Central command blunts sensitivity of arterial baroreceptor-heart rate reflex at onset of voluntary static exercise

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Submitted 6 January 2005; accepted in final form 13 August 2005

Matsukawa, Kanji, Hidehiko Komine, Tomoko Nakamoto, and Jun Murata. Central command blunts sensitivity of arterial baroreceptor-heart rate reflex at onset of voluntary static exercise. Am J Physiol Heart Circ Physiol 290: H200–H208, 2006.—We have reported that baroreflex bradycardia by stimulation of the aortic depressor nerve is blunted at the onset of voluntary static exercise in conscious cats. Central command may contribute to the blunted bradycardia, because the most blunted bradycardia occurs immediately before exercise or when a forelimb is extended before force development. However, it remained unknown whether the blunted bradycardia is due to either reduced sensitivity of the baroreflex stimulus-response curve or resetting of the curve toward a higher blood pressure. To determine this, we examined the stimulus-response relationship between systolic (SAP) or mean arterial pressure (MAP) and heart rate (HR) at the onset of and during the later period of static exercise in seven cats (n = 348 trials) by changing arterial pressure with infusion of nitroprusside and phenylephrine or norepinephrine. The slope of the MAP-HR curve decreased at the onset of exercise to 48% of the preexercise value (2.9 ± 0.4 beats min⁻¹ mmHg⁻¹); the slope of the SAP-HR curve decreased to 59%. The threshold blood pressures of the stimulus-response curves, at which HR started to fall due to arterial baroreflex, were not affected. In contrast, the slopes of the stimulus-response curves during the later period of exercise returned near the preexercise levels, whereas the threshold blood pressures elevated 6–8 mmHg. The maximal plateau level of HR was not different before and during static exercise, denying an upward shift of the baroreflex stimulus-response curves. Thus central command is likely to attenuate sensitivity of the cardiac component of arterial baroreflex at the onset of voluntary static exercise without shifting the stimulus-response curve.

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onset of static exercise but not by the muscle mechanoreflex or metaboreflex from the contacting skeletal muscle. However, it was uncertain whether the attenuated baroreflex bradycardia at the onset of static exercise was caused by either a reduction in the slope of the baroreflex stimulus-response curve or a resetting of the curve toward a higher blood pressure.

To address this uncertainty, we attempted to identify the characteristics of the baroreflex stimulus-response curve at the onset of voluntary static exercise performed by conscious cats. Arterial baroreflex function was partly characterized by the threshold blood pressure and the sensitivity of the stimulus-response curve. The threshold blood pressure was estimated as a blood pressure at which the baroreflex started to operate. To specify these parameters, voluntary static exercise was repeatedly conducted when AP was lowered by constant infusion of sodium nitroprusside and raised by phenylephrine or norepinephrine. The exercise period was arbitrarily divided into the initial period (up to ~10 s after the exercise onset) and the later period until the cessation of static exercise. The relationship between systolic or mean AP and HR was constructed before, at the onset of, and during the later period of exercise to identify the threshold blood pressure and the slope of the arterial baroreflex curve.

**METHODS**

The present study was conducted using seven cats, weighing between 2.8 and 4.3 kg, in accordance with the “Guiding Principles for the Care and Use of Animals in the Fields of Physiological Sciences” approved by the Physiological Society of Japan. The experimental protocols were approved by the Committee of Research Facilities for Laboratory Animal Science, Natural Science Center for Basic Research and Development, Hiroshima University.

**Static exercise training.** The cats were operantly conditioned to perform static exercise as previously described in detail (5, 9, 12, 17). They were trained to sit quietly in a transparent plastic box (width 35 × height 40 × depth 50 cm) with a small window (width 5 × height 7 cm), extend a forelimb through the window, and press a bar for 10–40 s while maintaining a sitting posture. As long as the cats pressed the bar, the sound of a buzzer was emitted as an audio feedback. If the animal completed the static exercise, food was given as a reward. The training was conducted over a period of 2–4 mo (5 days/wk).

