Inhibition of Adrenergic Outflow to Peripheral Blood Vessels by Vagal Afferents from the Cardiopulmonary Region in the Dog

By Giuseppe Mancia, David E. Donald, and John T. Shepherd

ABSTRACT

In closed-chest atropinized dogs anesthetized with chloralose and ventilated artificially, the aortic nerves were cut beneath the nodose ganglion, and the carotid sinuses were either denervated or vascularly isolated and maintained at a pressure of 40 mm Hg. Denervation was established by the failure of the pressoreceptor systems to respond to mechanical stimulation. Cold block or section of the cervical vagi resulted in statistically significant increases in arterial blood pressure (18%), heart rate (3%), central venous pressure (13%), hind-limb perfusion pressure (12%), pressure within the occluded spleen (18%), and efferent renal sympathetic nerve activity (28%). There was no change in saphenous vein perfusion pressure. The vascular responses were abolished by a-receptor blockade and were not affected by section of the vagi at the diaphragm. When the pressure in the vascularly isolated innervated carotid sinuses was varied from 40 to 220 mm Hg, the vascular responses to vagal block decreased steeply between 100 and 160 mm Hg and were absent at 200 mm Hg. Thus, receptors in the cardiopulmonary region with afferent vagal fibers exert a continuous restraint on the sympathetic adrenergic outflow to resistance and capacitance vessels, especially when the input from the carotid baroreceptors is decreased.

KEY WORDS

cardiopulmonary afferents low-pressure receptors resistance vessels

aortic depressor nerves carotid baroreceptors circulatory reflexes

capacitance vessels vagal block renal sympathetic activity

There is ample evidence in the literature that vagally innervated receptors in the cardiopulmonary region of the dog can produce circulatory reflexes when they are stimulated by chemical substances or by distention of the structures in which they are located (1, 2). However, there is no evidence that these reflexes are active under more normal circumstances in the closed-chest dog.

Studies (3-5) in closed-chest anesthetized rabbits and cats have shown that interruption of vagal afferents from cardiopulmonary receptors causes an increase in systemic arterial blood pressure. Additional studies (6) in cats have shown that the increase in pressure is accompanied by constriction of the resistance vessels in the skeletal muscle, the intestines, and the kidney. These experiments indicate that the receptors continuously inhibit the sympathetic outflow.

The experiments described in this paper were conducted to determine if a similar continuous inhibition is present in dogs and, if so, to study its effect on peripheral resistance and capacitance vessels and its modification by changes in activity in the carotid sinus nerves.

Methods

A total of 44 dogs (10-22 kg) were anesthetized with sodium thiopental (15 mg/kg, iv) and chloralose (80 mg/kg, iv). Anesthesia was maintained by periodic infusion of smaller doses of chloralose. To prevent hemodynamic changes produced mechanically by changes in respiration, the dogs were paralyzed with gallamine triethiodide (3 mg/kg, iv) and artificially
ventilated with 100% \(\text{O}_2\) with a Harvard respirator. The respiratory rate was 10–14 cycles/min, and the tidal volume was adjusted to yield peak inspiratory pressures of 10–12 cm \(H_2O\). Arterial \(P_{\text{O}_2}\), \(P_{\text{CO}_2}\), and pH were measured periodically. \(P_{\text{CO}_2}\) was kept between 30 and 40 mm Hg by adjusting the tidal volume, and bicarbonate was infused as needed to maintain the pH between 7.30 and 7.40. Heparin (3 mg/kg, iv) was administered each hour to ensure that the effects of vagal block or section were due only to interruption of vagal afferents.

**Carotid Sinuses.**—The Moisjeff technique (7) was used to achieve vascular isolation of the carotid sinuses. The isolated sinuses were perfused at the desired steady pressure with oxygenated Krebs-Ringer's-bicarbonate solution. The occipital arteries were ligated at their origin from the external carotid arteries to exclude the carotid body chemoreceptors from the influence of pressure changes (8). The common carotid arteries were stripped in the neck to denervate the baroreceptors located in this region (9). When desired, the carotid sinuses were denervated by stripping the adventitia of all the vessels at the bifurcation between the internal and external carotid arteries. The abolition of the increase in systemic arterial blood pressure during occlusion of the common carotid arteries was considered to be evidence of denervation.

