N = 15) and NCR (N = 14); hypotension = NCR (4194 ± 336 to 3695 ± 463 ms) vs SHR (3475 ± 354 to 4494 ± 300 ms); bradycardia = NCR (1618 ± 152 to 1358 ± 185 ms) vs SHR (1911 ± 323 to 1852 ± 431 ms), and the fall in hindquarter vascular resistance = NCR (6054 ± 486 to 6550 ± 847 ms) vs SHR (4849 ± 918 to 4926 ± 646 ms); mesenteric = NCR (5574 ± 790 to 5752 ± 539 ms) vs SHR (5638 ± 648 to 6777 ± 624 ms). In addition, ADN stimulation produced baroreflex responses characterized by a faster cardiac effect followed by a vascular effect, which together contributed to the decrease in arterial pressure. Therefore, the results indicate that there is no alteration in the conduction of the electrical impulse after the site of baroreceptor mechanical transduction in the baroreflex pathway (central and/or efferent) in conscious SHR compared to NCR.

Key words: Electrical stimulation; Arterial pressure; Aortic depressor nerve; Baroreflex; Spontaneously hypertensive rats; Vascular tone

## Introduction

It has been well demonstrated that the arterial baroreflex acts, in particular, to buffer acute changes in arterial pressure by reciprocal modulation of the sympathetic and parasympathetic activities that control heart rate (HR) and vascular resistance (1,2). Remarkable attenuation of the baroreflex control of HR (3-6), of baroreceptor afferent sensitivity (7-11) and of baroreflex control of sympathetic nerve activity (12-14) in spontaneously hypertensive rats (SHR) has been thoroughly documented in the literature.

Studies evaluating the hemodynamic responses caused by electrical stimulation of the baroreceptors in anesthetized rats demonstrated that the hemodynamic responses were reduced in SHR, suggesting an alteration of the baroreflex circuitry (15,16). Nevertheless, in a recent study, Salgado

et al. (17) demonstrated that electrical stimulation of the aortic depressor nerve (ADN) in conscious SHR produced equivalent or even greater depressor responses compared to normotensive rats, indicating that conscious SHR exhibited a well-preserved baroreflex response to the electrical stimulation of the ADN, probably because this maneuver bypasses the mechanical transduction of the baroreceptors. Although Salgado et al. (17) reported a well-preserved baroreflex function elicited by electrical activation of the ADN in SHR, they did not compare the dynamic characteristics, i.e., the time course of the hemodynamic responses (arterial pressure, HR, and vascular resistance) to electrical stimulation of SHR and normotensive control rats (NCR).

Thus, it is possible that an anomalous time course of

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the hemodynamic responses could indicate a derangement of the conduction of the electrical impulse in the baroreflex pathway in SHR. Therefore, in the current investigation, we determined the time elapsed to reach maximum hypotension, bradycardia and the decrease in vascular resistance caused by electrical stimulation of the ADN in SHR and NCR.

## Material and Methods

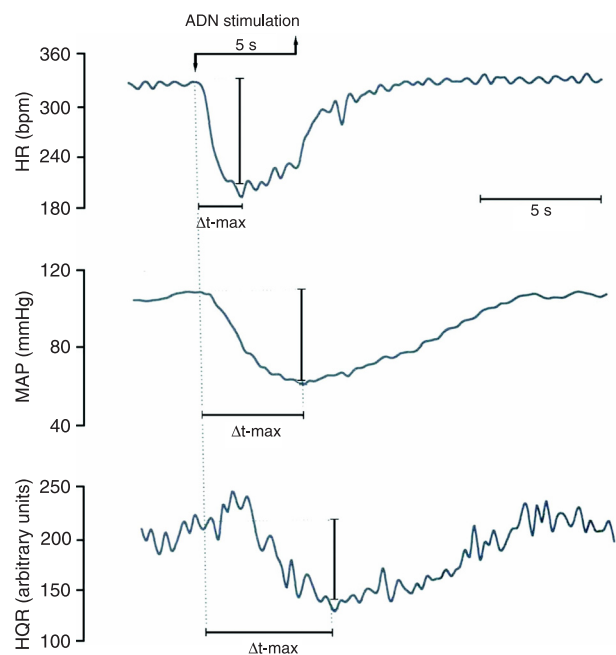
This is a retrospective analysis of previously published data (17). Male Wistar NCR and SHR 18-22 weeks of age (270-340 g) were used in the present study. The procedures were reviewed and approved by the Committee of Ethics in Animal Research of Faculdade de Medicina de Ribeirão Preto, Universidade de São Paulo (Protocol #040/2005).

