Arterial baroreflex control of muscle sympathetic nerve activity in the transition from rest to steady-state dynamic exercise in humans

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Ogoh S, Fisher JP, Raven PB, Fadel PJ. Arterial baroreflex control of muscle sympathetic nerve activity in the transition from rest to steady-state dynamic exercise in humans. Am J Physiol Heart Circ Physiol 293: H2202–H2209, 2007. First published August 3, 2007; doi:10.1152/ajpheart.00708.2007.—We sought to investigate arterial baroreflex (ABR) control of muscle sympathetic nerve activity (MSNA) in the transition from rest to steady-state dynamic exercise. This was accomplished by assessing the relationship between spontaneous variations in diastolic blood pressure (DBP) and MSNA at rest and during the time course of reaching steady-state arm cycling at 50% peak oxygen uptake (V\textsubscript{O\textsubscript{2peak}}). Specifically, DBP-MSNA relations were examined in eight subjects (25 ± 1 yr) at the start of unloaded arm cycling and then during the initial and a later period of arm cycling once the 50% V\textsubscript{O\textsubscript{2peak}} work rate was achieved. Heart rate and arterial blood pressure were progressively increased throughout exercise. Although resting MSNA [16 ± 2 burst/min; 181 ± 36 arbitrary units (au) total activity] was unchanged during unloaded cycling, MSNA burst frequency and total activity were significantly elevated during the initial (27 ± 4 burst/min; 367 ± 76 au; P < 0.05) and later (36 ± 7 burst/min; 444 ± 91 au; P < 0.05) periods of exercise. The relationships between DBP and burst incidence, burst strength, and total MSNA were progressively shifted rightward from unloaded to the initial to the later period of 50% V\textsubscript{O\textsubscript{2peak}} arm cycling without any changes in the slopes of the linear regressions (i.e., ABR sensitivity). Thus a continuous and dynamic resetting of the ABR control of MSNA occurred during the transition from rest to steady-state dynamic exercise. These findings indicate that the ABR control of MSNA was well maintained throughout dynamic exercise in humans, progressively being reset to operate around the exercise-induced elevations in blood pressure and MSNA without any changes in reflex sensitivity.

arterial blood pressure; sympathetic nervous system; arm cycling; exercise onset

IT IS WELL ESTABLISHED THAT in humans, the arterial baroreflex (ABR) control of heart rate and mean arterial pressure (MAP) are reset to operate around the prevailing blood pressure during dynamic exercise without changes in maximal gain (7, 23, 26, 28, 29). In contrast, little information is available regarding baroreflex control of sympathetic nerve activity (SNA) during dynamic exercise in humans, primarily because of the technical difficulties of maintaining quality muscle SNA (MSNA) recordings. To date, only two studies have examined baroreflex regulation of MSNA during dynamic exercise, reporting that carotid baroreflex control of MSNA was preserved during one-legged kicking (15) and moderate-intensity arm cycling (6). An important distinction with these studies as well as the majority of studies examining baroreflex regulation in humans is that baroreflex function was assessed after steady-state exercise conditions were achieved. However, whether ABR characteristics derived during steady-state exercise are representative of the entire exercise period is unclear. Indeed, recent studies have indicated that the functional characteristics of the baroreflex dynamically change throughout a given bout of static exercise (9, 11, 18). Along with a progressive resetting of the MSNA-diastolic blood pressure (DBP) relationship during 3 min of handgrip, Ichinose et al. (11) reported time-dependent increases in ABR-MSNA baroreflex sensitivity. However, whether temporal changes in ABR control of MSNA occur during dynamic exercise is presently unknown.

A fundamental difference between static and dynamic exercise is that during static exercise, blood flow can be occluded even at low exercise intensities (2), whereas during light to moderate dynamic exercise, muscle perfusion progressively increases (30). As such, one would predict less accumulation of metabolites and activation of the metaboreflex during dynamic forms of exercise. Importantly, it has been suggested that the muscle metaboreflex was the primary mediator of the time-dependent changes in the ABR control of MSNA observed during static handgrip (11). Thus, given the known differences in muscle blood flow between static and dynamic exercise and the consequential influences on metaboreflex activation (1), it is difficult to extrapolate findings regarding alterations in ABR-MSNA function observed during static exercise to dynamic exercise.