**Aortic Baroreceptors.**—Under a dissecting microscope, the aortic nerves were isolated from the cervical vagosympathetic trunks at their junction with the superior laryngeal nerves and were sectioned. Identification was often aided by recording the electrical activity in the aortic nerves. Section of the aortic nerves, performed at low steady pressure in the carotid sinuses, usually resulted in a large, sustained increase in arterial blood pressure. The efficacy of aortic nerve section in denervating the aortic chemoreceptors was tested by injecting cyanide into the aortic root (see Results). Further evidence for denervation of the aortic baroreceptors was obtained by using the aortic sleeve technique described by Donald and Edis (10). Through a left thoracotomy, the descending aorta was incised between two clamps, and a large, curved, stainless steel cannula was inserted into the aortic arch. The proximal end was just distal to the orifice of the coronary arteries, and the distal end was 1–2 cm below the left subclavian artery. The distal aorta was cannulated, and the two cannulas were connected through a wide Silastic tube. In this way the systemic circulation continued to be perfused. Cerebral flow was preserved by connecting the femoral arteries with the common carotid and vertebral arteries. By tying the brachiocephalic and left subclavian arteries, the space between the upper cannula and the aortic wall was vascularly isolated, and the aortic arch could be distended with blood or saline at the desired pressure. In two dogs, ligatures were placed around the vertebral, common carotid, omocervical, and costocervical arteries; thus, the brachiocephalic trunk, the right subclavian artery, and the origins of the common carotid arteries were included in the vascularly isolated area subjected to pressure distention.

**Nerve Section or Block.**—The cervical vagi were sectioned below the nodose ganglion after the administration of procaine (2%). Vagal block was achieved by cooling. The temperature was decreased to between 0°C and −1°C and was monitored by a small thermoprobe interposed between the thermode and the nerve. Block was reversed by circulating water at 37°C.

At the end of each experiment, cold block was again performed, and the vagi were severed just below the site of the block. Because section of the nerve did not alter the hemodynamic changes caused by the block, it was assumed that the afferent vagal fibers involved in the circulatory response had been blocked by this degree of cooling. In addition, the arterial hypotension produced by injecting veratridine (1 \(\mu g/kg\)) into the right atrium was abolished by vagal cold block. In some experiments, cold block of the vagi was performed above and below the point of nerve section with no hemodynamic effects. Therefore, the vagal fibers were not directly stimulated by cooling.

**Blood Pressures and Heart Rate.**—Arterial blood pressure and central venous pressure were measured through catheters inserted into a brachial or a femoral artery and a jugular vein, respectively. From the jugular vein the catheter was positioned in or near the right atrium. The carotid sinus pressure was monitored through a polyethylene tube inserted into the perfusion line of the carotid sinuses. Heart rate was calculated from the arterial blood pressure record or from an electrocardiogram. All pressures were measured using strain-gauge transducers.

**Resistance Vessels.**—The left hind limb and the left kidney were perfused with blood at constant flow and 37°C via roller pumps. Blood for perfusion was taken from the right external iliac artery. Perfusion pressure was measured just upstream from the input cannula, and pump speed was adjusted to provide an initial perfusion pressure similar to the mean arterial blood pressure. All branches of the terminal aorta, the last two pairs of lumbar arteries, the deep circumflex iliac artery, and the deep caudal epigastric artery were ligated to eliminate other sources of arterial inflow to the hind limb. The limb was considered to be vascularly isolated if (1) the perfusion pressure decreased to about 20 mm Hg when the pump was stopped and (2) the increase in arterial blood pressure produced by an intravenous injection of norepinephrine was not accompanied by a concomitant increase in the perfusion pressure of the hind limb. Under these conditions, changes in the perfusion pressure reflected changes in vascular resistance.

