Arterial baroreflex resetting mediates postexercise reductions in arterial pressure and heart rate

MARGARET P. CHANDLER, DAVID W. RODENBAUGH, AND STEPHEN E. DiCARLO
Department of Physiology, Wayne State University, School of Medicine, Detroit, Michigan 48201

Chandler, Margaret P., David W. Rodenbaugh, and Stephen E. DiCarlo. Arterial baroreflex resetting mediates postexercise reductions in arterial pressure and heart rate. Am. J. Physiol. 275 (Heart Circ. Physiol. 44): H1627–H1634, 1998.—We tested the hypothesis that postexercise reductions in arterial pressure and heart rate (HR) are mediated by a lowering of the operating point and a reduction in the gain of the arterial baroreflex. To test this hypothesis, spontaneous changes in arterial pressure and the reflex responses of HR were examined before and after a single bout of mild to moderate dynamic exercise in 19 spontaneously hypertensive rats (SHR, 10 male and 9 female). Eleven SHR subjected to sinoaortic denervation (SAD) (6 male, 5 female) were also studied. All rats were instrumented with an arterial catheter for the measurement of arterial pressure and HR. After exercise, arterial pressure and HR were reduced below preexercise levels. Furthermore, the operating point and spontaneous gain (G) of the arterial baroreflex were reduced. Specifically, after exercise, the spontaneous range of HR (P1, 50%), the pressure at the midpoint of the pressure range (P3, 13%), and the HR at the midpoint of the HR range (H3, 10%), the spontaneous minimum HR (P4, 8%) and maximum HR (10%), and G (76%) were significantly attenuated. SAD significantly attenuated the relationship between arterial pressure and HR by reducing G (males 94%, females 95%). These results demonstrate that acute exercise resulted in a postexercise resetting of the operating point and a reduction in the gain of the arterial baroreflex. Furthermore, these data suggest that postexercise reductions in arterial pressure and HR are mediated by a lowering of the operating point of the arterial baroreflex.

spontaneous arterial baroreflex; hypertension; postexertional hypotension; exercise

THE ARTERIAL BAROREFLEX has two important functions. First, the arterial baroreflex is a negative feedback reflex that regulates arterial pressure around a preset value called a set or operating point. Second, the arterial baroreflex also establishes the prevailing systemic arterial pressure by setting the operating point (36). That is, modulating the response of barosensitive neurons in the central nervous system establishes the operating point or prevailing arterial pressure (13, 35). Therefore, the operating point of the arterial baroreflex is not fixed but is variable over a wide range of pressures and is determined by a variety of inputs from the peripheral and central nervous systems (36).

Raising the operating point of the arterial baroreflex mediates the simultaneous increase in arterial pressure, heart rate (HR), and sympathetic nerve activity (SNA) during exercise (13, 25) and spontaneous motor activity (postural changes, exploration, grooming, and eating) (26). Similarly, it was suggested that a lowering of the operating point of the arterial baroreflex mediates the postexercise reductions in arterial pressure (postexertional hypotension, PEH), HR, and cardiac sympathetic tonus in hypertensive rats (6). This was suggested because sinoaortic denervation (SAD) prevented PEH and sympathoinhibition in the spontaneously hypertensive rats (SHR). It was suggested that if the mechanisms mediating PEH operated independently of the arterial baroreflex, then SAD should have elicited an exaggerated postexercise reduction in arterial pressure (6).

Therefore, this study was designed to test the hypothesis that postexercise reductions in arterial pressure are mediated by a lowering of the operating point of the arterial baroreflex. This was accomplished by recording spontaneous changes in arterial pressure and the reflex responses in HR before and after a single bout of dynamic exercise in SHR.

