Influence of Cardiopulmonary Vagal Afferent Activity on Carotid Chemoreceptor and Baroreceptor Reflexes in the Dog

By Hiroyuki Koike, Allyn L. Mark, Donald D. Heistad, and Phillip G. Schmid

ABSTRACT

The goal of this study was to determine if physiological levels of cardiopulmonary vagal afferent activity modulate carotid chemoreceptor and baroreceptor reflexes. In anesthetized, ventilated dogs, the aortic nerves and the cervical sympathetic trunks were cut, and atropine was administered so that vagotomy would interrupt only cardiopulmonary afferent impulses. Reflex vascular responses were observed in perfused gracilis muscle and hindpaw. Carotid chemoreceptors were activated with nicotine or hypoxic, hypercapnic blood; carotid baroreceptors were stimulated by changes in carotid pressure. Interruption of vagal afferents augmented reflex vascular responses during changes in carotid pressure from 75 to 125 mm Hg. Interruption of cardiopulmonary vagal afferents potentiated reflex vasoconstrictor (muscle), vasodilator (paw), and vasopressor responses to activation of the carotid chemoreceptors. The potentiation of the chemoreceptor reflex frequently occurred in the absence of increases in base-line vascular resistance. Vagotomy also potentiated ventilatory responses to stimulation of the carotid chemoreceptors in spontaneously breathing dogs. The results indicate that interruption of cardiopulmonary afferents potentiates the vascular and ventilatory responses to activation of the carotid chemoreceptors and augments the gain of the carotid baroreceptor reflex at low carotid pressures. These findings suggest that physiological levels of cardiopulmonary vagal afferent impulses suppress carotid baroreceptor and chemoreceptor reflexes through an interaction in the central nervous system. The suppressive effect on the chemoreceptor reflex may be distinct from tonic restraint of the vasomotor center by vagal afferents, since it involves sympathetic vasodilator as well as vasoconstrictor responses and may occur without suppression of base-line adrenergic constrictor tone.

Vagal afferent pathways originating in cardiopulmonary receptors have been recognized for many years (1-4), and investigators (5-9) have recently demonstrated that impulses arising in these receptors exert a tonic restraint on adrenergic discharge and contribute to physiological control of the circulation.

In delineating the role of cardiopulmonary vagal afferents in circulatory control, it is important to consider an influence of vagal afferent impulses on other reflexes, such as the carotid chemoreceptor and baroreceptor reflexes. The observation of Heistad et al. (10) and of Mancia (11) that afferent impulses arising in carotid baroreceptors inhibit the chemoreceptor reflex through a central interaction prompted us to test the hypothesis that afferent impulses originating in cardiopulmonary receptors inhibit the chemoreceptor reflex. Toubes and Brody (12) have reported that activation of vagal afferents during myocardial ischemia does not alter the chemoreceptor reflex, but inhibition of the chemoreceptor reflex by vagal afferents might have been masked in their study by hypotension, which is reported to potentiate the chemoreceptor reflex (10).

Previous investigators (5, 6) have reported that interruption of vagal afferent activity potentiates the response to carotid occlusion. Although these studies suggest that vagal afferent activity inhibits the carotid baroreceptor reflex, quantitative conclusions regarding interaction of cardiopulmonary and carotid baroreceptor reflexes are difficult without knowledge of the magnitude of change in carotid sinus pressure. In the previous studies (5, 6), carotid sinus pressures were not controlled or measured. Since base-line systemic arterial blood pressure increased after vagotomy, the magnitude of fall in carotid arterial blood pressure during coronary carotid occlusion, i.e., the stimulus to the carotid baroreceptor reflex, was probably greater after vagotomy; this situation may have been responsible for some of the potentiation.

The purpose of the present study was, thus, to determine the influence of physiological levels of cardiopulmonary vagal afferent activity (1) on

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vascular responses to graded stimulation of carotid baroreceptors under conditions in which base-line carotid sinus pressure and changes in carotid pressure are controlled and (2) on vascular and ventilatory responses to pharmacologic and physiological activation of carotid chemoreceptors. A goal of this study was to determine if physiological levels of vagal afferent activity exert a suppressive effect on the carotid chemoreceptor reflex that is distinct from restraint of tonic adrenergic discharge by cardiopulmonary vagal afferents.

