Influence of Carotid Baroreceptors on Vascular Responses to Carotid Chemoreceptor Stimulation in the Dog

By Giuseppe Mancia

ABSTRACT
The modification by the carotid baroreceptors of the vascular responses to chemoreceptor stimulation was studied in anesthetized, artificially ventilated, vagotomized dogs. The carotid bifurcations were vascularly isolated and perfused with blood at constant pressures of 134, 215, and 51 mm Hg to cause intermediate, maximal, and minimal inhibition, respectively, of the vasomotor center. At each pressure, stimulation of the carotid chemoreceptors was achieved by perfusion with hypoxic hypercapnic blood. With intermediate inhibition, the chemoreceptor stimulation increased the aortic pressure by 50% and decreased the hind-limb and kidney blood flow (perfusion at constant pressure) by 59% and 19%, respectively. At carotid sinus pressures of 215 and 51 mm Hg, the effects of chemoreceptor stimulation were absent or markedly attenuated. With intermediate sinus pressure, chemoreceptor stimulation decreased the perfusion pressure of the saphenous vein by 27% (perfusion at constant flow). When the sinus pressure was increased to 215 mm Hg, the tone of the vein did not change, but chemoreceptor stimulation was without effect. The present study indicates a central interaction (which may be presynaptic) between the chemoreceptor and baroreceptor inputs such that the vascular responses to chemoreceptor stimulation are inhibited when the carotid sinus activity is maximal or minimal.

KEY WORDS renal circulation hind-limb circulation chemoreflexes baroreflexes cutaneous veins circulatory reflex control

In an analysis of the role of different reflexes that control the circulation, it is customary and proper to vary a single input to the central nervous system while eliminating or maintaining constant the inputs from other sensory zones. However, in the intact organism, the reflex effects on the vascular system of any disturbance are a function of the central interaction of information from many afferents (1). For example, when the ventilation is not controlled, the reflex increase in ventilation caused by stimulation of the carotid chemoreceptors activates the stretch receptors in the lungs; in this circumstance, the resulting vasodilator reflex opposes the primary vasoconstrictor reflex caused by the chemoreceptor stimulation (2, 3).

Heistad et al. (4) recently have shown that, in the gracilis muscle of the dog, the vasoconstrictive responses to chemoreceptor stimulation are potentiated by hypotension and inhibited by transient hypertension. However, the reflex influence exerted by the carotid sinus baroreceptors is not uniform throughout the peripheral circulation; therefore, the interaction between carotid baroreceptor and chemoreceptor reflexes could differ in different vascular beds. Hind-limb and kidney resistance vessels and cutaneous veins were studied in the present experiments, because the reflex influence of the carotid baroreceptors is greatest in the first, less in the second, and absent in the third (5-7). The carotid chemoreceptors were stimulated by local perfusion with blood of low oxygen and high carbon dioxide tensions; the stimulation was performed with the carotid sinuses maintained at pressures representing maximal, intermediate, and minimal activity of the carotid baroreceptors.

Methods
A total of 19 dogs (18-29 kg) was studied. Each dog was anesthetized with thiopental (15 mg/kg, iv) and chloralose (80 mg/kg, iv, initially, and 10 mg/kg, iv, hourly), paralyzed with gallamine (3 mg/kg, iv, hourly), and artificially ventilated at 12-14 cycles/min. Arterial blood samples were withdrawn periodically for measurement of oxygen tension (Po2), carbon dioxide tension (PCO2), and pH. PO2 was maintained above 400 mm Hg by ventilating with Q2, and PCO2 was kept between 30 and 40 mm Hg by adjusting the tidal volume; a 7.5% bicarbonate solution was administered intravenously, when needed, to maintain the pH between 7.30 and 7.40.
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Blood was taken from the terminal aorta and delivered the blood vessels and then hourly (1 mg/kg, iv). A bilateral cervical vagotomy was performed below the nodose ganglion.

PERFUSION OF CAROTID BARORECEPTORS AND CHEMORECEPTORS

The carotid bifurcations were vascually isolated by ligating the internal carotid arteries distal to the sinuses, the occipital arteries and their branches distal to the carotid bodies, and the ascending pharyngeal, lingual, and external carotid arteries (8). In the control state the isolated bifurcations were perfused at constant flow (172 ± 13 ml/min) with blood taken from the right common carotid artery and pumped through a depulsator and a heat exchanger (set at 37°C) into the cephalad portion of the cannulated common carotid arteries. Cannulas placed in the external carotid arteries returned the perfusing blood through a Starling resistance to the right external jugular vein. The Starling resistance maintained the pressure in the perfused carotid bifurcations (measured upstream from the inflow cannula) constant at the level demanded by the protocol.

