Demonstration that the Atria, Ventricles, and Lungs Each Are Responsible for a Tonic Inhibition of the Vasomotor Center in the Dog

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ABSTRACT
To localize the areas of the cardiopulmonary region involved in tonic inhibition of the vasomotor center, anesthetized dogs were subjected to sinoaortic denervation and diaphragmatic vagotomy. Afferent vagal nerve traffic was interrupted in the neck by cooling. With the venous return taken from the venae cavae, oxygenated extracorporeally, and returned to the aorta, the heart was removed, leaving the ventilated lungs (condition 1), and the lungs and the ventricles were removed, leaving the beating atria (condition 3). With the venous return taken from the pulmonary arteries, oxygenated extracorporeally, and returned to the left atrium, the lungs were removed, leaving the intact working heart (condition 2), and the lungs were removed and the atria were denervated, leaving the working innervated ventricles (condition 4). Vagal cooling increased aortic pressure by 25 ± 2 (SE) mm Hg in condition 1, by 36 ± 2 mm Hg in condition 2, by 29 ± 2 mm Hg in condition 3, and by 29 ± 7 mm Hg in condition 4. Removing the atria in condition 3 or denervating the ventricles in condition 4 abolished the reflex response. Thus, afferent vagal nerves from the lungs and the heart tonically inhibit the vasomotor center. The inhibition exerted by the heart is caused by receptors in the atria and the ventricles.

KEY WORDS
aortic nerves atrial receptors cardiac receptors
ventricular receptors pulmonary receptors vagal afferents
veratrum vagal blockade

In the rabbit, cat, and dog (1-4), interruption of vagal nerve traffic from the cardiopulmonary region results in a widespread sympathetically mediated vasoconstriction, indicating that receptors in this region exert a tonic inhibition on the sympathetic outflow to the systemic circulation. The question whether this tonic inhibition originates from receptors distributed generally throughout the cardiopulmonary region or from receptors localized to specific areas such as the lungs, the atria, and the ventricles is as yet unanswered.

The present investigation was undertaken to resolve this point. The experiments were performed on extracorporeally oxygenated dogs in which the afferent vagal nerve traffic from the cardiopulmonary region was interrupted when (1) the heart had been removed and the lungs left in situ, (2) the lungs had been removed and the heart left in situ, (3) the lungs and the ventricles had been removed and the atria left in situ, and (4) the lungs had been removed and the atria denervated so that afferent traffic came only from the ventricles. This study showed that the lungs, atria, and ventricles each exert a tonic inhibition on the sympathetic vasomotor outflow.

Methods

GENERAL PROCEDURE
In 32 dogs, anesthesia was induced with sodium thiopental and chloralose (15 and 80 mg/kg body weight, respectively, iv) and maintained with chloralose (10 mg/kg, hourly). The dogs were paralyzed with gallamine triethiodide (3 mg/kg, hourly) and artificially ventilated with oxygen. The ventilation rate was 12-14 cycles/min, and the tidal volume was adjusted to give peak inspiratory pressures of 12-14 cm H2O. Arterial oxygen tension (P02), carbon dioxide tension (Pco2), and pH were measured periodically; P02 was always higher than 400 mm Hg, Pco2 was between 30 and 40 mm Hg, and pH was between 7.30 and 7.40. Heparin was given intravenously prior to cannulation of blood vessels (3 mg/kg) and thereafter at hourly intervals (0.2 mg/kg). The cervical vagi were placed within thermodes. Nerve conduction was blocked by cooling until the temperature of a thermistor situated at the surface of the nerve was between 0° and −1°C. Cold block was maintained for 2-3 minutes and then was terminated by circulating water at 37°C. Cooling produced effective blockade of afferent nerve traffic since nerve section during the period of low temperature did not produce further circulatory effects. The rapid return to control values with rewarming indicated that the nerves had not been damaged by the cooling (4).
In all of the dogs, the vagi were divided at the diaphragm. The common carotid arteries were isolated from their connection with the vagal nerves, and the carotid sinuses were denervated by stripping the external carotid, internal carotid, ascending pharyngeal, and occipital arteries at their origin. Abolition of the hypertensive response to occlusion of the common carotid arteries was taken as evidence of denervation. Under a dissecting microscope, the aortic nerve on each side was identified at the junction of the vagal nerve with the cranial laryngeal nerve, traced caudally to its junction with the vagosympathetic trunk, and cut (4, 5). Several studies have documented that this technique acutely abolishes the baroreceptor and chemoreceptor reflexes from the aortic arch and the major intrathoracic arteries (2, 4–8). Additional proof of abrogation of the aortic baroreceptor reflex was afforded in the present experiments by failure of cervical vagal blockade to elicit a pressor response after denervation or after removal of the heart and lungs at the end of the experiment.