**Capacitance Vessels.**—The isovolumetric spleen was used as an index of the reaction of the splanchic capacitance vessels (11). The spleen was vascularly isolated except for the splenic artery and vein, which were occluded by snares when observations were made. During these periods, blood volume in the spleen remained constant, and changes in splenic venous pressure indicated changes in tone of the splenic capacitance elements. Electrical stimulation of the left splanchic nerve (10 v, 15 cycles/sec, 3 msec) at the end of the experiment increased pressure in the occluded spleen by 30–40 mm Hg.
To study a cutaneous capacitance bed, the right lateral saphenous vein was cannulated at the ankle and perfused at constant flow with a roller pump. Blood was taken from the right external iliac artery and maintained at 37°C by a heat exchanger. The speed of the pump was set to give initial perfusion pressures of 20-40 mm Hg. The iliac vein pressure was monitored through a catheter inserted into the vein from a small collateral. Other sources of inflow to the saphenous vein were eliminated by ligating all vessels in the region of the terminal aorta, as described for hind limb perfusion, and by clamping the left external iliac artery while observations were made. Since blood flow in the vein was constant, changes in perfusion pressure (inflow pressure minus outflow pressure) were caused by contraction or relaxation of the smooth muscle of the vein.

Cardiac Output.—Indocyanine green was injected into the right atrium and sampled at the aortic root. A densitometer attached to the aortic sampling catheter provided continuous recording of the dye concentration. The densitometer was calibrated at the end of each experiment.

Renal Nerve Activity.—A branch of the left renal nerve was isolated, dissected free, and teased so that a few fibers could be used for recording. The prepared nerve was crushed or cut distally, and the central end was mounted on a pair of platinum electrodes 2-3 mm apart. The nerve signals were amplified (amplifier A105 and a-c amplifier A103/B, Lexington Instruments, Inc.) and transformed into standardized pulses which then were integrated. By visually adjusting a discriminator at the beginning of the recording, the noise level was excluded from the standardized pulses. The equipment permitted counting of signals at 0.25-msec intervals (14).

Protocol and Data Analysis.—Recording was started about 2 hours after the initial dose of the anesthetic; at this time rectal temperature, arterial Pco2 and pH were within the normal limits, and arterial Po2 was greater than 300 mm Hg. The tidal volume was maintained constant during vagal cooling. The vags were cooled at intervals of 5-10 minutes. Cooling was maintained for 1-2 minutes, which usually was the time necessary for the hemodynamic changes to stabilize. The circulatory changes observed during cooling reflected the degree of activity in the nerves before cooling; the rapid return of control values with rearming indicated that the nerves had not been damaged by the cooling. Measurements made immediately before and after cooling were averaged and compared with the data obtained during cooling. Data from single experiments were averaged and means, SD, and SE were calculated for the groups. The statistical significance of the difference in the means was evaluated by Student's t-test for paired observations.

Results

SECTION OR COLD BLOCK OF CERVICAL VAGI WITH BILATERAL DENERVATION OF CAROTID SINUS

In closed-chest dogs, the aortic nerves were cut in the neck after denervation of the carotid sinuses. In this experiment, only systemic arterial blood press-ure and heart rate were monitored to determine the effect of sectioning or blocking the vags in the absence of extensive surgical interference with the cardiovascular system.

Bilateral Section or Block (14 Dogs).—The control mean arterial blood pressure and heart rate were 172 ± 7 (SE) mm Hg and 203 ± 10 (SE) beats/min, respectively. The plateau values after the vagal section or the values sustained through the period of vagal block were 210 ± 7 mm Hg and 211 ± 10 beats/min for mean changes of 22% and 4%, respectively. These changes were statistically significant (P < 0.001 and P < 0.02, respectively). The higher arterial blood pressure produced by the nerve section was still present 20 minutes later. In some of the dogs, pulsus alternans developed after vagal block or section; it most frequently developed during the initial steep increase in arterial blood pressure.