To stimulate the carotid chemoreceptors, the isolated bifurcations were perfused, at the same flow used in the control condition, with blood from a donor dog that had been equilibrated in an extracorporeal oxygenator (Travenol Laboratories) with 5% CO₂ in N₂, N₂ and Pco₂ of this blood were < 40 mm Hg and > 70 mm Hg, respectively; the pH was usually < 7.15. As shown by Pelletier (9), stimulation of the carotid chemoreceptors by this combination of hypoxia, hypercapnia, and acidosis results in a near maximal reflex vascular response.

The isolation of the carotid bifurcations was considered to be adequate when (1) no backflow occurred after the incision of the common carotid arteries during cannulation for perfusion, (2) stopping the pump, emptying the bifurcations of blood, and clamping the lines of the circuit resulted in a pressure in the bifurcations of less than 40 mm Hg despite an increase in systemic arterial blood pressure to more than 200 mm Hg, and (3) the volume of the blood perfusing the excluded regions from an extracorporeal circuit showed no visible gain or loss when the perfusion pressure was lower or higher, respectively, than the systemic arterial blood pressure.

MEASUREMENTS

The pressures were measured with strain-gauge transducers (Statham P23De); flows were measured with electromagnetic flow transducers (Carolina Medical Electronics, Inc.). The transducer signals were displayed on an ultraviolet recorder (Honeywell Visiörder).

Aortic Blood Pressure.—Mean pressure was obtained by electronic damping of the pulsatile signal measured through a catheter inserted in the right brachial artery or a femoral artery.

Hind-Limb Resistance Vessels.—In some experiments, the left hind limb was perfused at constant flow (10). Blood was taken from the terminal aorta and delivered via a roller pump, depulsator, and heat exchanger (set at 37°C) to the left external iliac artery. To eliminate other sources of arterial inflow to the limb, all branches of the terminal aorta, the right external iliac artery, the last two pairs of lumbar arteries, and the deep circumflex iliac and caudal epigastric arteries were ligated. The perfusion pressure was measured just upstream from the cannulated left terminal iliac artery and set, at the beginning of the experiment, to be similar to the mean aortic pressure by adjusting the speed of the pump. Because the blood flow to the limb was constant, the changes in perfusion pressure reflected changes in vascular resistance.

In another series of experiments, the vascually isolated hind limb was perfused at constant pressure. Blood was pumped from the terminal aorta into a Windkessel system that supplied the left external iliac artery. The Windkessel was primed with donor blood and pressurized at 110 mm Hg. The hind-limb blood flow was measured with a cannulating electromagnetic flow probe placed in the arterial line. A bypass line around the flow probe allowed registration of the zero flow signal without interruption of the flow to the organ. On completion of the experiment, the flow probe was calibrated over the range of flows observed, using the dog's own blood and a pump-reservoir system. Because the pressure was constant, the changes in flow reflected changes in vascular resistance.

Kidney Resistance Vessels.—The left kidney was perfused at constant pressure by the technique described for the hind limb. The cannula was inserted into the renal artery as close as possible to its origin from the aorta, and the perfusion was started with the pressure in the reservoir maintained constant at 110 mm Hg. Interruption of the renal circulation during cannulation of the renal artery lasted less than 30 seconds. Kidney blood flow was measured with a cannulating electromagnetic flow probe inserted in the arterial line. Registration of the zero flow signal and calibration were as described for the hind limb.

Saphenous Vein.—The right lateral saphenous vein was cannulated at the ankle and perfused at constant flow by a roller pump, using blood from the terminal aorta. To eliminate other sources of inflow to the vein, all branches of the terminal aorta, the left external iliac artery, the last two pairs of lumbar arteries, and the deep circumflex iliac and deep caudal epigastric arteries were ligated; the right external iliac artery was occluded by a snare each time the observations were made. The perfusion pressure was measured just proximal to the cannulated vein, and the speed of the pump was adjusted at the beginning of the experiment to deliver a blood flow of 105 ± 6 ml/min. Because the blood flow through the vein was constant, changes in perfusion pressure reflected changes in tone of the vein wall (11).