**Implantation surgery.** After the training procedure was completed, surgery was conducted to implant catheters. After an overnight fast, atropine sulfate (0.1–0.2 mg/kg im) was given as a preanesthetic drug to reduce salivation and bronchial secretion. Anesthesia was introduced by inhalation of a mixture of 4% halothane (Fluothane; Takeda Chemical Industries, Osaka, Japan), N₂O (0.5 l/min), and O₂ (1.0 l/min), and an endotracheal tube was inserted. Subsequently, the cats inhaled the halothane-N₂O-O₂ mixture through the endotracheal tube. Electrocardiogram, HR, rectal temperature, and respiration were continuously monitored. To maintain an appropriate level of anesthesia during surgery, we usually set the concentration of halothane at 1.0–1.5% and increased it to 2.0–2.5% if an increase in HR, respiration, withdrawal of the limb in response to noxious pinch of the paw, and/or a surgical procedure was observed. Rectal temperature was maintained at 36.5–37.5°C with a heating pad. Polyethylene catheters were inserted into the left external jugular vein for administering drugs and into the left carotid artery for measuring AP. The arterial and venous catheters were tunneled subcutaneously and brought to the exterior in the interscapular region. After implantation surgery was finished, antibiotics (benzylpenicillin potassium, 20,000 U/kg im) were injected and the cats were housed in their cages. Antibiotics (benzylpenicillin benzathine, Bicillin tablets, 100,000 units; Banyu pharmaceutical, Tokyo, Japan) were given orally for 5–7 postoperative days.

**Data measurement.** AP was measured through the carotid artery catheter connected to a pressure transducer (DPTIII; Baxter, Tokyo, Japan). Systolic AP (SAP), mean AP (MAP), and diastolic AP (DAP) were calculated every pulse. HR was derived from arterial pressure pulse using a tachometer (model 1321; GE Marquette Medical Systems, Tokyo, Japan). The actual force that the cats applied to the bar was measured with strain gauges (KFG-2N-120; Kyowa Electronic Instruments, Tokyo, Japan) affixed on the bar. The onset and offset of static exercise were defined from the force development. Timing at the start of forelimb movement was manually marked with an electric switch. AP, HR, force, and the timing signal for the start of forelimb movement were simultaneously recorded on an eight-channel pen-writing recorder (8M14; GE Marquette Medical Systems) and stored in a computer via an analog-to-digital converter (MP100; BIOPACK Systems, Santa Barbara, CA) at a sampling frequency of 400–500 Hz.

**Experimental protocols and data analysis.** When the cats were in good condition and able to perform voluntary static exercise, the experiments were conducted. On an experimental day, each cat was put into the transparent plastic box. A period of ≥30 min was allowed to establish that the animal was quiescent and the cardiovascular variables became stable. When sitting quietly, the cat voluntarily extended the forelimb through the window and pressed the bar while maintaining the sitting posture. HR, AP, and force applied to the bar were measured during static exercise. A typical example of the data during voluntary static exercise is shown in Fig. 1. The increase in HR at the onset of exercise lasted for ~10 s, and then HR returned to the preexercise level. The initial cardiovascular responses were predominantly induced by central command (12, 17). The exercise period was arbitrarily divided into the initial period (up to 10 s from the exercise onset) and the later period until the cessation of static exercise.

We examined the effects of voluntary static exercise on the characteristics of the arterial baroreceptor-HR reflex (slope, threshold...
RESULTS

Changes in HR and AP during static exercise in the presence of nitroprusside. An example of the changes in HR, AP, and force during static exercise is shown in Fig. 1. HR began to increase immediately before a bar was pressed and reached the peak value at 5 s from the onset of exercise. Thereafter, HR returned to the preexercise level within ~11 s after the exercise onset, although static exercise was not ended. A slight decrease in AP was observed at the onset of exercise, suggesting peripheral vasodilatation in concert with increased cardiac output. The initial depressor response was followed by an increase in AP, which persisted throughout static exercise. After exercise, HR and AP increased in association with eating behavior.