The approach used to stimulate the left ADN and simultaneously record the arterial pressure, HR and regional blood flow has been described elsewhere (17-19). Briefly, under thiopental sodium anesthesia (40 mg/kg; *ip*), a 4- to 6-mm long portion of the left ADN was carefully isolated below the junction with the superior laryngeal nerve and placed on a bipolar platinum electrode with an inter-electrode distance of 2 mm. The correct identification of the nerve was confirmed by its typical pattern of discharge, which is synchronous with the arterial pulse pressure. The ADN was covered with a silicone impression material (Super-Dent; Carlisle Laboratories, USA). To ensure complete polymerization of the silicone impression material, the silicone that covered the ADN was left to stabilize for 30 min, and the activity of the nerve was again recorded to confirm the integrity of the signal. Once the integrity of the signal was confirmed, the fine platinum wires of the electrodes were exteriorized to the nape of the neck of the rats and soldered to a small plug connected to the electrical stimulator. While the animals were still under anesthesia the femoral artery was catheterized with polyethylene tubing (PE-50 and PE-10, Intramedic Becton Dickinson, USA) in order to record arterial pressure. In addition, a laparotomy was performed to place miniaturized Doppler flow probes (Iowa Doppler Products, USA) around the superior mesenteric artery or the inferior abdominal aorta to measure the changes in blood flow velocity and to calculate the mesenteric or hindquarter vascular resistance, respectively (18). Catheters and flow probes were also exteriorized along with the electrodes to the nape of the neck and the surgical incision sites were closed using sutures. Twenty-four hours after the end of surgery, the rats were connected to the recording system, which consisted of a pressure transducer (P23Gb; Statham Instruments, USA), a pulsed Doppler flowmeter (545C-4; Department of Bioengineering, The University of Iowa, USA) and an electrical stimulator (EMG/EP, N200/A; BioMed, Hungary). The signals, i.e., the pulsatile arterial pressure, mean arterial pressure (MAP), and regional (mesenteric or hindquarter) blood flow velocity, were fed to an IBM

personal computer equipped with a 12-bit analog-to-digital interface (CAD 12/36 Lynx Eletrônica, Brazil) and were continuously sampled (500 Hz). The HR was derived from the arterial pressure trace (pulse intervals), and the vascular resistances were calculated online as the ratio between the MAP and mean blood flow velocity using computer software (Advanced CODAS; Dataq Instruments, USA). Cardiovascular variables were recorded for at least 15 min before electrical stimulation of the ADN (1 mA, 2 ms pulse length, for 5 s) at 10, 30 and 90 Hz in a random sequence.

Each stimulus with a particular frequency was applied for a period of 5 s with intervals of at least 5 min. The difference between the pre-stimulation baseline level of the MAP, HR, and vascular resistance (% of basal) and the maximum change in these variables elicited by each frequency of ADN stimulation was quantified in milliseconds (Figure 1). The results of the maximum changes of each variable (MAP, HR and vascular resistance) have been published in a previous study from our laboratory (17). The protocol was successfully carried out in 14 NCR and 15 SHR.