Protocol and Data Analysis.—The carotid bifurcations were perfused with arterial hyperoxic normocapnic blood in the following sequence: (1) at a pressure similar to the existing mean aortic pressure, (2) at pressures of about 50 mm Hg and 215 mm Hg in random order, and (3) at a pressure similar to the existing mean aortic pressure. At each of these pressures, the carotid chemoreceptors were stimulated by perfusion with hypoxic hypercapnic blood. The perfusion pressures of 50 and 215 mm Hg were selected because at these pressures the carotid baroreceptors cause minimal and maximal vasomotor inhibition, re-

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TABLE 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of dogs</th>
<th>Carotid sinus pressure of 134 ± 3 mm Hg</th>
<th>Carotid sinus pressure of 215 ± 2 mm Hg</th>
<th>Carotid sinus pressure of 51 ± 2 mm Hg</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Change with chemoreceptor stimulation</td>
<td>Change with chemoreceptor stimulation</td>
<td>Change with chemoreceptor stimulation</td>
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<tr>
<td>Aortic pressure (mm Hg)</td>
<td>16</td>
<td>133 ± 3</td>
<td>80 ± 6</td>
<td>219 ± 5</td>
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<td></td>
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<td>+67 ± 4</td>
<td>+9 ± 1</td>
<td>+8 ± 2</td>
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<tr>
<td>Hind-limb perfusion pressure (mm Hg)</td>
<td>6</td>
<td>154 ± 7</td>
<td>99 ± 6</td>
<td>250 ± 6</td>
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<td></td>
<td></td>
<td>+73 ± 10</td>
<td>+11 ± 6</td>
<td>+11 ± 4</td>
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<tr>
<td>Hind-limb blood flow (ml/min)</td>
<td>5</td>
<td>64 ± 11</td>
<td>89 ± 16</td>
<td>20 ± 6</td>
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<tr>
<td></td>
<td></td>
<td>-38 ± 6</td>
<td>0 ± 1</td>
<td>-3 ± 2</td>
</tr>
<tr>
<td>Kidney blood flow (ml/min)</td>
<td>5</td>
<td>177 ± 14</td>
<td>195 ± 17</td>
<td>123 ± 9</td>
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<tr>
<td></td>
<td></td>
<td>-33 ± 12</td>
<td>-1 ± 1</td>
<td>-4 ± 2</td>
</tr>
<tr>
<td>Saphenous vein perfusion pressure</td>
<td>5</td>
<td>86 ± 10</td>
<td>83 ± 10</td>
<td>81 ± 12</td>
</tr>
<tr>
<td>(mm Hg)*</td>
<td></td>
<td>-23 ± 6</td>
<td>-1 ± 2</td>
<td>-10 ± 1</td>
</tr>
</tbody>
</table>

All values are means ± se.

*Perfusing blood was at 29 °C to augment the basal tone of the vein (12).

respectively (12-14). In the present study, this finding was verified by the absence of further pressor or depressor effects when the sinus pressure was decreased below 50 mm Hg or increased above 215 mm Hg. The chemoreceptor stimulation was maintained for 2-4 minutes, which normally was the time necessary for the reflex vascular effects to become stable. Each dog was subjected to two sequences of chemoreceptor stimuli at various carotid sinus pressures. The mean response from each dog was used to calculate the mean changes for the groups.

Results

Aortic Blood Pressure.—The changes in aortic blood pressure in response to carotid chemoreceptor stimulation at different carotid sinus pressures are summarized in Table 1. At the intermediate sinus pressure the chemoreceptor stimulation caused a mean increase in aortic pressure of 50% but at the high and low sinus pressures the mean increases were 11% and 4%, respectively. This decrease in the hypertensive response to chemoreceptor stimulation at the high and the low carotid sinus pressure was statistically significant as were the differences in the control values at the three sinus pressures (P < 0.001).

Hind Limb.—In six dogs in which the hind limb was perfused at constant flow, there was a mean increase in perfusion pressure of 47% with chemoreceptor stimulation at the intermediate sinus pressure, but at high and low sinus pressures the mean increases were 11% and 4%, respectively (Table 1). The decrease in the hypertensive response to the chemoreceptor stimulation at the high and the low sinus pressure was statistically significant (P < 0.01); the differences in the control values at the three sinus pressures were also statistically significant (P < 0.001).