METHODS

Design

Nineteen SHR (10 male and 9 female) were weaned at 4 wk of age and were housed in standard rat cages at all times. Between 12 and 13 wk of age all rats were instrumented with arterial catheters and were subsequently allowed 3–4 days to recover. After recovery, spontaneous arterial baroreflex regulation of HR was assessed under two experimental conditions, preexercise and postexercise. Spontaneous arterial baroreflex regulation of HR was also assessed in 11 (6 male and 5 female) SAD rats to verify the role of the arterial baroreflex in the reflex control of HR. The mean recovery time for the SAD rats was 8 ± 3 days. All procedures were performed in accordance with the guidelines established by the institutional animal care and use committee.

Surgical Procedures

Intact group. All instrumentation was performed using aseptic surgical procedures. The rats were anesthetized with an intramuscular injection of ketamine hydrochloride (40 mg/kg), xylazine (8 mg/kg), and chlorpromazine hydrochloride (4 mg/kg). Supplemental doses were administered as needed. All rats were instrumented with a Teflon catheter inserted into the descending aorta via the left common carotid artery for measurements of arterial pressure and HR. The arterial catheter was flushed daily, filled with heparin (1,000 U/ml), and plugged with a paraffin-filled obturator. Rats were carefully monitored for signs of infection and changes in body weight during the recovery period. During this time, the rats were familiarized with the treadmill and experimental procedures during three or four training sessions. The familiarization sessions ensured that the experimental procedures would not be novel to the rat and that the rat would run without the use of aversive stimuli. At the time of the experimental
protocols, all rats had recovered, were healthy, and were gaining weight.

SAD group. Four rats from the intact group and seven additional rats underwent complete SAD procedures. The additional rats were instrumented with arterial catheters (as described for the intact group). Subsequently, all rats were anesthetized as described for the intact group, an anterior cervical incision was made, and the carotid arteries were isolated at the region of the carotid sinus. All nerves and tissue were stripped from the sinus, the carotid artery, and all branches above and below the area of the sinus. The region was painted with 10% phenol in alcohol. The aortic depressor nerves were isolated bilaterally and sectioned.

Experimental Measurements

Arterial pressure was determined by connecting the arterial catheter to a Gould P23 XL pressure transducer coupled to a MacLab BRIDGE Amplifier. Arterial pressure analog signals were digitized at 200 samples/s by a MacLab 8 analog-to-digital converter and laboratory computer (Macintosh Performa) for calculation of real-time HR and for subsequent mean arterial pressure (MAP) analysis.

Experimental Protocol

On the day of the experiment, each rat was allowed to adapt to the laboratory environment for 1 h so that baseline hemodynamic variables could be obtained. After a 1-h adaptation period, spontaneous arterial baroreflex responses were recorded every 10 min for a period of 4 min. The mean time for data collection after the 1-h adaptation period was 28 ± 5 and 34 ± 4 min for male and female SHR, respectively. At the end of the preexercise period, the rats ran on a motor driven treadmill at 12 m/min, 10% grade, for 40 min. Blood pressure and HR were recorded every 10 min during exercise. Postexercise hemodynamic and spontaneous arterial baroreflex responses were recorded for an additional 60 min. Postexercise arterial pressure and the reflex responses in HR were recorded from 20 to 60 min after exercise so that the arterial baroreflex was assessed at a time when postexertional hypotension was present (7). The mean time for postexercise baroreflex assessment was 34 ± 4 and 41 ± 4 min for male and female SHR, respectively.

The SAD group was studied only in the preexercise condition. Resting hemodynamic and spontaneous arterial baroreflex responses were recorded every 10 min for a period of 4 min.