Data are reported as means  $\pm$  SEM. The time elapsed for the cardiovascular variables to reach the maximum response due to ADN stimulation in SHR and NCR and pooled data was analyzed by repeated measures two-way ANOVA (groups: SHR vs NCR; pooled data: hemodynamic



**Figure 1.** Typical tracings from a normotensive control rat showing the time ( $\Delta t_{max}$ ) to reach the maximum decrease in heart rate (HR, upper panel), mean arterial pressure (MAP, middle panel) and hindquarter resistance (HQR, lower panel) for 5 s of electrical stimulation (1 mA amplitude, 2 ms duration, 30 Hz) of the aortic depressor nerve (ADN).

variables MAP vs HR vs vascular resistance; repeated measures: stimulus frequency). When ANOVA demonstrated statistical significance, the Tukey multiple comparison *post hoc* test was used to test for differences between means. Baseline values for NCR vs SHR were compared by the unpaired Student *t*-test. Differences were considered to be significant when  $P < 0.05$ .

## Results

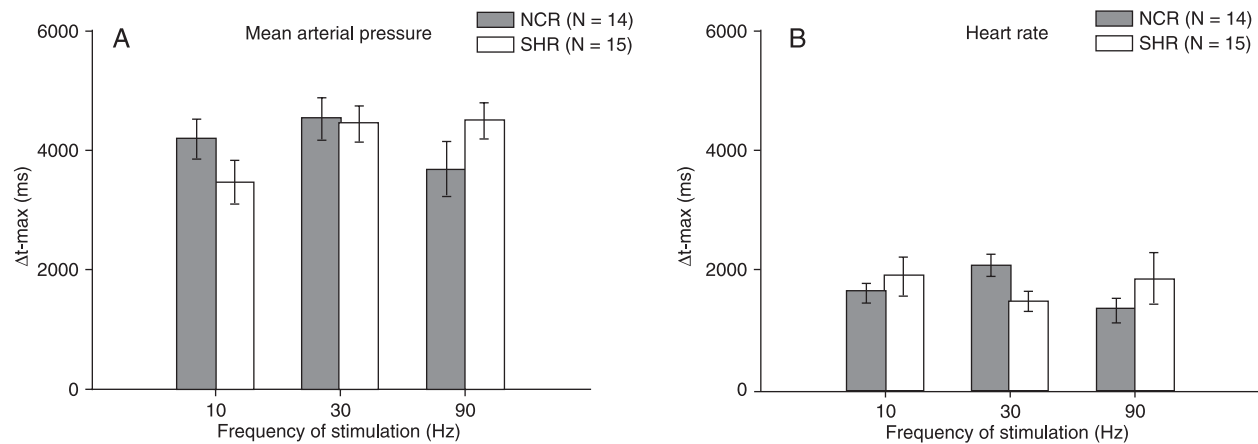
### Basal hemodynamics

The basal MAP and HR of SHR ( $150 \pm 5$  mmHg and  $393 \pm 9$  bpm, respectively) were higher than those of NCR ( $103 \pm 2$  mmHg and  $360 \pm 5$  bpm, respectively;  $P < 0.001$ ).

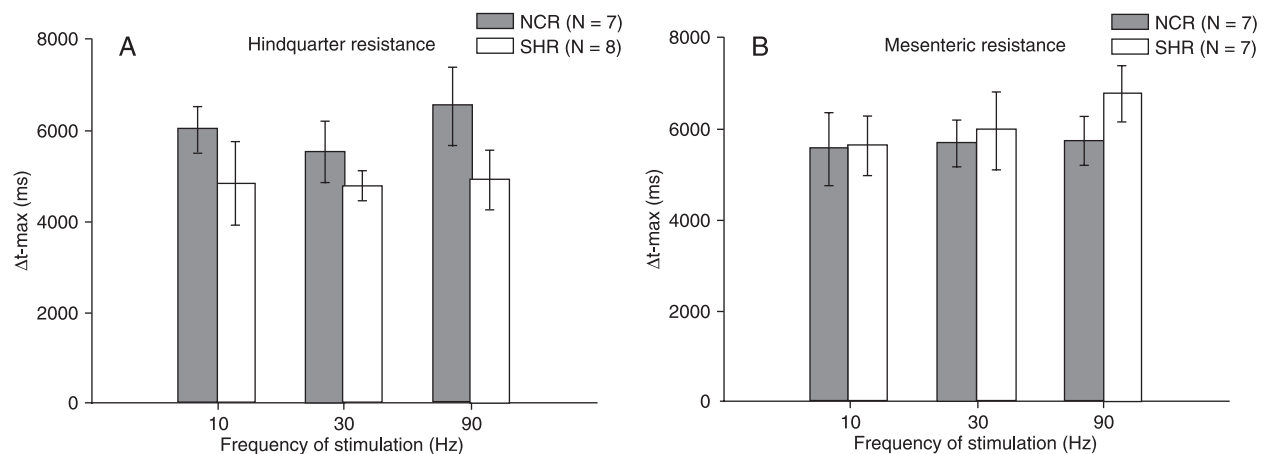
### Time course of the hemodynamic baroreflex responses