In five other dogs, perfusion of the hind limb was performed at constant pressure (Table 1, Fig. 1). When the sinus pressure was at the intermediate level, chemoreceptor stimulation caused a sustained decrease in flow of 59% followed, at the end of the stimulation, by a brief marked increase. When the sinus pressure was high or low, the chemoreceptor stimulation had little effect on the flow. The decreases in the constrictor responses to the chemoreceptor stimulation at the high and the low sinus pressure and the differences in the control flows at the three sinus pressures were statistically significant (P < 0.01).

In five of the six dogs in which one hind limb
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was perfused at constant flow, the ipsilateral sympathetic chain was electrically stimulated at L4 (10 cycles/sec, 10 v, 5 msec). This stimulation increased the perfusion pressure of the limb to 306 ± 10 mm Hg, a value significantly greater (P < 0.05) than that observed during chemoreceptor stimulation at a sinus pressure of 50 mm Hg. Thus, the neurally mediated constriction of the hind-limb vessels produced by the combined baroreflex and chemoreflex was not the maximum possible.

Kidney.—The kidney was perfused at constant pressure simultaneously with the hind limb in the same five dogs described previously (Table 1, Fig. 1). With the sinus pressure at the intermediate level, the chemoreceptor stimulation produced a sustained decrease in blood flow of 19% that was not followed by an overshoot at the end of the stimulation. When the sinus pressure was high or low, the chemoreceptor stimulation had little effect on the flow. The decrease in the constrictor response to the chemoreceptor stimulation at the high and the low sinus pressure and the differences in the control flows at the three sinus pressures were statistically significant (P < 0.05 and P < 0.02, respectively).

At the end of the experiment with the kidney perfused at constant pressure, the renal nerves were cut, and the peripheral stumps were stimulated electrically (10 cycles/sec, 10 v, 5 msec); this stimulation caused a decrease in renal blood flow to below the level reached during the combined baroreflex and chemoreflex, indicating that these two reflexes did not produce maximal neurogenic vasoconstriction.

Saphenous Vein.—In these studies in five dogs, to make the reflex venodilation in response to the chemoreceptor stimulation more evident, the basal tone of the saphenous vein was augmented by decreasing the temperature of the perfusing blood to 29°C (14) (Table 1, Fig. 2). The differences in perfusion pressures at the different sinus pressures were not statistically significant. During chemoreceptor stimulation the perfusion pressure decreased by a mean of 27% at the intermediate carotid sinus pressure; there was little response at the high sinus pressure and a decreased response (12% decrease) at the low sinus pressure. The decreases in the venodilatory responses to the chemoreceptor stimulation at the high and low sinus pressures were statistically significant (P < 0.02).

Dissociation of Chemoreceptor and Baroreceptor Stimulation.—Two sets of experiments were performed to determine (1) if stimulation of the chemoreceptors contributed to the hypertension accompanying perfusion of the carotid bifurcations with hyperoxic normocapnic blood at a pressure of 50 mm Hg and (2) if the absence or attenuation of the hypertensive response to perfusion of the carotid bifurcations with hypoxic hypercapnic blood at pressures of 220 and 50 mm Hg was due to lack of chemoreceptor stimulation.

In four dogs, the carotid bodies were excluded from the isolated carotid bifurcations by ligation of the occipital arteries at their origin from the external carotid arteries; this procedure abolished the hypertension produced by perfusion of the carotid bifurcations with the hypoxic hypercapnic blood but did not decrease the hypertension produced by the decrease in the carotid sinus pressure to 50 mm Hg, indicating that the hypertension elicited by this degree of sinus hypotension did not depend on chemoreceptor excitation.