**Methods**

Mongrel dogs weighing 17-25 kg were anesthetized with chloralose (50 mg/kg, iv) and urethane (500 mg/kg, iv) and treated with decamethonium bromide (0.3 mg/kg, iv) and heparin (500 units/kg, iv). The dogs were ventilated artificially (tidal volume 15 ml/kg and rate 12-14/min) through an endotracheal tube with room air and supplemental oxygen at 1-2 liters/min to maintain normal blood gases. Blood gases and pH were measured with an Instrumentation Laboratory blood-gas analyzer before and after vagotomy. Systemic arterial and right atrial pressures were measured. Atropine (0.2 mg/kg, iv) was administered each hour to block vagal cholinergic effects on the heart.

**SECTION OF AORTIC DEPRESSOR NERVES**

Through a longitudinal midline incision in the neck, the nodose ganglion was exposed bilaterally. Using a dissecting microscope, the aortic nerves were located near the junction of the cranial laryngeal nerve and the nodose ganglion (13). The aortic nerves were identified by electrically stimulating the central cut end of the nerves. High-voltage, low-frequency (10 v, 10 Hz, 0.1-1 msec) stimulation produced hypertension; low-voltage, high-frequency (4 v, 100 Hz, 0.1-1 msec) stimulation produced hypotension (13).

The efficacy of interrupting the aortic nerves was demonstrated by injecting sodium cyanide (0.2-0.4 mg/kg) into the aortic root while the carotid sinuses were being perfused with a delay circuit. When the aortic nerves were intact, an aortic injection of sodium cyanide promptly increased systemic arterial blood pressure and muscle perfusion pressure (13). After the aortic depressor nerves had been sectioned, an aortic injection of sodium cyanide failed to increase systemic arterial blood pressure and muscle perfusion pressure (13). After the aortic depressor nerves had been sectioned, an aortic injection of sodium cyanide failed to increase systemic arterial blood pressure and muscle perfusion pressure (13). Although these procedures directly indicate only denervation of aortic chemoreceptors, Edis and Shepherd (13) have demonstrated that they also confirm denervation of aortic baroreceptors. Cervical sympathetic nerves were identified and cut just below the superior cervical ganglia bilaterally. Thus, the aortic depressor nerves and the cervical sympathetic nerves were eliminated from the vagosympathetic trunk, and vagal cholinergic effects were blocked with atropine (0.2 mg/kg, iv). Preservation of intact vagal cardiopulmonary afferents after dissection was demonstrated by observing vasodepressor responses to right atrial injections of veratrine (2-4 µg/kg) (13).

**CAROTID CHEMORECEPTOR AND BARORECEPTOR REFLEXES**

After the internal carotid and all branches of the external carotid arteries had been ligated bilaterally, the common carotid arteries were cannulated and perfused separately at constant flow (60-90 ml/min) with blood from the femoral artery. The external carotid arteries were cannulated, and blood from the external carotid arteries flowed through Starling resistors into the jugular veins. The occipital arteries were ligated 1-2 cm from their origin from the carotid arteries to preserve the blood supply to carotid chemoreceptors. Carotid perfusion pressures could thus be regulated with the Starling resistors. Stimulus-response curves for carotid baroreceptors were obtained by producing graded changes in carotid sinus pressure. The sequence of changes in carotid pressure was 75 to 125 to 175 to 75 mm Hg. Carotid sinus pressures were maintained at each level for 90 seconds, and reflex changes in muscle perfusion pressure and systemic arterial blood pressure were observed. Carotid sinus pressure was maintained at 125 mm Hg during the remainder of the study. The gain of the reflex (Δoutput/Δinput) was calculated as Δmuscle perfusion pressure/Δcarotid perfusion pressure and also as Δsystemic arterial blood pressure/Δcarotid perfusion pressure. The gain of the reflex when carotid sinus pressure was increased from 75 to 175 mm Hg was similar to that when pressure was decreased from 175 to 75 mm Hg. The data which are presented are responses to increases in carotid sinus pressure from 75 to 125 to 175 mm Hg.