Evaluation of Spontaneous Arterial Baroreflex Regulation of HR

We modified procedures from previous reports (4, 5, 18, 33) to assess the spontaneous arterial baroreflex regulation of HR. Absolute values for HR (in beats/min) were used to evaluate the capability of the arterial baroreflex to increase or decrease HR during spontaneous changes in arterial pressure. Raw data points were collected on a beat-to-beat basis. For the analysis of the relationship between systolic blood pressure (SBP) and the reflex responses in HR, the computer recorded SBP for each heartbeat. This pressure was then plotted against the HR recorded during the subsequent heartbeat at the point of the diastolic pressure (18). The number of heartbeats sampled ranged from 400 to 600 beats. To illustrate the results of this analysis and the "goodness of fit," actual data points during a preexercise period and after sinoaortic denervation in one animal are presented (see Fig. 2). Data points representing the spontaneous changes in arterial pressure and corresponding reflex changes in HR were fit by a linear regression with HR being regressed on SBP. The slope of the regression line was used as an index of the overall spontaneous arterial baroreflex sensitivity or gain (G) for each animal under each condition. The method for determining spontaneous arterial baroreflex function parameters for the arterial baroreflex regulation of HR is also presented (see Fig. 2). P1 represents the spontaneous range of HR, P2 is the spontaneous minimum HR response, and the maximum HR response (spontaneous maximum) is the sum of P3 and P4. P5 and P6 represents the pressure at the midpoint of the spontaneous pressure range, and H3 represents the HR at the midpoint of the spontaneous HR range. Actual data points illustrate the relationship between SBP and HR in the pre- and postexercise conditions for a male and a female rat (see Fig. 3).

The absolute data were then plotted as group means to determine the relationship between SBP and HR. The data points for SBP were divided into 5-mmHg bins for each curve under both conditions. Each of these pressure points and the corresponding HR were then averaged and plotted for each of the four groups (see Fig. 4).

Data Analysis

All data are expressed as means ± SE. A two-way ANOVA was used for determining differences between the male and female intact and SAD animals for each of the following variables: age, body weight, resting HR, and resting MAP (see Table 2). Significant interactions observed by ANOVA were further evaluated by a Bonferroni post hoc analysis. A two-way ANOVA with repeated measures was used to evaluate 1) MAP before, during, and after exercise and 2) HR before, during, and after exercise, between male and female SHR. A two-way ANOVA with repeated measures (gender × exercise) was also used to evaluate the differences in spontaneous arterial baroreflex function parameters (P1, P2, H3, P4, maximum HR, and G for the arterial baroreflex regulation of HR) between groups (see Table 2). Significant interactions observed by ANOVA were further evaluated using a test of simple-effects post hoc analysis (37). An α-level of 0.05 was used to determine statistical significance.

RESULTS

MAP before, during, and after exercise is presented in Fig. 1A for male and female SHR. MAP was significantly decreased 10 min after exercise in male (12 ± 5 mmHg, P < 0.05) and 20 min after exercise in female (14 ± 3 mmHg, P < 0.05) SHR. PEH persisted for the duration of the postexercise period. Although the preexercise MAP responses were not different between the male and female rats, male SHR had significantly greater MAP responses at 20 and 30 min during exercise (P < 0.05).

HR before, during, and after exercise is presented in Fig. 1B for male and female SHR. Female SHR had significantly higher HR during the preexercise period compared with male rats (345 ± 7 vs. 303 ± 8 beats/min; P < 0.05). In contrast, HR responses during the entire 40 min of exercise were significantly higher in the male SHR (mean HR during exercise 511 ± 5 vs. 471 ± 5 beats/min). Forty minutes after exercise, HR significantly decreased in female SHR (18 ± 5 beats/min; P < 0.05). Fifty minutes after exercise, HR significantly decreased (12 ± 5 beats/min; P < 0.05) in
male SHR. This bradycardia persisted for the duration of the postexercise period.

Table 1 presents age, body weight, resting HR, and resting MAP during the preexercise protocol in male and female intact and SAD rats. There were no significant differences in age or resting MAP between the four groups. There were significant main effects of gender for body weights and resting HR. Female rats had lower body weights than male rats (40%, P < 0.05). Resting HR was significantly greater in female versus male rats (P < 0.05).

Figure 2 presents actual data points that illustrate the relationship between spontaneous changes in SBP and reflex responses in HR for a preexercise period in the intact and SAD conditions. SAD significantly attenuated the relationship between arterial pressure and HR by reducing the spontaneous gain (males 94%, females 95%). This figure also illustrates how the arterial baroreflex function parameters were obtained from data for each animal.