In three dogs, the left carotid sinus was vascularly isolated according to the Moissejeff technique (15) and filled with oxygenated Krebs-Ringer's-bicarbonate solution at pH 7.4. The left occipital artery was ligated at its origin from the external carotid artery, thereby excluding the carotid body chemoreceptors from the isolated left carotid sinus. The right carotid bifurcation was pre-
pared as described in Methods; in addition, however, the right carotid sinus was stripped to denervate the baroreceptors. The left carotid sinus was maintained at a constant pressure of 140 mm Hg, and the right carotid bifurcation was perfused with the arterial normoxic normocapnic blood and with the hypoxic hypercapnic blood at pressures of 140, 220, and 50 mm Hg. In nine observations (three in each dog) at each of these three perfusion pressures, the results were as follows. (1) Changes in right carotid sinus pressure from 140 mm Hg to 220 mm Hg to 40 mm Hg, with the sinus perfused with arterial blood, did not cause significant changes in aortic pressure (158 ± 8, 151 ± 8, and 163 ± 11 mm Hg, respectively), confirming the finding that denervation of the right carotid baroreceptors had been achieved. (2) Perfusion of the right carotid sinus with hypoxic hypercapnic blood caused, at each of the aforementioned right carotid sinus pressures, significant increases in aortic pressure (26 ± 2, 32 ± 4, and 27 ± 2 mm Hg, respectively). (3) These increases were not significantly different from each other, indicating that the chemoreceptor stimulation was independent of the changes in perfusion pressure over this range.

Discussion

In the present experiments, near maximal chemoreceptor stimulation was obtained by perfusing the isolated carotid regions with blood of PO₂ < 40 mm Hg, PCO₂ > 70 mm Hg, and pH < 7.15 (9, 16, 17). The stimulation was applied when the intrasinus pressure was maintained at levels that caused maximal (215 mm Hg), intermediate (134 mm Hg), and minimal (51 mm Hg) activity of the carotid baroreceptors (12, 13). Section of the cervical vagosympathetic nerves interrupted the fibers from the aortic arch and the cardiopulmonary region (18); it also interrupted the sympathetic nerves to the carotid bodies, thus preventing any reflex modification of their afferent nerve traffic (19–21).

Carotid Baroreflex.—Withdrawal of the inhibition exerted by the carotid baroreceptors caused a constriction of the hind-limb (77% decrease in flow) and kidney resistance vessels (36% decrease in flow) but had no significant effect on the saphenous vein. This finding confirms the work of others which shows that the carotid baroreflex has more effect on the hind-limb resistance vessels than it does on the kidney resistance vessels (5, 6) and that the saphenous vein does not participate (7).

Carotid Chemoreflex.—Stimulation of the chemoreceptors while the carotid sinus pressure was maintained at the intermediate level caused an increase in aortic blood pressure, a constriction of the hind-limb resistance vessels, and a dilatation of the saphenous vein, thereby confirming previous studies (2, 9, 22, 23). In addition, these experiments provided evidence that the carotid chemoreceptors cause a constriction of the kidney resistance vessels (19% decrease in flow) that is less than that of the hind-limb vessels (59% decrease in flow). Thus, the carotid chemoreflex and baroreflex are similar in the relative control that they exert on these two vascular beds.

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Maximal Carotid Baroreceptor Activity.—The reflex vascular effects of the chemoreceptor stimulation were abolished or markedly attenuated when the input of the carotid baroreceptors was maximal. This finding confirms the observation of Heistad et al. (4) that the reflex vasoconstrictor response of the gracilis muscle to carotid chemoreceptor stimulation is inhibited by increased arterial blood pressure. The current study showed that this inhibition occurs not only in the vascular areas in which the carotid baroreceptors exert their major control (hind limb) but also in those areas in which this control is less (kidney) or absent (saphenous vein).

The experiments in which the carotid sinus was denervated and the carotid area was perfused at different pressures (40–220 mm Hg) with hypoxic hypercapnic blood showed chemoreceptor stimulation to be independent of perfusion pressure. Thus, the ineffectiveness of the chemoreflex when the input of the carotid baroreceptors was maximal is not due to failure of the chemoreceptors to be stimulated. With maximal carotid baroreceptor activity, the hind-limb and kidney vessels were dilated and thus capable of constriction if the chemoreceptor stimulation had increased the sympathetic adrenergic outflow. Because the cutaneous vein was partially constricted by perfusion at 29°C, venodilatation could have occurred if the chemoreflex had operated. Thus, the abolition of the carotid chemoreflex by the carotid baroreflex is due to a central interaction of their afferent signals.