The bradycardia, hypotension and fall in hindquarter vascular resistance elicited by the electrical stimulation of the ADN in NCR are illustrated in Figure 1. The time elapsed for the MAP and HR response to reach their maximum value during ADN stimulation was similar in SHR and NCR for all frequencies studied (Figure 2). The time elapsed to reach the maximum response of vascular resistance in the hindquarter and mesenteric bed was similar for the two strains (Figure 3). Moreover, there was no difference between the SHR and NCR for either of the frequencies examined (Figure 3).

When NCR and SHR data were pooled, the time elapsed



**Figure 2.** Time elapsed to reach ( $\Delta t_{\text{max}}$ ) the maximum hypotensive (A) and bradycardic (B) response to electrical stimulation of the aortic depressor nerve in normotensive control rats (NCR) and spontaneously hypertensive rats (SHR) at different frequencies of stimulation (10, 30, 90 Hz). Data are reported as means  $\pm$  SEM (mean arterial pressure = group:  $P = 0.706$ , frequency:  $P = 0.093$ , group vs frequency:  $P = 0.070$ ; HR = group:  $P = 0.932$ , frequency:  $P = 0.843$ , group vs frequency:  $P = 0.137$ ; two-way ANOVA).



**Figure 3.** Time elapsed to reach ( $\Delta t_{\text{max}}$ ) the maximum decrease in hindquarter (A) and mesenteric (B) resistance to electrical stimulation of the aortic depressor nerve in normotensive control rats (NCR) and spontaneously hypertensive rats (SHR) at different frequencies of stimulation (10, 30, 90 Hz). Data are reported as means  $\pm$  SEM (hindquarter resistance = group:  $P = 0.056$ , frequency:  $P = 0.825$ , group vs frequency:  $P = 0.900$ ; mesenteric resistance = group:  $P = 0.523$ , frequency:  $P = 0.392$ , group vs frequency:  $P = 0.576$  (two-way ANOVA).

to reach the maximum decrease in mesenteric and hind-quarter vascular resistance (10 Hz =  $5529 \pm 351$ ; 30 Hz =  $5520 \pm 315$ ; 90 Hz =  $5999 \pm 355$  ms) was longer than the time elapsed to reach the maximum decrease in HR (10 Hz =  $1780 \pm 190$ ; 30 Hz =  $1764 \pm 131$ ; 90 Hz =  $1634 \pm 255$  ms;  $P < 0.001$  vs MAP and vascular resistance) and MAP (10 Hz =  $3794 \pm 252$ ; 30 Hz =  $4486 \pm 221$ ; 90 Hz =  $4142 \pm 270$  ms;  $P < 0.001$  vs HR and vascular resistance). These data demonstrate that the electrical stimulation of the ADN elicits a faster cardiac effect, followed by a vascular effect.

## Discussion

The present study demonstrates that there was no significant difference in the time elapsed for the baroreflex responses of MAP, HR, and vascular resistance to electrical stimulation of the ADN in SHR compared to NCR for any of the frequencies (10, 30, and 90 Hz) examined. In addition, when NCR and SHR data were pooled, it was also observed that the electrical stimulation of the ADN produced a prompt cardiac effect followed by a vascular effect. These findings indicate that, as expected, the baroreflex acted immediately upon the heart and later on the periphery (vessels) with a similar time course in SHR and NCR.