Unilateral vs. Bilateral Section or Block (9 Dogs).—No attempt was made to quantitatively compare the effects of block of the left and the right vagus, because the surgical manipulation involved in isolating the aortic nerves might have damaged the vagal trunks to varying degrees. However, it was evident that interrupting both vags had a greater effect on arterial blood pressure than did interrupting only one. The control mean arterial blood pressure was 174 ± 11 (SE) mm Hg. After random block or section of the right or the left vagus, the arterial blood pressure was increased by 22 ± 4 mm Hg; after block or section of the other vagus, a further increase of 23 ± 5 mm Hg occurred. These increases were statistically significant (P < 0.001).

A small but statistically significant (P < 0.02) increase in heart rate (10 ± 3 beats/min) occurred after bilateral vagal block or section. Inconsistent changes in heart rate were recorded after unilateral block or section.

CIRCUITARY EFFECT OF INTERRUPTION OF VAGAL AFFERENTS DERIVED FROM RECEPTORS IN THE CARDIOPULMONARY REGION

To establish that the reflex changes in blood pressure originated from receptors in the cardiopulmonary region, it was necessary to exclude the possibility that the effects of vagal cooling depended on accessory aortic fibers, that is, fibers subserving aortic baroreceptors and running with the vags outside the aortic nerves, or on receptors in the abdomen. The following evidence indicates that the reflex under study originated in the cardiopulmonary region.
Completeness of Denervation of Chemoreceptors and Baroreceptors in Aortic Arch after Section of Aortic Nerves in Neck.—In 21 closed-chest dogs (including the 14 dogs in the preceding section), a catheter was inserted into a common carotid artery, manipulated into the left ventricle, and withdrawn until the tip was in the ascending aorta. The position of the catheter was verified at the end of the experiment. The carotid sinuses were denervated or excluded from the systemic circulation and maintained at a constant pressure. Injection of sodium cyanide (0.2–1 mg/kg) into the aortic root produced an immediate increase in mean arterial blood pressure (31 ± 2 [SE] mm Hg). However, after aortic nerve section, hypotension (—18 ± 5 mm Hg) occurred. The abolition of the hypertensive response to cyanide indicated denervation of the aortic chemoreceptors (15). Because the fibers that subserve baroreceptors and chemoreceptors in the aortic region travel in the same nerve bundles (16), denervation of the aortic chemoreceptors can be taken as presumptive evidence for baroreceptor denervation. After chemical confirmation of aortic arch denervation, vagal cooling or section produced an increase in arterial blood pressure in each dog.

In five open-chest dogs with denervated carotid sinuses, the response of the systemic arterial blood pressure to distention of the aortic arch was tested before and after section of the aortic nerves in the neck. In two other dogs, the aortic arch was distended only after the aortic nerves were cut. An original record is shown in Figure 1 and the results are summarized in Figure 2. In all instances, section of the aortic nerves in the neck abolished the hypotensive response to a 300-mm Hg distention of the isolated aortic arch. In two of the five dogs the vascularly isolated region included the brachiocephalic, right subclavian, and origins of the common carotid arteries (16, 18–20). These observations do not deny the possibility that fibers from the aortic arch region may travel in the vagal trunks outside the aortic nerves, but they do indicate that, if present, such fibers do not play an important role in cardiovascular control.

During cooling of the vagi in the neck after the aortic nerves had been cut and denervation of the aortic baroreceptors had been established, the pressure in the aortic arch and its major branches was maintained constant at 40 mm Hg. Vagal cooling was accompanied by an increase in arterial blood pressure (Figs. 1 and 2). These results confirm the conclusion of Edis and Shepherd (17) that section of the aortic nerves in the neck is a reliable means of acute denervation of the baroreceptors in the aortic arch. Section of the aortic nerves in the neck also appears to denervate the baroreceptors located in the area of the brachiocephalic trunk, the right subclavian artery, and the origin of the common carotid arteries (16, 18–20). These observations do not deny the possibility that fibers from the aortic arch region may travel in the vagal trunks outside the aortic nerves, but they do indicate that, if present, such fibers do not play an important role in cardiovascular control.
Absence of Contribution of Vagal Afferents From Abdomen to Circulatory Effect of Vagal Interruption.—In five dogs the increases in mean arterial blood pressure during cervical vagal cooling were 40, 45, 72, 65, and 23 mm Hg, respectively. Cooling after section of the vagi at the diaphragm gave increases of 30, 62, 72, 38, and 25 mm Hg in the same dogs, respectively.