Carotid chemoreceptors were stimulated by injecting nicotine (0.3-1.2 µg/kg) into the tubing perfusing the carotid arteries upstream from the pump. The low dose of nicotine (nicotine1) was 0.3 or 0.6 µg/kg; the high dose (nicotine2) was twice the low dose. The same two doses were administered before and after vagotomy. Previous studies (14) in our laboratory have demonstrated that vasoconstrictor responses in muscle and vasodilator responses in the paw during intracarotid injections of nicotine are abolished by denervating the carotid bodies. In three separate experiments, carotid chemoreceptors were also activated by perfusing the carotid bifurcations intermittently with hypoxic, hypercapnic blood for 90-120 seconds while systemic normoxia and normocapnia were maintained. Blood from a femoral artery was passed through a regional perfusion bubble oxygenator, rewarmed, and pumped through the carotid bifurcations. A gas mixture of 5% CO2-95% N2 and supplemental CO2 was bubbled through the oxygenator to produce hypoxic, hypercapnic blood; the same mixture was used before and after vagotomy.

**GRACILIS MUSCLE AND HINDPAW**

An innervated gracilis muscle was perfused at constant flow to evaluate reflex vascular responses to stimulation of carotid baroreceptor and chemoreceptor reflexes. The muscle was dissected free from surrounding tissues except for the gracilis artery, vein, and nerve. The gracilis artery was cannulated and perfused at constant flow (8-12 ml/min) with blood from a femoral artery. With flow constant, changes in perfusion pressure indicate changes in gracilis vascular resistance. In three experiments on the chemoreceptor reflex, the cranial tibial artery to the hindpaw was exposed near the tarsus,
VENTILATORY RESPONSES

In three other experiments, ventilatory responses to stimulation of carotid chemoreceptors with nicotine were obtained before and after blockade of cardiopulmonary afferents. In these experiments, decamethonium was not administered, and the dogs breathed spontaneously through an endotracheal tube. Air flow was measured with a Fleisch pneumotachograph and electronically integrated to measure minute volume and tidal volume. End-tidal CO₂ was measured with a Beckman LB 2 gas analyzer.

Statistical analysis was performed with the t-test for paired data.

Results

CAROTID CHEMORECEPTOR REFLEX

Effects of Cardiopulmonary Vagal Afferent Activity on Vascular Responses.

—Stimulation of the carotid chemoreceptors with nicotine increased systemic arterial blood pressure and vascular resistance in muscle and decreased vascular resistance in the paw (Fig. 1, Table 1). Vagotomy produced a significant potentiation of the reflex responses to stimulation of the carotid chemoreceptors (Fig. 1, Table 1).

Vagotomy increased baseline muscle perfusion pressure or vascular resistance in some experiments (Table 1), but the increases were not consistent. Striking potentiation of the responses to chemoreceptor stimulation often occurred in the baseline values.

Figure 1

Effects of interruption of cardiopulmonary vagal afferents on baseline vascular resistance (left) and reflex vasodilator responses (right) in the paw to activation of carotid chemoreceptors with nicotine, i.e., intracarotid administration. 

BASELINE VALUES

Fig. 1

CHEMORECEPTOR RESPONSES

After

Before

Results

TABLE 1

Effects of Vagotomy on Responses to Carotid Chemoreceptor Stimulation with Nicotine

<table>
<thead>
<tr>
<th></th>
<th>Muscle perfusion pressure (mm Hg)</th>
<th>Mean arterial blood pressure (mm Hg)</th>
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<tbody>
<tr>
<td></td>
<td>Control, ΔNicotine¹</td>
<td>Control, ΔNicotine²</td>
</tr>
<tr>
<td>Before vagotomy</td>
<td>158 ± 6.3, 23 ± 2.2</td>
<td>156 ± 5.8, 29 ± 2.6</td>
</tr>
<tr>
<td>After vagotomy</td>
<td>168 ± 5.9, 45 ± 5.7</td>
<td>171 ± 6.1, 56 ± 7.5</td>
</tr>
<tr>
<td>P</td>
<td>&gt; 0.05</td>
<td>&lt; 0.05</td>
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All values are means ± s.e for experiments on 14 dogs except for those for the responses to local intra-arterial injection of norepinephrine (NE) where n = 9. ΔNicotine¹ and ΔNicotine² indicate the responses to intracarotid injections of low and high doses, respectively. The low dose was 0.3 or 0.6 μg/kg, and the high dose was twice the low dose. The same doses were administered before and after vagotomy. ΔNE indicates the response to an injection of 0.5 μg/kg norepinephrine into the tubing perfusing the gracilis muscle. Systemic arterial Po₂ averaged 106 ± 13 mm Hg, Po₅ averaged 36 ± 1 mm Hg, and pH averaged 7.36 ± 0.02 before vagotomy; after vagotomy Po₂ averaged 165 ± 9 mm Hg. Po₅ averaged 35 ± 1 mm Hg, and pH averaged 7.35 ± 0.02. P values are for the comparison of values determined before and after vagotomy.
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absence of increases in base-line vascular resistance (Figs. 1 and 2). Right atrial pressure averaged 4.3 ± 0.8 mm Hg before vagotomy and 4.3 ± 0.8 mm Hg after vagotomy (P > 0.05). Right atrial pressure did not change during stimulation of the carotid chemoreceptors.