Therefore, the present study was designed to investigate ABR control of MSNA in the transition from rest to steady-state dynamic exercise. This was accomplished by assessing the relationship between spontaneous variations in DBP and MSNA at rest and during the time course of reaching steady-state arm cycling at 50% peak oxygen uptake (V\textsubscript{O\textsubscript{2peak}}). On the basis of our previous findings indicating preserved carotid baroreflex control of MSNA during steady-state dynamic exercise (6), we hypothesized that the arterial baroreflex control of MSNA would be progressively reset from rest to operate around the prevailing blood pressure throughout the bout of dynamic exercise without any changes in reflex sensitivity.

METHODS

Five men and three women (means ± SE: age of 25 ± 1 yr, height of 176 ± 4 cm, and weight of 75 ± 5 kg) were recruited for voluntary participation.
peroneal nerve of the right leg was instrumented for the continuous monitoring of MSNA. After a 20-min rest period, the arm cycling bout began with an initial period of unloaded cycling for 1 min, and then the workload was increased by 10 or 20 W every minute until the desired work rate was reached. Once the desired work rate was achieved, subjects continued exercise for a minimum of 8 min. In cases in which the MSNA signal was lost during exercise, the protocol was repeated after reacquiring a nerve signal. All eight subjects completed the protocol successfully.

Data Analysis

Cardiovascular and sympathetic variables were examined during the last 4 min of the 20-min rest period, at the onset of unloaded arm cycling, and during the initial and a later period of arm cycling at 50% $V_{\text{O}_2\text{peak}}$. Specifically, unloaded cycling was the first minute of the exercise bout (unloaded EX), the initial period at 50% $V_{\text{O}_2\text{peak}}$ was the first 2 min once the subjects reached the 50% $V_{\text{O}_2\text{peak}}$ work rate (initial 50% EX), and the later period at 50% $V_{\text{O}_2\text{peak}}$ incorporated minutes 6–8 at this work rate (later 50% EX). The average time to reaching the 50% $V_{\text{O}_2\text{peak}}$ work rate was 249 ± 16 s. Analog signals of the ECG, ABP, and mean voltage neurogram were digitized at 200 Hz (DI-720, Dataq Instruments, Akron, OH) and analyzed using LabView software (National Instruments, Austin, Texas, TX). MSNA bursts were identified from the mean voltage neurogram using a customized computer program employing fixed criteria, which accounted for the latency from the R wave of the ECG to the sympathetic burst (8) and incorporated a signal-to-noise ratio of at least 3:1. Computer-identified bursts were subsequently evaluated and confirmed by an experienced investigator. The burst with the largest amplitude during the rest period was allocated a value of 100 (arbitrary units; au), and then all bursts within a trial were normalized with respect to this standard in each subject. MSNA burst frequency (bursts/min) and total activity (i.e., product of burst frequency and mean burst amplitude) were calculated. In addition, as described in detail below, MSNA measurements of burst incidence (bursts/100 heartbeats), burst strength, and total MSNA were used for the assessment of ABR control of MSNA.

ABR control of MSNA was evaluated by analyzing the relationship between spontaneously occurring variations in DBP and MSNA. These analyses were performed on data segments obtained during the last 4 min of the 20-min rest period, during the 1 min of unloaded arm cycling, and during 2 min at the initial and a later period of arm cycling at the 50% $V_{\text{O}_2\text{peak}}$ work rate. Because of the close correlation between changes in MSNA and DBP, DBP was used for this analysis (34). Briefly, the diastolic pressures for each cardiac cycle during an experimental phase (i.e., rest, unloaded EX, initial 50% EX, and later 50% EX) were grouped into 1-mmHg intervals (bins). Importantly, the number of pressure bins used was consistent among the conditions studied, with 17 ± 2 bins at rest and 16 ± 2, 21 ± 1, and 20 ± 1 bins during unloaded EX, initial 50% EX, and later 50% EX, respectively. The burst incidence within each diastolic pressure bin was calculated by determining the percentage of diastoles that were associated with an MSNA burst and expressed as bursts per 100 heartbeats. The burst strength within each diastolic pressure bin was calculated as the area of the average MSNA signal (burst strength/beat), and as such, only cardiac cycles during which a burst occurred were used (i.e., cardiac cycles with “zero” burst area were omitted). The total MSNA was determined for each diastolic pressure bin by calculating the average MSNA associated with each bin (i.e., total burst area of all cardiac cycles within a given diastolic pressure bin divided by the no. of cardiac cycles that occurred within that bin) and expressed as total MSNA per beat. In this way, the averaged MSNA for analyzing total MSNA included heartbeats in which a sympathetic burst did not occur.