The animals performed voluntary static exercise for 20 ± 0.5 s. The peak force applied to the bar during static exercise was 0.41 ± 0.02 kg. The cardiovascular variables before, at the onset of, and during the later period of exercise are summarized in Table 1. HR increased by 28 ± 3 beats/min at 2 ± 0.1 s from the onset of exercise but returned near the control during the later period of exercise. SAP rose slightly by 8 ± 2 mmHg at the onset of exercise and further increased by 18 ± 5 mmHg during the later period of exercise (at 18 ± 0.4 s from the exercise onset) as long as static exercise was sustained. MAP and DAP had the same tendency as SAP.

Figure 2 exemplifies the changes in the peak force and the values of HR and MAP taken before, at the onset of, and during the later period of exercise in a series of exercise trials performed by a conscious cat. Sodium nitroprusside was infused in the middle of the experiment. Before infusion of nitroprusside, the trace of the HR values taken at the onset of static exercise is shown above the HR traces before and during the later period of exercise. With respect to MAP, the traces obtained at the onset of and during the later period of static exercise are found above the MAP traces before exercise. After MAP was decreased by nitroprusside, there is no clear difference in the traces of HR among the three periods; also, there is no significant difference in the traces of MAP among the three periods. The same tendency was observed for SAP. The peak force was well maintained throughout the experiment, irrespective of infusion of nitroprusside. The overall data were utilized to construct the baroreflex relationship between MAP or SAP and HR before, at the onset of, and during the later period of voluntary static exercise in individual cats. Baroreflex relationship before, at onset of, and during later period of static exercise. Figure 3 compares the baroreflex stimulus-response curves between MAP and HR obtained before, at the onset of, and during the later period of voluntary static exercise in two cats. The slope of the stimulus-response curve between MAP and HR reduced at the onset of static exercise, as shown in Fig. 3, except in one cat. However, it seemed that the threshold blood pressure (MAPth) of the stimulus-response curve at the onset of exercise did not change from that of the stimulus-response curve before exercise. In contrast, during the later period of static exercise, the slope of

Table 1. Baseline HR, SAP, MAP, and DAP and their responses to static exercise

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<th>Baseline</th>
<th>At Onset of Exercise</th>
<th>During Later Period of Exercise</th>
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<tbody>
<tr>
<td>HR, beats/min</td>
<td>155 ± 14</td>
<td>183 ± 15*</td>
<td>163 ± 14*</td>
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<tr>
<td>SAP, mmHg</td>
<td>116 ± 5</td>
<td>122 ± 5</td>
<td>133 ± 8*</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>95 ± 3</td>
<td>103 ± 3</td>
<td>110 ± 5*</td>
</tr>
<tr>
<td>DAP, mmHg</td>
<td>75 ± 3</td>
<td>84 ± 2*</td>
<td>89 ± 5*</td>
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HR, heart rate; SAP, systolic arterial blood pressure; MAP, mean arterial blood pressure; DAP, diastolic arterial blood pressure. *P < 0.05, significant difference from baseline value.
the SAP-HR curve was also decreased to 1.6 beats/min/mmHg, and the MAP-HR curve was decreased by constant intravenous infusion of sodium nitroprusside in the middle of the experiment. The peak force was well maintained throughout the experiment, irrespective of infusion of nitroprusside. Before nitroprusside is infused, the trace of the HR values obtained at the onset of static exercise is found above the HR traces before and during the later period of exercise. When MAP is decreased by nitroprusside, there is no clear difference in the traces of HR among the 3 periods (before, at the onset of, and during the later period of exercise).

The average slopes and the threshold blood pressures (MAPth and SAPth) of the SAP-HR and MAP-HR stimulus-response curves are compared among the three periods (before, at the onset of, and during the later period of exercise) in Fig. 4. The slope of the MAP-HR curve was significantly blunted to 1.4 ± 0.3 beats·min⁻¹·mmHg⁻¹ at the onset of exercise from the control of 2.9 ± 0.4 beats·min⁻¹·mmHg⁻¹; the slope of the SAP-HR curve was also decreased to 1.6 ± 0.3 beats·min⁻¹·mmHg⁻¹ at the onset of exercise from the pre-exercise control of 2.7 ± 0.3 beats·min⁻¹·mmHg⁻¹. However, those slopes during the later period of static exercise did not change significantly from the control value before exercise, although they tended to be less. On the other hand, the threshold blood pressures of SAPth and MAPth were not affected at the onset of static exercise, but they significantly increased by 6 ± 1 and 8 ± 2 mmHg, respectively, during the later period of static exercise. The maximal plateau level of HR was not different among the three periods (Fig. 5), suggesting that an upward shift of the baroreflex stimulus-response curve is not appreciable during voluntary static exercise in conscious cats.