Figure 3 presents actual data points that illustrate the relationship between spontaneous changes in SBP and reflex responses in HR during a preexercise and postexercise period in one male and one female SHR. Data from each animal were fit by a linear regression with HR being regressed on SBP. A single bout of dynamic exercise significantly attenuated the spontaneous gain and the correlation coefficient of the spontaneous arterial baroreflex regulation of HR in both a male and a female SHR. Furthermore, in the preexercise condition, females had a significantly greater spontaneous gain for the arterial baroreflex control of HR than did male SHR.

Figure 4 presents the group means for the spontaneous arterial baroreflex regulation of HR in the preexercise and postexercise conditions in male and female SHR. These results indicate that a single bout of dynamic exercise altered the spontaneous arterial baroreflex regulation of HR similarly in male and female SHR. After exercise, both arterial pressure and

| Table 1. Age, body weight, and resting heart rate and mean arterial pressure in male and female intact and sinoaortic-denervated rats |
|---|---|---|---|
| | Male | Female | |
| | Intact | SAD | Intact | SAD |
| n | 10 | 6 | 9 | 5 |
| Age, days | 86 ± 1 | 88 ± 3 | 85 ± 1 | 84 ± 2 |
| Body wt, g | 332 ± 5 | 315 ± 9 | 184 ± 2* | 180 ± 4* |
| Resting HR, beats/min | 312 ± 8 | 321 ± 9 | 345 ± 7* | 335 ± 9* |
| Resting MAP, mmHg | 164 ± 4 | 167 ± 5 | 170 ± 4 | 172 ± 6 |

Values are means ± SE; n, no. of rats. SAD, sinoaortic denervation. There were no differences in age or resting mean arterial pressure (MAP) between the 2 conditions. Female rats had lower body weight than male rats (40%, P < 0.05). Resting heart rate (HR) was significantly greater in female vs. male rats (P < 0.05). *P < 0.05, male vs. female.

Fig. 2. Actual data points illustrating relationship between spontaneous changes in systolic blood pressure (SBP) and reflex changes in HR for a preexercise period in intact and sinoaortic denervation (SAD) conditions. SAD significantly attenuated relationship between arterial pressure and HR by reducing gain (males 94%, females 95%). Arterial baroreflex function parameters [spontaneous range of HR (P1), pressure at midpoint of pressure range (P3), HR at midpoint of HR range (H3), spontaneous minimum response of HR (P4), spontaneous maximum response of HR (Max, P1 + P4), and spontaneous gain of arterial baroreflex regulation of HR (G)] were determined from actual data points for each animal.
HR were lower in male and female rats. The spontaneous arterial baroreflex was operating at a lower pressure and HR with a reduced gain.

Table 2 presents the spontaneous arterial baroreflex function parameters for both male and female rats in the preexercise and postexercise conditions. Female SHR had significantly greater spontaneous range ($P_1$, 37%), midpoint of the HR range ($H_3$, 10%), spontaneous minimum HR ($P_4$, 14%), spontaneous maximum HR (16%), and spontaneous slope (G, 47%) compared with male SHR in the preexercise condition. After a single bout of dynamic exercise, male and female SHR had reduced $P_1$ (46 and 54%), $P_3$ (15 and 10%), $H_3$ (10 and 10%), $P_4$ (7 and 9%), maximum HR (12 and 7%), and G (75 and 76%). These results demonstrate that a single bout of dynamic exercise shifts the operating point of the arterial baroreflex function downwards and to the left and this shift was accompanied by a reduction in the spontaneous range, maximum, and G in both male and female SHR.

DISCUSSION

PEH is most often associated with no change or a reduction in HR and cardiac (6, 7, 9, 10) and peripheral SNA (17, 19). At least two possibilities could account for a reduction in arterial pressure without a compensatory tachycardia or sympathetic system activation (14). First, the arterial baroreflex may have a reduced gain or sensitivity after exercise, i.e., the reduction in arterial pressure is no longer a sufficient stimulus to elicit a reflex tachycardia or sympathetic excitation. A second possibility is that a single bout of dynamic exercise resets the operating point of the arterial baroreflex to a lower pressure so that it now operates (considers "normal") around the new lower pressure.