Vagotomy also potentiated the reflex responses to stimulation of the carotid chemoreceptor reflexes with local hypoxia and hypercapnia (Table 2). The magnitude of potentiation of responses to hypoxia and hypercapnia was similar to the magnitude of potentiation of responses to nicotine.

**Effects of Sham Vagotomy.**—Base-line muscle perfusion pressure and responses to carotid chemoreceptor stimulation were not altered by sham vagotomy. Increases in muscle perfusion pressure in response to the intracarotid administration of nicotine (0.6 and 1.2 μg/kg, respectively) averaged 25 ± 7.1 and 29 ± 11.1 mm Hg, respectively, after sham vagotomy. Changes in systemic arterial blood pressure with chemoreceptor stimulation were also not altered by sham vagotomy.

### Table 2

<table>
<thead>
<tr>
<th>Muscle perfusion pressure (mm Hg)</th>
<th>Mean arterial blood pressure (mm Hg)</th>
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<tbody>
<tr>
<td><strong>Control</strong></td>
<td><strong>ΔNicotine</strong></td>
</tr>
<tr>
<td><strong>Before vagotomy</strong></td>
<td><strong>After vagotomy</strong></td>
</tr>
<tr>
<td>120 ± 7.1</td>
<td>125 ± 6.2</td>
</tr>
<tr>
<td>120 ± 9.4</td>
<td>128 ± 14.2</td>
</tr>
<tr>
<td><strong>P</strong></td>
<td><strong>&lt; 0.05</strong></td>
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All values are means ± se for three experiments that were separate from the experiments reported in Table 1. Systemic blood gases and pH were: Pco2 213 ± 3 mm Hg, Pco3 39 ± 3 mm Hg, and pH 7.42 ± 0.04. ΔHypoxia and hypercapnia indicates the responses to perfusion of the carotid bifurcations with blood having a Pco2 of 54 ± 6 mm Hg, a Pco3 greater than 100 mm Hg, and a pH of 7.10 ± 0.12 before vagotomy and a Pco2 of 59 ± 6 mm Hg, a Pco3 greater than 100 mm Hg, and a pH of 7.10 ± 0.13 after vagotomy. See the legend to Table 1 for additional explanations.
CAROTID BARORECEPTOR REFLEX

Effects of Cardiopulmonary Afferent Activity.—Changes in muscle perfusion pressure and systemic arterial blood pressure in response to changes in carotid sinus pressure from 75 to 125 mm Hg were potentiated after vagotomy (Figs. 3 and 4), but changes in muscle perfusion pressure and systemic arterial blood pressure during changes in carotid pressure from 125 to 175 mm Hg were not augmented after vagotomy (Figs. 3 and 4).

Right atrial pressure did not change significantly during stimulation of carotid baroreceptors either before or after vagotomy.

Effects of Sham Vagotomy.—Muscle perfusion pressures during changes in carotid sinus pressure were similar before and after sham vagotomy. Muscle perfusion pressures at carotid pressures of 75, 125, and 175 mm Hg averaged 147 ± 4.9, 125 ± 7.1, and 92 ± 7.6 mm Hg, respectively, before sham vagotomy and 145 ± 13.1, 122 ± 14.2, and 97 ± 8.3 mm Hg, respectively, after sham vagotomy. Changes in systemic arterial blood pressure during carotid baroreceptor stimulation also were not altered by sham vagotomy.

Discussion

This study suggests that reflexes arising in cardiopulmonary receptors exert an inhibitory effect on carotid chemoreceptor and baroreceptor reflexes.

CAROTID CHEMORECEPTOR REFLEX

 Interruption of cardiopulmonary afferents augmented vasoconstrictor (muscle), vasodilator (paw), and ventilatory responses to activation of chemoreceptors.