Previous reports have examined the time course of baroreflex activation using carotid sinus nerve stimulation combined with recording of cardiac vagal fiber activity (20,21). Kunze (20) demonstrated that the time elapsed for cardiac vagal fiber activation following carotid sinus nerve stimulation was 30-72 ms. In the present study, the bradycardic response occurred within 1.5 s after the start of ADN stimulation. Previous studies have demonstrated that the reflex bradycardia evoked by increases in arterial pressure is primarily mediated by rapid parasympathetic activation (22-25). Coleman (23) reported that the peak of the reflex bradycardia following the hypertensive response caused by phenylephrine was attained approximately 1 s after the maximum increase in arterial pressure. Thus, in the current study, the prompt bradycardia evoked by ADN stimulation in NCR and SHR is mainly attributable to rapid parasympathetic activation, which involves direct excitatory projection from the nucleus tractus solitarius to both the neurons of the nucleus ambiguus and the dorsal motor nucleus of the vagus (26,27).

On the other hand, the time elapsed to reach the maximum hypotensive response and the maximum decrease in vascular resistance in both groups was longer than the time to reach the bradycardic response. It has been demonstrated that these depressor responses are mainly caused by the reflex withdrawal of sympathetic activity (19,28) and a small contribution of other vasodilatory mechanisms, such as the activation of sympathetic fibers, which release nitric oxide (29). Thus, it is possible that the difference in the time elapsed to reach the bradycardic and vascular responses

in both groups of rats was due to the differential central processing of the baroreceptor input. It should also be noted that Coleman (23) demonstrated that the reflex withdrawal of sympathetic activity has a slower onset compared to parasympathetic activation.

Afferent baroreceptor sensitivity (7-10) and baroreflex-mediated changes in HR (3,4) have been shown consistently to be impaired in SHR. The decreased baroreflex sensitivity for the control of the HR has been primarily attributed to a derangement of parasympathetic function (3,4,30-32). Nevertheless, in the present study, no difference was observed between the time elapsed to reach reflex bradycardia in SHR and NCR, indicating that the baroreflex parasympathetic pathway is preserved in SHR.

Moreover, it was also observed that the time elapsed to reach maximum hypotensive and vasodilatory responses, mainly caused by withdrawal of the sympathetic vasoconstrictor tone, was similar in NCR and SHR. Several studies have evaluated the baroreflex control of sympathetic function by recording the renal sympathetic nerve activity in SHR (12-14,33,34). However, conflicting results have been reported. There are reports of decreased (12,14), normal (33,34), and increased (13) baroreflex control of sympathetic nerve activity in SHR. However, studies that evaluated the dynamic characteristics of baroreflex regulation of sympathetic activity using transfer functions reported no change in baroreflex control in SHR compared to Wistar-Kyoto rats (34,35). Thus, based on the results obtained in the current study, we propose that the transmission of the inhibitory baroreceptor input throughout the sympathetic pathway is not altered in SHR compared to NCR.

The electrical stimulation of afferent fibers bypasses the site of baroreceptor mechanosensory transduction. Therefore, the reflex response to ADN stimulation provides information about the central processing of the afferent input and the properties of the central and efferent components of the baroreflex. In a previous study from our laboratory, it was demonstrated that electrical stimulation of the ADN of conscious SHR produced equivalent or greater depressor responses compared to NCR, suggesting that the central and efferent components of the baroreflex were preserved in SHR (17). These data suggested that there is no change in the transmission of the electrical impulse through the central/efferent baroreflex pathway of SHR. Thus, if there is some alteration in the baroreflex of SHR, it could be in the mechanosensory transduction of the baroreceptors, as reported previously (7-11).

In conclusion, electrical stimulation of the ADN demonstrated no differences in the time elapsed to reach the maximum responses of MAP, HR and vascular resistance in conscious SHR compared to NCR. In addition, the results also demonstrate that electrical stimulation of the ADN produced a prompt cardiac effect followed by a vascular effect, with a quite similar time course of the baroreflex response

in both the SHR and NCR. Therefore, these data indicate that the central/efferent processing of the baroreceptor input, which is elicited by electrical stimulation of the ADN, is well preserved in SHR.

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