Results from this study demonstrate that a single bout of dynamic exercise reset the operating point and reduced the spontaneous gain of the arterial baroreflex control of HR. Specifically, after exercise both arterial pressure and HR were reduced below preexercise levels (see Fig. 1). Furthermore, the spontaneous range, the pressure at the midpoint of the pressure range, the HR at the midpoint of the HR range, the spontaneous minimum and spontaneous maximum HR, and the spontaneous gain of the arterial baroreflex control of HR were reduced after exercise. These data suggest that PEH is mediated by both a reduction in gain and a resetting of the operating point of the arterial baroreflex. The concept of arterial baroreflex resetting mediating changes in arterial pressure and HR is not new. A number of investigators (31, 32, 34) have suggested that an upward resetting of the arterial baroreflex mediated the sympathoexcitatory response associated with exercise. Similarly, Ludbrook and Potocnik (26) reported that the hemodynamic responses associated with spontaneous motor activity are also mediated by changing the operating point of the arterial baroreflex. Our results are an extension of these studies, in that lowering the operating point of...
the arterial baroreflex mediated the postexercise reductions in arterial pressure and HR.

**Influence of SAD on Arterial Baroreflex Regulation of HR**

To verify that the arterial baroreflex was responsible for the reflex responses in HR after spontaneous changes in arterial pressure, we compared arterial baroreflex function between intact and SAD rats. SAD significantly attenuated the relationship between arterial pressure and HR by reducing the gain and the correlation coefficient of the linear regression analysis. Thus the arterial baroreflex is required for the reflex responses in HR after spontaneous changes in arterial pressure.

**Autonomic Mechanisms Mediating Spontaneous Baroreflex Responses**

There is a physiological temporal relationship between the fast-acting vagal and the slower-responding sympathetic components of the arterial baroreflex regulation of HR. Specifically, although changes in sympathetic and parasympathetic nerve activity occur at virtually the same time, end-organ responses to sympathetic stimulation are considerably delayed. Furthermore, it is well documented that the initial HR response to an alteration in arterial pressure is predominantly vagally mediated. However, it is important to note that this vagal response is modulated by the background level of sympathetic activity, e.g., the concept of accentuated antagonism (24). Coleman (see Fig. 3 in Ref. 12) demonstrated that the reflex HR response to an increase in pressure was eliminated after vagal blockade, leading to the interpretation that the reflex change in HR is solely a vagally mediated response with no contribution from sympathetics. However, a careful look at Coleman’s Fig. 3 demonstrates that sympathetic blockade attenuated the reflex HR response to an increase in arterial pressure. Taken together, Coleman’s data suggest that the sympathetic nervous system is actually modulating the vagal response to a change in pressure. This is an important consideration when evaluating methods that assess arterial baroreflex function. It has been suggested that a change in arterial pressure must be held constant for 10–20 s to fully engage end-organ responses from the sympathetic component of the arterial baroreflex. However, holding the change in pressure constant for 10–20 s may disrupt the physiological temporal relationship between the sympathetic and vagal components of the arterial baroreflex. That is, vagal responses occur immediately and subsequently wane at the time when the sympathetic component is fully engaged (12, 38).

However, the spontaneous arterial baroreflex method maintains the physiological temporal relationship between the sympathetic and parasympathetic nervous systems. That is, a change in arterial pressure mediates a predominantly vagal response that is modulated by the baseline level of sympathetic activity in the physiological temporal sequence. In this study, changes in HR therefore reflect a combined parasympathetically and sympathetically mediated response.