The calculated burst incidence, burst strength, and total MSNA obtained within each diastolic pressure bin were plotted against the corresponding DBP and a linear regression applied. The resulting
slopes of this relationship was used to provide a measure of the sensitivity of the ABR control of each variable. The data were weighted for linear regression analysis to account for the number of cardiac cycles within each diastolic pressure bin, thus removing bias due to bins containing very few cardiac cycles (14, 16). DBP bins containing zero MSNA were included in the linear regression analysis to maintain consistency among conditions and to avoid introducing subjectivity to the analyses. Since poor fits could increase the chance of committing a type II error when comparing slopes at rest with those obtained during exercise, a minimum r-value of 0.5 was used as criteria for accepting slopes. For burst incidence, seven of the eight subjects exhibited an r-value of 0.59 and 0.84 at rest, which remained relatively consistent throughout the exercise conditions (0.50–0.74 unloaded EX; 0.52–0.84 initial 50% EX; 0.59–0.89 later 50% EX). The other subject had an average r-value of 0.38 among the conditions. Results were identical for total MSNA, with the same subject exhibiting an r-value of 0.40 among the conditions, whereas all other subjects were >0.5 (0.53–0.84 rest; 0.54–0.71 unloaded EX; 0.53–0.85 initial 50% EX; 0.57–0.90 later 50% EX). Importantly, removal of the subject with an r-value <0.5 did not statistically change the results for burst incidence or total MSNA, and therefore this subject is included in the results. The r-values for burst strength were consistently lower compared with those obtained for the other measures of MSNA. In this regard, four subjects exhibited r-values <0.5 under all conditions. This finding is consistent with previously published data (12, 14, 16) and likely represents the differential control of the occurrence and strength of sympathetic bursts. Indeed, along with baroreflex control, burst strength appears to be particularly influenced by other neural inputs, as described in detail by Kienbaum et al. (16). The operating point for a given regression line was calculated as the mean DBP vs. the mean value of burst incidence, DBP was not significantly changed from rest throughout the arm cycling protocol, demonstrating preserved baroreflex sensitivity (rest −3.67 ± 0.67; initial 50% EX −2.77 ± 0.37 bursts·100 heartbeats−1·mmHg−1; P = 0.307, Fig. 2B). In addition, the averaged operating point was shifted to the right from rest to unloaded cycling, whereas it increased further rightward during the initial and later period of arm cycling with a tendency for upward relocation (Table 1; Fig. 2A). Correlation coefficients describing the relationship between burst incidence and DBP were significant under all conditions for each subject (−0.70 ± 0.06 rest; −0.65 ± 0.03 unloaded EX; −0.65 ± 0.05 initial 50% EX; and −0.68 ± 0.09 later 50% EX).

**Arterial Baroreflex Control of MSNA Burst Incidence**

From rest to the later period of arm cycling at 50% V̇O_{peak}, the linear relationship between burst incidence and DBP was progressively shifted rightward, indicating a progressive resetting of ABR control of MSNA burst incidence (Figs. 1 and 2A). The slope of the linear regression line between burst incidence and DBP was not significantly changed from rest throughout the arm cycling protocol, demonstrating preserved baroreflex sensitivity (rest −3.67 ± 0.67; initial 50% EX −2.77 ± 0.37 bursts·100 heartbeats−1·mmHg−1; P = 0.307, Fig. 2B). In addition, the averaged operating point was shifted to the right from rest to unloaded cycling, whereas it increased further rightward during the initial and later period of arm cycling with a tendency for upward relocation (Table 1; Fig. 2A). Correlation coefficients describing the relationship between burst incidence and DBP were significant under all conditions for each subject (−0.70 ± 0.06 rest; −0.65 ± 0.03 unloaded EX; −0.65 ± 0.05 initial 50% EX; and −0.68 ± 0.09 later 50% EX).

**Arterial Baroreflex Control of MSNA Burst Strength**

The relationship between burst strength and DBP was also progressively shifted rightward from rest to the later period of
arm cycling at 50% VO₂peak without any significant changes in
the slope of the linear regression line from rest (rest 0.26 ± 0.13; initial 50% EX 0.26 ± 0.09 units·beat⁻¹·mmHg⁻¹; P = 0.142, Fig. 3). In addition, the averaged operating point of total MSNA was shifted to the right throughout the exercise protocol, and there was a tendency for upward relocation during the initial and later 50% EX, but this did not reach statistical significance. All subjects showed significant negative correlations between total MSNA and DBP at rest and throughout exercise (0.71 ± 0.06 rest; −0.62 ± 0.03 unloaded EX; −0.66 ± 0.05 initial 50% EX; and −0.66 ± 0.09 later 50% EX).