EFFECT OF VAGAL COOLING ON DIFFERENT COMPONENTS OF CARDIOVASCULAR SYSTEM

To study how the vagal inhibitory control was distributed throughout the systemic circulation, the response of different circulatory components to vagal cooling was examined in 23 closed-chest dogs in which the aortic nerves had been cut and the carotid sinuses had been vascularly isolated. To reproduce the condition of the earlier experiments in which the carotid sinuses were denervated, the vagi were cooled at a sinus pressure of 40-50 mm Hg. In this way, the vasomotor inhibition exerted by the carotid baroreceptors was minimal or absent (21, 22).

Heart Rate and Circulatory Pressures.—The grouped data referred to in this paragraph are given in Table 1. Vagal cooling caused statistically significant increases in mean arterial blood pressure, hind-limb perfusion pressure, central and splenic venous pressures, and heart rate. All variables returned to control levels on rewarming. Systemic arterial and hind-limb perfusion pressures showed a transient undershoot during rewarming; the undershoot of the hind limb was greater. Vagal block had no consistent effect on saphenous vein perfusion pressure. This condition was not caused by damage to the venous innervation, because electrical stimulation of the right paravertebral chain at L-4 (10 v, 15 cycles/sec, 10 msec) produced a rapid increase in saphenous vein perfusion pressure.

Kidney Circulation.—In three dogs, the kidney was perfused at constant flow. When renal perfusion pressure was increased by decreasing the pressure in the carotid sinuses, a plateau value of less than 250 mm Hg was reached. Vagal block was followed by a large, rapid increase in renal perfusion pressure to values over 300 mm Hg; at this time the perfusion pump was stopped to prevent damage to the kidney (Fig. 3, left).

Because of the difficulty in the interpretation of the results when the kidney was perfused at constant flow, the renal effenter sympathetic activity was recorded in five dogs. When the vagi were cooled, the impulse frequency increased in four
dogs and did not change in one dog. When the nerves were warmed, the impulse frequency often decreased transiently below the level existing prior to the cooling. With respect to the mean initial impulse frequency (119 ± 6/sec [SE]), the increase with cooling was 28 ± 10%, and the decrease after cooling was 13 ± 4%. Both the increase and the decrease were statistically significant (P < 0.05). An example of the response of the sympathetic activity to vagal cooling is shown in Figure 4.

Cardiac Output.—The possible contribution of cardiac output to the increase in arterial blood pressure caused by vagal block was examined in two dogs. Indicator-dilution curves were recorded before, during, and after vagal cooling. Four coolings were done at intervals of 15-20 minutes to allow the dye to be eliminated from the circulation (23). In the first dog the mean aortic pressure before and after vagal cooling was 174 ± 6 (sd) mm Hg, and during cooling it was 213 ± 6 mm Hg. The corresponding values for cardiac output were 3 ± 0.4 liters/min before and after cooling and 2.9 ± 0.4 liters/min during cooling. In the second dog, the mean arterial blood pressure was 187 ± 11 mm Hg before and after vagal cooling, and 204 ± 10 mm Hg during cooling. The corresponding values for cardiac output were 2.6 ± 0.2 liters/min before and after cooling and 2.6 ± 0.2 liters/min during cooling.

Alpha-Receptor Blockade.—In three dogs the increases in arterial blood pressure, hind-limb perfusion pressure, and splenic venous pressure in response to vagal cooling were abolished by injection of phenoxybenzamine (5-10 mg/kg, iv).