**Potential Mechanisms for Postexercise Arterial Baroreflex Resetting**

Although not investigated in this study, a shifting of the operating point of the arterial baroreflex may occur centrally at the nucleus tractus solitarii (NTS) by altering the response of barosensitive neurons. An alteration of barosensitive neurons may occur as a result of concurrent afferent input from a number of peripheral receptor groups such as cardiopulmonary (CP) baroreceptors or muscle afferents. Mifflin and Felder (28) reported that some single NTS neurons receive afferent input from more than one cardiovascular afferent nerve. An alteration in the response of barosensitive neurons could be mediated by input from one or more of these peripheral sites. Specifically, both CP and muscle afferents were shown to alter arterial baroreflex function and both afferent groups were implicated in the mechanisms mediating PEH (14, 39). Afferent input from either CP or muscle receptors could alter barosensitive neurons in the NTS, resulting in elevated NTS activity for any given arterial baroreceptor input. This elevated NTS activity could reset the operating point of the arterial baroreflex to a lower pressure.

**Influence of Acute Exercise on Postexercise HR Regulation**

A number of studies have examined the influence of a single bout of dynamic exercise on the arterial barore-
flex regulation of HR in normotensive humans and animals. These studies reported a postexercise decrease (16, 40) or an increase in the gain of arterial baroreflex regulation of HR (20). Furthermore, Halliwell and colleagues (19) reported a postexercise reduction in arterial pressure and muscle SNA, which is consistent with a resetting of the arterial baroreflex regulation of sympathetic outflow. However, because of the differences in autonomic regulation between hypertensive and normotensive populations (23), we will focus on the responses in hypertensive subjects.

Postexercise reductions in arterial pressure and peripheral resistance (11, 21) occur without a corresponding increase in HR (3), muscle SNA, or plasma norepinephrine concentrations (21) in individuals with hypertension. These data suggest that acute exercise mediated a postexercise resetting of the arterial baroreflex and possibly a reduction in the gain. Although no reports have directly examined arterial baroreflex function after dynamic exercise in hypertensive individuals or animals, the response to lower body negative pressure (LBNP) has been investigated. LBNP unloads both arterial and CP receptors. Furthermore, only the responses to a hypotensive challenge are examined, and thus only the sympathoexcitatory and vagoinhibitory responses are elicited (15). Investigators reported either no change (11) or an increase in the gain of the baroreflex regulation of HR and vascular resistance in response to LBNP (3).

Influence of Gender on Arterial Baroreflex Regulation of HR

Female SHR had a significantly greater resting HR than male SHR. Furthermore, the HR range, HR at the midpoint of the HR range, maximum and minimum HR, and maximum gain of the spontaneous arterial baroreflex control of HR were higher in female vs. male SHR. However, there were no differences in the reductions in spontaneous baroreflex responses after acute exercise between male and female SHR. A gender influence on resting HR (1, 10) and for the arterial baroreflex regulation of HR has been previously reported by a number of investigators (1, 8, 22). Chen and DiCarlo (8) reported a higher gain and maximum HR in normotensive female compared with male rats. Similarly, Hudson and colleagues (22) reported that females had a significantly higher baroreflex sensitivity than males during LBNP. In contrast, females had a lower gain and range for vagally mediated bradycardia compared with males during increases in arterial pressure induced by bolus injections of phenylephrine (1). The mechanisms responsible for the gender-related differences in arterial baroreflex regulation of HR are unknown and merit further investigation.

Assessment of Perturbational Methods for Determining Arterial Baroreflex Function

Numerous investigators have examined the arterial baroreflex regulation of HR using spontaneous fluctuations in arterial pressure and the reflex responses in HR from sequences of three to six consecutive beats (4, 5, 33). However, Frankel and colleagues (18) were the first investigators to assess the arterial baroreflex regulation of HR by recording continuous spontaneous fluctuations in arterial pressure and the corresponding reflex changes in HR over several hundred consecutive beats. Although we adopted this methodology because of a number of theoretical and practical concerns regarding the more “traditional” methods for assessing cardiac baroreflex sensitivity, i.e., methods that require mechanical or pharmacological manipulations to alter arterial pressure (18), it is important to acknowledge that there is no perfect method for assessing the arterial baroreflex.