**DISCUSSION**

The present study is the first to examine the ABR control of MSNA throughout a given bout of dynamic exercise in humans. The major new finding is that the ABR control of MSNA, identified as the relationships between DBP and burst incidence, burst strength, and total MSNA, was progressively shifted rightward during the transition from rest to steady-state

**Arterial Baroreflex Control of Total MSNA**

Similar to the relationships between burst incidence and DBP and burst strength and DBP, the linear relationship between total MSNA and DBP was progressively shifted rightward from rest to the later period of arm cycling at 50% VO₂peak (Figs. 4 and 5A). In addition, no changes in baroreflex sensitivity were observed (rest 0.40 ± 0.11; initial 50% EX 0.36 ± 0.08 units·beat⁻¹·mmHg⁻¹; P = 0.493, Fig. 5B). The averaged operating point of total MSNA was shifted to the right throughout the exercise protocol, and there was a tendency for upward relocation during the initial and later 50% EX, but this did not reach statistical significance. All subjects showed significant negative correlations between total MSNA and DBP at rest and throughout exercise (0.71 ± 0.06 rest; −0.62 ± 0.03 unloaded EX; −0.66 ± 0.05 initial 50% EX; and −0.66 ± 0.09 later 50% EX).

![Fig. 1. Linear relationships between muscle sympathetic nerve activity (MSNA) burst incidence and diastolic blood pressure (DBP) from 1 subject at rest (A–C; solid line) and throughout arm cycling (dotted lines): unloaded EX (A; ○), initial 50% EX (B; ▽), and later 50% EX (C; +). EX, exercise.](fig1)

![Fig. 2. A: summary data showing the average operating points (○) with the corresponding mean linear regression lines relating MSNA burst incidence and DBP at rest, unloaded EX, initial 50% EX, and later 50% EX. B: group summary data for the slopes of the linear regression lines between MSNA burst incidence and DBP representing arterial baroreflex (ABR) sensitivity.](fig2)
dynamic arm cycling without any changes in reflex sensitivity (i.e., slope of the linear regression). These findings indicate that the ABR control of MSNA was well maintained throughout dynamic exercise. This appeared to be a result of an immediate and progressive resetting of the baroreflex control of MSNA to operate around the exercise-induced elevations in blood pressure and MSNA. We suggest that a continuous and dynamic resetting of the ABR control of burst exercise (7, 23, 29). Indeed, lack of a functional sympathetic nervous system results in a fall in blood pressure during dynamic exercise (5, 20). However, despite its clear importance for normal blood pressure control, little is known about the regulation of sympathetic outflow via the ABR during dynamic exercise in humans. Initial findings by Fadel et al. (6) using the variable-pressure neck collar indicated that the stimulus-response relationship for carotid baroreflex control of MSNA was reset from rest during steady-state arm exercise without any change in sensitivity. Similar results have been reported during one-legged kicking exercise (15). These studies concluded that that carotid baroreflex control of MSNA was well preserved during dynamic exercise. The results of the present study are in agreement and extend these previous findings to include a resetting of the ABR control of burst

![Graph A](https://example.com/graph1.png)

![Graph B](https://example.com/graph2.png)

![Graph C](https://example.com/graph3.png)

Fig. 3. Linear relationships between MSNA burst strength and DBP from 1 subject at rest (A–C; • and solid line) and throughout arm cycling (dotted lines): unloaded EX (A; ◦), initial 50% EX (B; ▼), and later 50% EX (C; +).

![Graph A](https://example.com/graph1.png)

![Graph B](https://example.com/graph2.png)

![Graph C](https://example.com/graph3.png)

Fig. 4. Linear relationships between total MSNA and DBP from 1 subject at rest (A–C; • and solid line) and throughout arm cycling (dotted lines): unloaded EX (A; ◦), initial 50% EX (B; ▼), and later 50% EX (C; +).
incidence, burst strength, and total MSNA without any changes in reflex sensitivity. In addition, for the first time, we have demonstrated a progressive resetting of the ABR control of MSNA during the transition from rest to the attainment of steady-state exercise conditions. This is an important distinction because previous studies only focused on the later period of dynamic exercise, building stimulus-response curves after the attainment of steady-state conditions. Thus, by taking advantage of several new methodological approaches to examine ABR control of MSNA on a minute-to-minute basis, we were able to identify that the regulation of MSNA via the ABR is dynamically modulated throughout exercise, being progressively reset to operate around the exercise-induced elevations in blood pressure and MSNA.