Changes in HR and AP during static exercise in the presence of phenylephrine or norepinephrine. To examine whether the slope of the baroreflex curve was also blunted at the onset of static exercise in a higher blood pressure range above the operating point, we repeatedly evoked static exercise in the presence of phenylephrine or norepinephrine as depicted in Fig. 6. Despite a profound decrease of the preexercise HR in the presence of norepinephrine, HR was capable of increasing to the peak value of nearly 200 beats/min at the onset of static exercise (Fig. 6C), which corresponded to the initial HR peak in the absence of norepinephrine (Fig. 6B). In contrast, when the pre-exercise HR was largely augmented in the presence of nitroprusside, the initial increase in HR was slight (Fig. 6A). In combination with the pressor and depressor data, the stimulus-response relationship between SAP or MAP and HR was reconstructed in the entire blood pressure range in Fig. 7. It is evident that the slope of the baroreflex curve was also blunted at the onset of static exercise in a higher blood pressure range above the operating point. On the other hand, the slope during the later period of static exercise did not change significantly from the pre-exercise control. Indeed, the average slope of the MAP-HR curve in the higher blood pressure range was significantly decreased to 0.50 ± 0.18 beats·min⁻¹·mmHg⁻¹ at the onset of exercise from the pre-exercise control of 0.83 ± 0.14 beats·min⁻¹·mmHg⁻¹; the slope of the SAP-HR curve was also blunted to 0.37 ± 0.11 beats·min⁻¹·mmHg⁻¹ at the onset of exercise from the control of 0.69 ± 0.14 beats·min⁻¹·mmHg⁻¹. These changes in the slope of the stimulus-response curves in the higher blood pressure range almost matched those changes in the low blood pressure range.

**DISCUSSION**

We recently found that the baroreflex bradycardia due to stimulation of the ADN was blunted at the beginning of voluntary static exercise in conscious cats (12). Fundamentally, the same effect on the ADN-stimulation induced bradycardia was observed at the onset of spontaneous contraction in unanesthetized decerebrate cats, whereas such an attenuating effect on the baroreflex bradycardia was not evident during electrically evoked contraction or passive stretch of skeletal muscle (26). Taking these findings together, it is likely that the central characteristics of the arterial baroreflex are dynamically modulated from moment to moment during voluntary static exercise. In particular, central command is considered to inhibit the cardiac component of the arterial baroreflex at the onset of static exercise. The blunted bradycardia can be explained by two different possibilities: one is that the stimulus-response curve is reset toward an appropriate direction without changing the average and/or maximal slope of the baroreflex curve; the other is that the slope of the stimulus-response curve is reduced without shifting the baroreflex curve. We attempted to clarify which possibility was responsible for the blunted baroreflex bradycardia. The major finding of this study is that...
the slope of the baroreflex stimulus-response curve decreased at the onset of voluntary static exercise in conscious cats, whereas the threshold blood pressure of the curve at which the arterial baroreflex started to operate was not affected. On the other hand, during the later period of static exercise, the slope of the baroreflex curve recovered near the preexercise level, whereas the threshold blood pressure increased to a higher level, suggesting a rightward shift of the baroreflex stimulus-response curve. On the basis of these findings, it is likely that central command descending from the higher brain centers attenuates the sensitivity of the cardiac component of the arterial baroreflex at the onset of voluntary static exercise without shifting the stimulus-response curve.