INTERRELATIONSHIP BETWEEN CARDIOPULMONARY AND CAROTID BARORECEPTORS

In five dogs, the vagi were cooled while different pressures were maintained in the carotid sinuses. Vagal block increased the mean arterial blood pressure maximally at a carotid sinus pressure of 50 mm Hg (Fig. 5). The response was almost as high when the block was performed at a sinus pressure of 100 mm Hg, but it rapidly decreased as sinus pressure was increased from 100 to 160 mm Hg and was abolished at 200 mm Hg. The response was restored when vagal block was repeated at a sinus pressure of 50 mm Hg. The maximal increase in arterial blood pressure obtained by blocking the vagi at low carotid sinus pressure was reproducible in each dog.

The responses of different parts of the cardiovascular system to vagal cooling at carotid sinus pressures of 200-220 mm Hg were compared in the same dog with those obtained at sinus pressures of

![Figure 3](http://circres.ahajournals.org/)

**(FIGURE 3)**

Effect of vagal cold block at low (left) and high (right) carotid sinus pressure on renal perfusion pressure in dog with aortic nerves cut. Scale at right refers to carotid sinus, aortic, and renal perfusion pressures. Blood flow pump was turned off when renal perfusion pressure exceeded 300 mm Hg during the vagal block (left). Temperature was recorded from thermistor on surface of one vagus. CS = carotid sinus pressure, CV = central venous pressure, R = renal perfusion pressure, and Ao = aortic pressure.

![Figure 4](http://circres.ahajournals.org/)

**(FIGURE 4)**

Effect of vagal cold block on renal sympathetic activity in dog with aortic nerves cut and carotid sinus pressure at 40 mm Hg. Mean renal nerve traffic was 70 impulses/sec before block, 110 impulses/sec during block, and 50 impulses/sec after block.
40–50 mm Hg. An example of the different responses of aortic pressure and pump-perfused kidney to the two levels of carotid sinus pressure is shown in Figure 3; the grouped data are shown in Table 1. Vagal cooling at sinus pressures of 200–220 mm Hg had no significant effect on arterial blood pressure, hind-limb perfusion pressure, heart rate, central venous pressure, and venous pressure within the occluded spleen. Under the same conditions, no changes were recorded in renal sympathetic activity (three dogs) and renal perfusion pressure (three dogs). These results contrasted with the well-defined changes seen when the vagi were cooled at sinus pressures of 40–50 mm Hg. Also, comparison with the base-line values before block showed that, with the exception of the central venous pressure, decreasing or abolishing inhibitory sinus nerve traffic by decreasing sinus pressure from 220 to 50 mm Hg had a larger effect on the circulation than did vagal cooling.

This dominant effect of the carotid sinus is seen in the experiment shown in Figure 6. An increase in sinus pressure from 50 to 200 mm Hg caused aortic pressure to decrease from 220 to 100 mm Hg. Vagal cooling at a sinus pressure of 50 mm Hg resulted in an increase in aortic pressure from 240 to 275 mm Hg. Repeated carotid sinus hypertension during sustained cooling again decreased aortic pressure to 100 mm Hg, despite the increased vasoconstriction resulting from interruption of the inhibitory vagal traffic.

**Discussion**

These experiments showed that, in the anesthetized atropinized dog with its aortic nerves sectioned and its carotid sinuses denervated or isolated and maintained at low constant intrasinus pressure, cold block or section of the cervical vagi caused a sustained increase in systemic arterial blood pressure, hind-limb perfusion pressure, central and splenic venous pressures, renal nerve traffic, and heart rate. Cardiac output and saphenous vein perfusion pressure were not changed during vagal cold block. The positive responses were abolished by α-receptor blockade. Therefore, under these conditions, the carotid sinus is a dominant modulator of arterial pressure.
conditions vagal afferents exert a continuous widespread inhibition on sympathetic outflow to the peripheral circulation. Although the principal response observed was an increase in peripheral resistance, this response does not deny that a tonic inhibitory influence was also exerted on the heart. Vagal block or section was accompanied by a small increase in heart rate. This increase might have been greater if the dogs had not been atropinized.