 Interruption of cardiopulmonary afferents increased base-line vascular resistance and arterial blood pressure in some experiments, but these effects were not consistent. This finding suggests that under the conditions of these experiments (carotid sinus pressure 125 mm Hg) the cardiopulmonary vagal afferents inhibited the vasomotor center in some but not all experiments. Since potentiation of the chemoreceptor reflex with vagotomy often occurred in the absence of an increase in base-line vascular resistance, it appears that suppression of the chemoreceptor reflex by the cardiopulmonary afferents may not have resulted from tonic restraint of the vasomotor center by the cardiopulmonary afferents.

Mancia et al. (8) have found that cardiopulmonary restraint of the vasomotor center decreases rapidly when the carotid sinus pressure is increased from 100 to 160 mm Hg but does not disappear until the carotid pressure reaches 160-200 mm Hg.
As just mentioned, we were frequently unable to demonstrate cardiopulmonary restraint of the vasomotor center with the carotid sinus pressure at 125 mm Hg. This difference might be explained by the fact that central venous or right atrial pressure—and, therefore, the stimulus to cardiopulmonary stretch receptors—was higher in their study (5.1 ± 0.6-5.7 ± 0.6 mm Hg) than it was in ours (4.1 ± 0.7 mm Hg). This difference would not seem to be important. Instead, the important point in interpreting our results is that under physiological conditions cardiopulmonary vagal afferents frequently exerted a suppressive effect on the chemoreceptor reflex in the absence of demonstrable tonic restraint of adrenergic discharge to the muscle resistance vessels.

Another observation also suggests that suppression of the chemoreceptor reflex did not result simply from inhibition of adrenergic discharge: interruption of cardiopulmonary afferents potentiated reflex vasodilation in the paw in addition to vasoconstriction in muscle. Reflex vasodilation in the paw during chemoreceptor stimulation has been reported to result from activation of a sympathetic dilator pathway (14). If cardiopulmonary afferents suppressed responses to chemoreceptor stimulation simply by restraining adrenergic discharge, potentiation of vasodilation in the paw after vagotomy would not have occurred. Since suppression of the chemoreceptor reflex involved reflex vasodilation in addition to vasoconstriction and since suppression frequently occurred in the absence of demonstrable tonic restraint of adrenergic discharge, we speculate that cardiopulmonary vagal afferents exert a specific suppressive effect on the chemoreceptor reflex.

We should consider the possibility that the chemoreceptor reflex was potentiated because the stimulus to carotid chemoreceptors or the firing of carotid chemoreceptors was greater after interruption of cardiopulmonary vagal afferents. It is unlikely that the stimulus was greater after vagotomy, since the carotid bifurcations were vascularly isolated and perfused at constant pressure and the doses of nicotine and the degree of hypoxia and hypercapnia were similar before and after vagotomy. Increases in adrenergic discharge to the carotid body can decrease blood flow to the carotid body and increase chemoreceptor activity and the firing of carotid chemoreceptors in response to a
given stimulus (15), but adrenergic vasoconstrictor influences on the carotid body were interrupted at the start of each experiment by sectioning the cervical sympathetic nerves. Thus, it seems doubtful that the stimulus and the firing of carotid chemoreceptors were greater after vagotomy.

The effects of vagotomy presumably did not result from interruption of afferents originating below the diaphragm, since Mancia et al. (8) have demonstrated that sectioning the vagi at the diaphragm does not exert cardiovascular effects. Ito and Scher (16) have demonstrated that some fibers originating in aortic baroreceptors course in the vagal trunk and are not interrupted by sectioning the cervical aortic depressor nerves as was done in this study. Although it might be suggested that inhibition of the chemoreceptor reflex resulted from aortic baroreceptor afferents that travel in the vagal trunk, we believe this possibility is improbable for two reasons. First, Ito and Scher (16) have found evidence for functionally significant aortic baroreceptor afferents in the vagal trunks only in a minority of dogs. Thus, if inhibition of the carotid chemoreceptor reflex in our study resulted from aortic baroreceptor afferents in the vagal trunk, one would have anticipated potentiation by vagotomy in only a minority of the experiments. In contrast, vagotomy potentiated the carotid chemoreceptor reflex in almost all of our experiments. Therefore, it seems improbable that the inhibition resulted from aortic baroreceptor afferent activity. Second, Edis and Shepherd (13) have demonstrated that the methods employed in this study to identify and section the aortic depressor nerves result in the selective denervation of aortic baroreceptors and chemoreceptors and in the preservation of cardiopulmonary vagal afferents. We suggest, therefore, that inhibition of the carotid chemoreceptor reflex resulted from the influence of cardiopulmonary vagal afferents on carotid chemoreceptor reflexes. Reflexes arising in atria, ventricles, and lung have been implicated in circulatory control, and a recent study (9) indicates that each contributes to tonic inhibition of the vasomotor center in the dog, but the precise location and characteristics of the cardiopulmonary receptors which modulate the carotid chemoreceptor reflex are not clear from this study.