Similar results indicating a time-dependent resetting of the ABR control of MSNA have been reported during static exercise in humans (11). However, in contrast to our present findings and previous studies (6, 15) during dynamic exercise, it appears that static exercise increases ABR sensitivity in the control of MSNA (11, 13). In these studies, it was suggested that activation of the muscle metaboreflex could account for the resetting of the MSNA-DBP relationships as well as the increases in sensitivity of ABR control of MSNA. The suggested dominance of the muscle metaboreflex in inducing alterations in ABR control of MSNA fits well within the paradigm of static exercise, where mechanical compression restricts muscle blood flow relative to the metabolic requirements of the exercise, causing a graded and robust activation of the muscle metaboreflex (19, 33, 36). Indeed, the delay in sympathetic activation and increases in sensitivity of ABR control of total MSNA until the second minute of handgrip strongly implicate the metaboreflex (19, 33, 36). The lack of changes in ABR sensitivity in the present study generally support a dominant role for the muscle metaboreflex in increasing ABR-MSNA sensitivity in that it would be expected that the metaboreflex would be minimally activated during the dynamic arm cycling protocol employed, and, therefore, no changes in ABR sensitivity were found. However, it is acknowledged that a small static component is likely present during arm cycling (31). The difficulty is discerning to what extent, if any, this would activate the muscle metaboreflex, particularly given the muscle pumping and high muscle blood flows reported during this dynamic form of exercise (38). In general, further studies are needed to better understand the role of the muscle metaboreflex on neural cardiovascular responses during dynamic exercise in humans.

From a functional standpoint, it may be that an increase in ABR-MSNA sensitivity is necessary during static exercise to offset and restrain the tremendous capacity of the muscle metaboreflex to increase MSNA and blood pressure during such a low-flow exercise condition compared with dynamic exercise. This concept is substantiated by the large increases in MSNA observed during static handgrip when ABR activation is prevented by pharmacologically clamping blood pressure at resting values and negating the exercise-induced rise in blood pressure (32). Thus it may be that only during higher intensities of dynamic exercise in which the muscle metaboreflex is activated that ABR-MSNA sensitivity is increased. Although the achievement of such high work rates during dynamic exercise in humans while maintaining an MSNA signal is quite difficult, recent findings by Miki et al. (22) suggest that the intensity of exercise may indeed be an important factor. In contrast to our findings of preserved ABR sensitivity during moderate-intensity dynamic exercise, these investigators reported that the sensitivity of the ABR control of renal SNA is increased during high-intensity (~90% maximal HR) treadmill exercise in rats. While differential control of renal SNA vs. muscle SNA (17) and/or species differences cannot be excluded, these findings generally support the idea that activation of the muscle metaboreflex during higher intensities of dynamic exercise increases ABR-MSNA sensitivity. However, a potential role for central command in increasing ABR-MSNA sensitivity cannot be overlooked, particularly during high-intensity exercise where central command has been shown to directly increase MSNA (37).

Although completely eliminating a role for muscle mechanoreceptors is not possible, we suggest that the progressive rightward shifting of the ABR control of MSNA to operate around the exercise-induced elevation in blood pressure and MSNA found in the present study is likely attributable to central command. This suggestion is based on several key observations supporting a role for central command in the progressive resetting of the ABR-MSNA relationship. First, a series of experiments has indicated a dominate role of central command in resetting the carotid baroreflex control of blood pressure (10, 21, 24), which is primarily induced by sympa-
ithetically mediated changes in vascular conductance (3, 23). Second, a rightward resetting of the ABR-MSNA relationship was also observed during the first minute of static handgrip, a condition in which the metaboreflex is minimally activated (11). Third, removal of central command (and the mechanoreflex) during postexercise ischemia results in a leftward resetting of the ABR-MSNA relations from peak exercise (11). Fourth, recent findings have demonstrated that baroresensory cells in the nucleus tractus solitarius receive inputs from central command as well as skeletal muscle afferents (4). Last, central descending cholinergic inputs appear to converge directly on the rostral ventral lateral medulla, the primary site for sympathoexcitation (25). Clearly, further studies are needed to better understand the individual and interactive roles of central command, muscle mechanoreflex, and muscle metaboreflex on the ABR control of MSNA during dynamic exercise in humans.