In these experiments there was no consistent modification of the response to cervical vagal block after section of the vagi at the diaphragm. Ito and Scher (24) have reported that fibers, presumably originating from baroreceptors in the major intrathoracic arteries, can travel in the left vagus outside the aortic nerve. The present data demonstrate that the pressure response to stimulation of the aortic chemoreceptors and the depressor response to stimulation of the aortic baroreceptors or the baroreceptors in the region of the brachiocephalic, right subclavian, and common carotid arteries were abolished by bilateral aortic nerve section. Therefore, receptors in the major intrathoracic arteries did not contribute to the pressure response to vagal block. The above data allow the conclusion that the vagal afferents originate in the cardiopulmonary region.

In sinoaortic denervated cats and rabbits (3-5), interruption of the cervical vagi causes an increase in systemic arterial blood pressure, indicating that a continuous inhibitory influence is exerted through afferent vagal fibers. The present experiments confirm this finding in the dog and reinforce the evidence that the continuous inhibitory influence comes from receptors in the cardiopulmonary region; however, it is not known if the receptors responsible are in the heart, the lungs, or both. Oberg and White (6) reported that in the cat the afferent vagal inhibitory fibers travel mainly in the cardiac nerves. Guazzi et al. (5) demonstrated in the cat and Pillsbury et al. (4) demonstrated in the rabbit that inhibitory fibers were in both the pulmonary and the cardiac vagal branches. However, as discussed by these authors (4-6), it is difficult to determine the precise source of the inhibitory reflex by selective section of intrathoracic branches of the vagi because of the frequent interconnections between cardiac and pulmonary plexuses.

Previous studies in the cat (6, 25) and the rabbit (14, 26) suggested that the vagal afferents exerted their inhibition mainly on the renal circulation, whereas the carotid baroreceptors predominantly affected the muscle vessels. In the present experiments, using constant-flow perfusion, the vasoconstrictive response to vagal cooling was more powerful in the kidney than it was in the hind limb. However, when changes in renal perfusion pressure during constant flow are used to estimate a reflex response of the resistance vessels, the results can be difficult to evaluate (27). This finding may be due to an unknown degree of activation of local vasoregulatory mechanisms in the kidney as a consequence of the neurogenic constriction. Thus, whether the reflex effects of interruption of vagal afferents are greater in the kidney than they are in the hind limb cannot be determined from the current studies.

The failure to find vasomotor changes in the cutaneous veins in response to vagal block was not surprising, since previous studies (28) have shown that they do not respond to vagal reflexes during hemorrhage. These veins represent a specialized component of the capacitance system: they do not participate in the baroreflexes from the high-pressure system (29), they dilate when the carotid chemoreceptors are activated (30), and they have a specific function in thermoregulation (31).

In their studies in cats, Oberg and White (6) found that the pressor responses to vagal cooling were only moderate when the arterial baroreceptors were functioning normally but were pronounced when the common carotid arteries were occluded. In the present study with the carotid sinuses isolated from the circulation and maintained at constant pressure, the degree of vasoconstriction with interruption of the vagal afferents depended on the pressure in the sinuses; vasoconstriction was absent when this pressure was high and maximal when it was low. It is possible that the circulatory changes produced by increasing the carotid sinus pressure decreased the stimulation of the cardiopulmonary receptors subserved by vagal afferents. However, the insignificant change in central venous pressure observed during carotid sinus hypertension argues against complete elimination of activity of these receptors. Therefore, it appears that a central interaction is involved. A complete description of such interaction at the vasomotor center of the afferent input from the carotid baroreceptors and the baroreceptors subserved by vagal afferents depends on a knowledge of the function and range of the activity of the different receptors in the heart and the lungs.
CARDIOPULMONARY DEPRESSOR NERVES

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References

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