Limitations. Several limitations are involved in this study. First, the maximal voluntary force that the cats could produce during static exercise was uncertain. The peak force produced during static exercise in conscious cats was 0.41 ± 0.02 kg, which corresponded to ~12% of their body weight. Because the maximally developed force was nearly 1.0 kg in the present and previous studies (5, 9, 12, 17), the cats seemed to produce ~40% of the maximal voluntary force at the most, and the intensity of static exercise in the present study appeared mild or moderate. The exercise intensity might be insufficient to initiate the muscle mechanoreflex and metaboreflex, whose contribution on the possible modulation of arterial baroreflex at the onset of voluntary static exercise remains to be studied with a higher intensity of static exercise. Second, the present findings do not always lead to a definite conclusion about an influence of central command on the vasomotor component of arterial baroreflex, because previous studies, including ours, have shown the differential effects of exercise on the cardiac and vasomotor components of arterial baroreflex function (12, 26, 33). Ludbrook (13) also questioned whether the baroreflex gain for HR gave useful information about blood pressure control in humans or experimental hypertension. Querry et al. (33) reported that central command, which was a primary mechanism for resetting of the carotid baroreflex curve during a steady state of static and dynamic exercise in humans, appeared to modulate the carotid baroreceptor-HR reflex, rather than the carotid baroreceptor-MAP reflex. When we recently evaluated the effect of static exercise on the cardiac and vasomotor components of arterial baroreflex function using stimulation of the ADN given at various times during exercise in conscious cats, we found that the ADN stimulation-induced bradycardia was remarkably attenuated at the onset of voluntary static exercise, whereas static exercise did not affect the ADN stimulation-induced depressor response (12). The same effects on the aortic baroreflex function were observed at the onset of spontaneous muscle contraction in unanesthetized decerebrate cats (26). Taking these findings together, it is likely that the cardiac component of the arterial baroreflex is modulated by central command at the onset of and during steady state of exercise, whereas its vasomotor component tends to be preserved. In other words, the present conclusion that central command blunts the sensitivity of the arterial baroreceptors-HR reflex at the onset of voluntary static exercise cannot be extrapolated to the stimulus-response curve of the vasomotor component of the arterial baroreflex. The third problem is that because nitroprusside is known as a donor of nitric oxide (NO), released NO may alter the viscoelastic property of the

Fig. 3. Original relationships between MAP and HR obtained before, at the onset of, and during the later period of voluntary static exercise are plotted on the x-y plane for 2 cats (A and B, respectively). After the paired MAP and HR data were sorted according to the size of MAP and divided into groups every 5 mmHg of MAP, the relationship between the average values of MAP and HR in the groups was plotted to obtain the baroreflex stimulus-response curve for each cat (C and D). Regarding characteristics of the baroreflex stimulus-response curve, we examined the maximal plateau level of HR, the threshold blood pressure (MAPth) at which HR started to fall as AP increased, and the slope of the linear regression line as the sensitivity of the baroreflex relationship.
vascular wall in the carotid sinuses and aortic arch and suppress activity of arterial baroreceptors by an action on voltage-dependent ion channels (3). Furthermore, released NO may not only alter cerebral blood flow due to relaxation of smooth muscles of cerebral vessels but also penetrate the vascular wall into the brain, which may cause a central influence on arterial baroreflex function. To avoid such pharmacological complications, transient mechanical perturbation of arterial baroreceptors, such as increasing carotid sinus pressure in an isolated carotid sinus preparation or increasing aortic blood pressure by inflating a balloon placed in the aorta, will be needed. Even though we cannot rule out the possibility that nitroprusside may modify activity of peripheral arterial baroreceptors and central neurons in the brainstem baroreflex circuit, the peripheral and central effects on arterial baroreflex function are expected to be uniform independently of static exercise. Therefore, it is difficult for the released NO to explain the dynamic influences of static exercise on the characteristics of the baroreflex stimulus-response curve.