Previous studies (17) suggest that the interaction of cardiovascular reflexes may be selective and involve one component of a reflex response, e.g., cardiac, but not another, e.g., vascular. Because of this selectivity, we studied the effects of interrupting cardiopulmonary afferents on ventilatory in addition to vascular responses to chemoreceptor activation. The increase in ventilatory responses to chemoreceptor stimulation after vagotomy suggests that the influence of cardiopulmonary reflexes involves both vascular and ventilatory responses.

We suggest from these studies that cardiopulmonary vagal afferent impulses exert a suppressive effect on the chemoreceptor reflex within the central nervous system. This effect appears to be distinct from vagal afferent tonic restraint of the vasomotor center. Although it is tempting to speculate that cardiopulmonary afferents act presynaptically on chemoreceptor afferent fibers, the data from this study do not permit conclusions regarding the location and mechanism of the interaction within the central nervous system.

**CAROTID BARORECEPTOR REFLEX**

 Interruption of cardiopulmonary vagal afferents augmented the gain of the carotid baroreceptor reflex when carotid sinus pressure was changed from 75 to 125 mm Hg but not when carotid sinus pressure was increased from 125 to 175 mm Hg. Guazzi et al. (5) and Pillsbury et al. (6) have demonstrated that interruption of cardiopulmonary vagal afferents potentiates the response to carotid occlusion. Although their observations suggest that vagal afferents modulate the carotid baroreceptor reflex through a central interaction, the possibility that the potentiation in their studies resulted from a greater stimulus to the carotid baroreceptors after vagotomy cannot be excluded. Base-line systemic arterial blood pressure increased after vagotomy, and, therefore, the decrease in carotid arterial blood pressure (the stimulus to carotid baroreceptors) during carotid occlusion was probably greater after vagotomy. In the present study, base-line carotid sinus pressure and the changes in pressure during the stimulation of the carotid baroreceptors were controlled to exclude the possibility that augmentation of the carotid sinus reflex after vagotomy resulted from augmentation of the stimulus.

 As indicated previously, blockade of the cardiopulmonary vagal afferents augmented the gain of the carotid sinus over the range of carotid sinus pressures from 75 to 125 mm Hg but not over the range of pressures from 125 to 175 mm Hg. In accord with this observation, several investigators (7, 8) have reported that responses to interruption of cardiopulmonary afferents are greatest when carotid sinus pressure is low and least when carotid sinus pressure is high.

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Based on these observations under physiological conditions, we can speculate on the possible significance of an inhibitory effect of exaggerated cardiopulmonary vagal afferent activity in pathologic states. For example, Higgins et al. (18) have demonstrated that reflex vasoconstrictor responses to carotid hypotension are attenuated in dogs with heart failure; we raise the possibility that this inhibition might result from exaggerated inhibitory cardiopulmonary afferent activity associated with stretch of the cardiac receptors. The results of this study also may relate to abnormalities in neurogenic control in several hypotensive states. For example, reflexes arising in cardiac receptors have been implicated in pathologic states such as coronary artery occlusion, myocardial infarction, and left ventricular outflow obstruction (12, 19-23). The results of the present study raise the possibility that reflexes arising in cardiopulmonary receptors might contribute to abnormal neurogenic control in these states partly by suppressing reflex adjustments to systemic hypotension and hypoxia through an inhibitory interaction with carotid baroreceptor and chemoreceptor reflexes.

In closing, it should be noted that this study has identified an inhibitory influence of cardiopulmonary vagal afferent activity. There is now substantial evidence supporting the existence of cardiopulmonary receptors with spinal sympathetic afferent pathways (24, 25). We did not in this study test for an interaction of cardiopulmonary sympathetic afferents and carotid reflexes.

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