In regard to the mechanisms of this central interaction, the simplest explanation is that the inhibition of the vasomotor neurons by the baroreceptors was sufficiently powerful to prevent excitation of these neurons by the chemoreceptors. However, the study of the behavior of the saphenous vein implies that this explanation is not the only one. This vein is unresponsive to changes in carotid baroreceptor activity and dilates with chemoreceptor stimulation at a carotid sinus pressure of 140 mm Hg. The results were as follows. (1) Changes in right carotid sinus pressure from 140 mm Hg to 220 mm Hg to 40 mm Hg, with the sinus perfused with arterial blood, did not cause significant changes in aortic pressure (158 ± 8, 151 ± 8, and 163 ± 11 mm Hg, respectively), confirming the finding that denervation of the right carotid baroreceptors had been achieved. (2) Perfusion of the right carotid sinus with hypoxic hypercapnic blood caused, at each of the aforementioned right carotid sinus pressures, significant increases in aortic pressure (26 ± 2, 32 ± 4, and 27 ± 2 mm Hg, respectively). (3) These increases were not significantly different from each other, indicating that the chemoreceptor stimulation was independent of the changes in perfusion pressure over this range.

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134 mm Hg but not at a pressure of 215 mm Hg. Thus, a maximal baroreceptor input prevents the influence of the chemoreceptors on this vein without having a direct effect on the central neurons controlling the vein. One explanation is that the afferent traffic from the chemoreceptors to the central neurons that control the saphenous vein is inhibited presynaptically by the baroreceptor afferents. Studies in the cat have shown that an increase in carotid baroreceptor activity can depress the neural structures that control the activity of the cortex (24), the rage behavior (25), and the somatic reflexes (26, 27). The present study demonstrates that the carotid baroreceptors also inhibit the neural structures involved in the carotid chemoreflex.

Minimal Carotid Baroreceptor Activity.—When the carotid baroreceptor activity was minimal, perfusion of the chemoreceptors with hypoxic hypercapnic blood caused a small but statistically significant dilatation of the saphenous vein (12%) but little or no additional constriction of the hindlimb and kidney resistance vessels. It could be argued that a decrease in carotid sinus pressure to 50 mm Hg when the isolated sinuses were perfused with blood from the systemic circulation might have activated the chemoreflex to such a degree that no further activation could have occurred when hypoxic hypercapnic blood was used. This possibility seems unlikely because the pressor response to a decrease in sinus pressure from 210 to 50 mm Hg was the same before and after exclusion of the carotid bodies. Biscoe et al. (28) have observed that, in the cat with normal arterial blood gases, a decrease in carotid sinus pressure to 50 or 60 mm Hg causes little or no excitation of the chemoreceptors. In the present study when the P02 was in excess of 400 mm Hg and the flow through the carotid bifurcations was constant, chemoreceptor excitation was not likely. Also, with the decrease in sinus pressure to 50 mm Hg, arterial blood might have leaked into the "isolated" sinus region through small vessels that had not been ligated, thereby preventing the chemoreceptor stimulation. Although this possibility cannot be excluded, the tests used to verify the completeness of vascular isolation (see Methods) suggest that any such leak would have been small. Also, with the right carotid baroreceptors denervated and the left carotid baroreceptors maintained at the intermediate pressure, the vascular responses to stimulation of the right chemoreceptors were the same regardless of the perfusion pressure, indicating that any leakage of arterial blood into the isolated carotid bifurcations was insufficient to modify the response. Third, the constriction of the hind-limb and kidney vessels with carotid sinus hypotension might have prevented a further constriction by the chemoreflex. However, further constriction was obtained when the sympathetic nerves to these beds were stimulated. The most likely explanation, therefore, is that in the absence of an inhibitory input from the carotid baroreceptors, the pool of central neurons cannot be further excited by a chemoreceptor input.

These observations differ from those obtained with the gracilis muscle (4) in which the vasoconstrictor response to stimulation of the carotid chemoreceptors by nicotine is enhanced during hemorrhagic hypotension.

In conclusion, when the carotid sinus pressure is normal, strong stimulation of the carotid chemoreceptors causes a constriction that is relatively greater in the hind-limb resistance vessels than that in the kidney resistance vessels. The same finding is true when the inhibition exerted by the carotid baroreceptors is removed in the absence of chemoreceptor stimulation. The response of the hind-limb and the kidney vessels to carotid chemoreceptor stimulation is absent or attenuated during maximal or minimal carotid baroreceptor activity. The same pool of central neurons that control the hind-limb and the kidney resistance vessels is acted on by the carotid baroreflex and chemoreflex. The central neurons controlling the cutaneous veins are not affected directly by the carotid baroreceptors but are inhibited by excitation of the carotid chemoreceptors; this inhibition is prevented when the carotid baroreceptors are strongly activated, suggesting a presynaptic inhibition.

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