Modulation of sensitivity of arterial baroreflex function. The reduction in the baroreflex sensitivity at the onset of voluntary static exercise in this study (41% for the SAP-HR stimulus-response curve and 52% for the MAP-HR curve) is in good agreement with the reduced bradycardia due to ADN stimulation by 38% at the onset of the identical static exercise in conscious cats (12) and by 45% at the onset of spontaneous contraction in unanesthetized decerebrate cats (26). These findings imply that the sensitivity of the arterial baroreceptors-HR reflex is temporarily inhibited at the onset of static exercise, which in turn contributes to the rapid cardiac acceleration. This concept is supported by the previous studies using humans, demonstrating that the baroreflex response of R-R interval to a step increase in carotid sinus transmural pressure is blunted at the beginning of isometric handgrip exercise, even in the anticipation period preceding the start of exercise (6, 15). Another important point is that because the baroreflex bradycardia in response to electrical stimulation of ADN reflects the central characteristics of aortic baroreflex function, central modulation of the brain stem neural circuit along the arterial baroreflex pathway is involved in temporal suppression of the sensitivity of the arterial baroreceptor-HR reflex.

The prompt attenuation in the sensitivity of the arterial baroreceptor-HR reflex at the onset of exercise is in favor of

Fig. 4. Slopes of the regression lines and threshold systolic AP (SAPth) and MAP (MAPth) in the SAP-HR and MAP-HR relationships are compared among the 3 periods (before, at the onset of, and during the later period of exercise). The slopes of the baroreflex relationships were significantly decreased at the onset of exercise (A and B). However, those slopes returned near the preexercise control levels during the later period of exercise, whereas the threshold pressures SAPth and MAPth significantly increased to a higher level by 6–8 mmHg (C and D).

Fig. 5. Maximal plateau level of HR in the baroreflex relationship was not different among the 3 periods (before, at the onset of, and during the later period of exercise). It is suggested that an upward shift of the baroreflex curve between SAP or MAP and HR is not appreciable.
either central command or the muscle mechanoreflex as a candidate mechanism (7, 19, 25, 37). McWilliam and Yang (22) found that prolongation of R-R interval in response to an increase in carotid sinus pressure was reduced as soon as skeletal muscle contraction was elicited by ventral root stimulation, suggesting involvement of the muscle mechanoreflex. However, McIlveen et al. (20) reported that neither tendon stretch nor electrically evoked contraction of skeletal muscle suppressed the sensitivity of the carotid sinus stimulus-response curve but that they shifted the baroreflex curve upward in the decerebrate cat. Furthermore, Murata et al. (26) reported that neither muscle intervention affected the baroreflex bradycardia due to ADN stimulation in the decerebrate cat. Thus these recent studies suggest that the muscle mechanoreflex may not play a predominant role in causing the attenuated sensitivity of the arterial baroreceptor-HR reflex at the onset of exercise. Instead, central descending output from higher brain centers may act on the arterial baroreflex circuit within the brain stem and modulate its central property at the start of exercise, because the attenuated baroreflex bradycardia due to ADN stimulation was observed immediately before voluntary exercise in conscious cats, i.e., in the absence of muscular exertion (12). Regarding a neural mechanism responsible for suppression by central command of the arterial baroreflex, it is known that electrical or chemical stimulation of a localized area in the hypothalamus or the midbrain periaqueductal gray, which is capable of inducing the defense reaction, suppresses reflex bradycardia evoked by stimulation of the aortic depressor nerve or carotid sinus nerve in anesthetized rats and cats (4, 10, 28). Indeed, stimulation of the higher brain centers imposes an inhibitory action on neurons in the nucleus of the solitary tract and the vagal preganglionic motor nuclei, which constitutes the arterial baroreflex arc (11, 24, 28).