In the present study, we observed a rightward resetting of the operating points and linear regression lines between DBP and burst incidence, burst strength and total MSNA during arm cycling; however, no significant upward resetting of the operating points was noted. This occurred despite a greater than doubling of MSNA total activity during the initial and later 50% EX periods (Table 1), which would be expected to increase the ABR-MSNA operating points upward. These results can likely be attributed to the MSNA analyses employed. Since burst strength (i.e., area) was essentially unchanged from rest during arm cycling, the increase in total activity observed was predominantly a function of increases in burst frequency (i.e., bursts/min). However, the analysis techniques employed to evaluate ABR control of MSNA describe how MSNA is modulated per heartbeat and thus do not directly take into account the increases in burst frequency. Overall, these findings suggest that beat-to-beat ABR control of MSNA is maintained despite the doubling of MSNA total activity during arm cycling.

An important point about the spontaneous techniques employed is that they permit the separate consideration of ABR control of MSNA in terms of burst incidence and burst strength as well as total MSNA. Indeed, it has recently been proposed that baroreflex mechanisms regulating the occurrence and strength of sympathetic bursts are not identical, and therefore these two factors should be considered separately in studies examining ABR control of MSNA (14, 16). In this regard, in the present study, we observed a strong relationship between DBP and burst incidence as well as total MSNA both at rest and during exercise, whereas the relationship between DBP and burst strength was weaker at all phases of this study, as evident from the lower correlation coefficients between these two variables. This observation is in agreement with previous studies (12, 14, 16) and suggests a predominant role of the ABR in the regulation of burst incidence, while other neural inputs may have a greater influence on burst strength (16). Nevertheless, our findings clearly demonstrate that the ability of the ABR to regulate MSNA is well maintained throughout dynamic arm cycling. These findings are in agreement with our previous results demonstrating that the percent change in MSNA total activity in response to neck pressure and neck suction from +40 to −80 Torr was not different from rest during steady-state dynamic exercise (6). Thus similar conclusions have been found using completely different methodological and analysis approaches to examine baroreflex control of MSNA. More importantly, by taking advantage of new methodological approaches to examine ABR control of MSNA on a minute-to-minute basis, we were able to identify that the regulation of MSNA via the ABR is dynamically modulated throughout a given bout of dynamic exercise, being progressively reset to operate around the exercise-induced elevations in blood pressure and MSNA.

Potential Limitations

In the present study, we used sympathetic measurements obtained from the inactive leg to describe overall ABR control of sympathetic outflow during arm cycling. Although necessitated by technical limitations, this does not preclude the possibility that ABR control of SNA differs among vascular beds. Also, consideration should be given to the spontaneous methods used to assess ABR control of MSNA. This noninvasive technique only provides an estimate of ABR sensitivity within the range of the normal spontaneously occurring beat-to-beat oscillations of blood pressure. However, even though the range of pressures studied is limited by the naturally occurring fluctuations in blood pressure, the DBP ranges obtained, particularly during exercise, were fairly robust, approximating 10 mmHg above and below the operating point. This range clearly provides significant inputs to the ABR and provides important information with regard to ABR control around normal operating pressures, although the relatively short data segments may also limit the amount of MSNA within a given pressure bin, particularly for higher pressures. Of note, although alternative methods such as the infusion of vasoactive drugs (i.e., modified Oxford technique) or the neck chamber technique may permit an assessment of baroreflex sensitivity over a greater range of DBP, these techniques also have shortcomings (27). In addition, these other techniques would not have allowed us to obtain the frequent measures necessary to begin to understand the dynamic modulation of ABR-MSNA control throughout dynamic exercise. Last, we cannot account for the potential influence of changes in respiration on the spontaneous assessment of ABR-MSNA control.

In conclusion, we have identified that the ABR control of MSNA was progressively shifted rightward during the transition from rest to steady-state dynamic arm cycling without any changes in reflex sensitivity. This appeared to be a result of an immediate and progressive resetting of the baroreflex control of MSNA to operate around the exercise-induced elevations in blood pressure and MSNA. Thus the ABR control of MSNA is well maintained throughout a given bout of dynamic exercise in humans.

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