For example, the vasoactive drug method uses pharmacological agents that can differentially affect the relationship between baroreceptor activity and pressure caused by changes in the coupling between the receptors and the vascular smooth muscle (29). It is also important to note that baroreceptors respond to the average arterial pressure as well as the rate of change of arterial pressure (36). Nitric oxide (NO) donors reduce pulse pressure (PP) as well as arterial pressure, whereas α-agonists increase PP as well as arterial pressure. However, the reduction in PP with NO donors magnifies the decrease in arterial pressure to a greater extent than the corresponding increase in PP magnifies the increase in arterial pressure with α-agonists (2). Furthermore, the time constants for sympathetic and parasympathetic responses are significantly different and are influenced by the experimental procedure (12). Thus it is not surprising that the method of drug administration determines the baroreflex responses. Therefore, results obtained from the bolus injection, steady-state infusion, or ramp infusion methods cannot be compared because of the different influences on PP and rate of change of pressure and vastly different time constants.

Recent evidence also suggests that vasoactive substances have a direct effect on both the heart and central nervous system. Systemic administration of phenylephrine and/or NO donors (e.g., nitroglycerin or sodium nitroprusside [SNP]) causes changes in HR that reflect a baroreflex-mediated response to changes in arterial pressure. These vasoactive substances also have a direct effect on the heart, independent of the arterial baroreflex. NO increased the spontaneous beating rate in isolated sinoatrial node preparations (30). Furthermore, α-agonists have a direct chronotropic influence on the heart (41). Thus changes in HR in response to vasoactive substances reflect an arterial baroreflex-mediated response and a direct effect on the heart.

Similarly, vasoactive substances act centrally to influence cardiovascular regulation. NO can directly increase the spontaneous discharge rate of NTS neurons, independent of alterations in arterial pressure (27). Similarly, microinjections of SNP into the paraventricular nucleus (PVN, a site of central integration of autonomic cardiovascular responses) elicited a significant decrease in renal SNA, arterial pressure, and HR.
(42). Thus, by directly altering the excitability of NTS and/or PVN neurons, NO has a direct effect on central autonomic regulation of the cardiovascular system. These data suggest that changes in HR are a result of baroreflex-mediated responses to changes in arterial pressure as well as a direct effect on the heart and central nervous system. Taken together, these results document the need for a method to examine arterial baroreflex responses that are consistent and not subjected to the multiple effects of vasoactive agents.

Finally, there are no methods that selectively alter arterial baroreflex function. For example, the hemodynamic effects of vasoactive drugs (e.g., venous pooling and elevations in afterload) elicit CP responses. Similarly, vascular occluders also cause venous pooling and elevations in afterload. Furthermore, the responses obtained by altering carotid sinus pressure via the neck collar method are opposed by intact aortic baroreceptors (15).

Similarly, the spontaneous method for assessing the arterial baroreflex is also subject to specific limitations. It is possible that spontaneous changes in central venous pressure and respiratory gating of baroreflex sensitivity may have a greater impact on nonperturbational baroreflex determinations than when large changes in arterial pressure are induced pharmacologically. In addition, this method does not allow for an assessment of arterial baroreflex function through a large range of pressures (i.e., from threshold to saturation) as is possible with other perturbational methods. Thus comparisons of spontaneous arterial baroreflex function parameters with arterial baroreflex function parameters reported in previous studies are not appropriate, because these parameters were derived under entirely different physiological conditions.

In summary, results from this study demonstrate that a single bout of dynamic exercise reset the operating point and reduced the gain of the arterial baroreflex control of HR. A shifting of the operating point of the arterial baroreflex may occur centrally at the NTS by altering the response of barosensitive neurons, resulting in an elevated NTS activity. Similarly, an alteration of barosensitive neurons could result in a reduced NTS response for any given change in arterial pressure. The results from this study suggest that postexercise reductions in arterial pressure and HR are mediated by both a reduction in gain and a resetting of the operating point of the arterial baroreflex to a lower pressure.

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Address for reprint requests: S. E. DiCarlo, Dept. of Physiology, Wayne State Univ., School of Medicine, Scott Hall, 540 E. Canfield Ave., Detroit, MI 48201.

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