Modulation of threshold pressure of arterial baroreflex function. Previous studies (29, 30) suggested that central command might contribute to resetting the carotid sinus baroreflex curve to a higher sinus pressure during exercise without changing the sensitivity of the baroreflex curve. However, the previous conclusion is not always applied to the initial moment of exercise, because the data were taken during a steady state of exercise with augmented central command. In this study, neither threshold blood pressure (SAPth or MAPth) of the baroreflex stimulus-response curves was affected at the onset of voluntary static exercise, suggesting that resetting of the baroreflex curve to a higher blood pressure is not appreciable at that moment of static exercise in conscious cats. On the other hand, during the later period of voluntary static exercise, the baroreflex stimulus-response curve shifted rightward to a higher blood pressure, restoring the baroreflex sensitivity (Figs. 3 and 4). This finding is in agreement with the previous results demonstrating that the stimulus-response curve of the carotid sinus baroreflex is reset toward a higher level of carotid sinus pressure during a steady-state period of exercise without changing the baroreflex sensitivity in humans (8, 14, 29, 30, 32, 34) and dogs (23). The present finding also corresponds with our previous finding that the ADN stimulation-induced bradycardia is not significantly blunted during the later period of the identical static exercise (12). Thus the sensitivity of the baroreflex stimulus-response curve is temporarily suppressed at the onset of voluntary exercise, after which the baroreflex

![Fig. 6. Changes in HR and AP during voluntary static exercise in the absence of any drug (control; B) and in the presence of nitroprusside (A) or norepinephrine (C) in the same cat. Nitroprusside was infused at a rate of 4 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \). Norepinephrine was infused at a rate of 0.7 \( \mu \text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} \).](image)

![Fig. 7. Original relationships between MAP and HR obtained before, at the onset of, and during the later period of voluntary static exercise are plotted in a cat. Voluntary static exercise was repeatedly conducted when MAP was lowered by constant infusion of sodium nitroprusside and raised by phenylephrine or norepinephrine. In combination with the pressor and depressor data, the stimulus-response relationship between SAP or MAP and HR was reconstructed in the entire blood pressure range.](image)
curve is shifted toward a higher level of AP with restoring the baroreflex sensitivity. On the contrary, the maximal plateau level of HR in the baroreflex stimulus-response curve was not different before, at the onset of, and during the later period of voluntary static exercise (Fig. 5), suggesting that an upward shift of the baroreflex curve is not appreciable in this study. Differential responses in HR and renal sympathetic outflow at onset of static exercise. We have reported that cardiac and renal sympathetic efferent discharges measured in conscious cats increase abruptly in association with voluntary static exercise, treadmill exercise, and natural body movement such as postural changes, walking, and grooming (16–18, 27, 38). It is therefore considered that the initial tachycardia at the start of static exercise is elicited by stimulation of cardiac sympathetic outflow in concert with cardiac parasympathetic withdrawal. Interestingly, when AP is highly elevated with norepinephrine, baseline renal sympathetic discharge is almost completely inhibited and the centrally induced increase in renal sympathetic outflow is not evoked during static exercise (17). Thus the sensitivity of the arterial baroreceptor-renal sympathetic nerve activity reflex may not be blunted at the onset of static exercise, and the centrally induced increase in renal sympathetic outflow is not able to overcome the baroreflex inhibition. On the contrary, although baseline HR is reduced by arterial baroreflex due to a rise in AP, the centrally induced tachycardia at the beginning of voluntary static exercise is preserved, as mentioned previously. In agreement with these observations, neither voluntary static exercise in conscious cats nor spontaneous contraction in decerebrate cats affected the baroreflex-evoked depressor response due to ADN stimulation, despite the blunted baroreflex bradycardia (12, 26). We therefore propose that central command may have differential influence on the cardiac and vasomotor components of arterial baroreflex and that it may selectively inhibit the cardiac component of arterial baroreflex at the onset of exercise but preserve the vasomotor component.

In conclusion, the sensitivity of the cardiac component of the arterial baroreflex was temporarily blunted at the onset of voluntary static exercise in conscious cats, after which the baroreflex stimulus-response curve was shifted toward a higher blood pressure level, restoring the baroreflex sensitivity. We conclude that central command inhibits the baroreflex sensitivity of the arterial baroreceptor-heart rate reflex at the onset of static exercise, which in turn produces rapid cardiac acceleration.

ACKNOWLEDGMENTS
We greatly thank Tomoko Ishida for her technical assistance.

GRANTS
This study was supported by Grants-in-Aid for Scientific Research from the Ministry of Education, Culture, Sports, Science and Technology, Japan.

REFERENCES