Systemic arterial blood pressure was measured through a catheter placed in the abdominal aorta. Mean pressure was obtained by electronic damping of the pulsatile signal. During extracorporeal circulation, systemic blood flow was constant; hence, changes in aortic pressure reflected changes in total peripheral resistance. Aortic pressure was measured during stable periods before, during, and after vagal cold block. The values before and after blockade were averaged, and these values were compared with those measured during the block. This difference reflected the tonic influence of the afferent vagal nerves.

EXTRACORPOREAL CIRCULATION

The lungs of a second dog were isolated and used to oxygenate the blood of the experimental dog (9). Blood was pumped from a reservoir in the oxygenator circuit into the cannulated pulmonary trunk of the isolated dog lungs. Oxygenated blood was returned from the lungs to the reservoir through a cannula tied into the left atrium. A T-shaped cannula was placed in this return line to allow a second pump to deliver oxygenated blood to the experimental dog whose systemic venous flow was returned to the reservoir. The mean transit time through the extracorporeal circuit was greater than 2 minutes. The isolated lungs were ventilated with a 95% O₂-5% CO₂ mixture, and the ventilation was adjusted to maintain the PCO₂ and pH of the experimental dog in the normal range. Blood flow in the oxygenator circuit always was 15% greater than the flow pumped into the experimental dog.

Condition 1: Only Lungs In Situ.—A schema of this preparation is shown in Figure 1 (left). The chest was opened through a midsternal split. The subclavian artery and both venae cavae were divided between ligatures, cannulated, connected with the oxygenator circuit, and rendered air-free. Total extracorporeal circulation used the venae cavae as the outflow of venous blood from the systemic circulation and the left subclavian artery as the inflow of arterial blood to the systemic circulation. The aorta and the pulmonary trunk were divided at their origins. The pulmonary veins and the venae cavae were sectioned at the pericardial reflection, and the entire heart was removed. Inflow to the left subclavian artery was nonpulsatile.

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and was stabilized between 80 and 100 ml/kg body weight of the experimental dog. Ventilation of the lungs remained at the level established prior to removal of the heart; the lungs continued to be perfused by the bronchial arteries whose blood drained from the sectioned pulmonary veins.

The cervical vagi were cold blocked after the chest had been opened but while the circulation was intact with both heart and lungs in situ; vagal block was repeated after removal of the heart with the dog on extracorporeal circulation.

**Condition 2: Only Heart In Situ.**—A schema of this preparation is also shown in Figure 1 (right). The right side of the chest was opened by removal of the fifth rib, and the pulmonary vessels and bronchi were divided individually, thus permitting removal of the entire right lung. The right pulmonary artery was exposed and cannulated near its origin at the pulmonary trunk. The cannula was connected to a Silastic tube (0.25 inches, i.d.) filled with heparinized saline, and the tube was exteriorized between the tenth and the eleventh rib. The chest then was closed. The left side of the chest was opened by removal of the fifth rib, and a length of Silastic tubing with a multiperforated tip was placed within the left atrial appendage, connected to the perfusion circuit, and rendered air-free.

Perfusion used the right pulmonary artery perfusion line to direct systemic venous blood into the oxygenator circuit. Oxygenated blood was returned through the left atrial catheter. The left pulmonary artery was exposed at its origin at the pulmonary trunk, cannulated, and used to return systemic venous blood. The left lung was removed in the same manner as that described for the right lung. The pericardium was removed. Thus, on total extracorporeal circulation, systemic and coronary venous blood returned in a normal manner to the right atrium and was pumped by the right ventricle into the systemic circulation as described under condition 2. Vagal block was repeated with only the atria in situ after the ventricles had been removed. At the end of the experiment, after the atria had been removed, the reactivity of the dog was confirmed by the arterial hypotension or hypertension that resulted from electrical stimulation of the cephalic end of the divided vagal trunks.

**Condition 4: Only Heart In Situ, Atria Denervated.**—The lungs were removed and the heart was perfused in situ as described under condition 2. The following steps were then taken to denervate the atria.

First, the venae cavae were mobilized, stripped of their surrounding tissues, and crushed at their entrance into the heart. Second, with caval occlusion, the lateral wall of the right atrium was widely incised and the interatrial septum was divided and reanastomosed from its superior to its inferior margin. The line of division was as close as possible to the ventricular base. The incision of the lateral wall of the atrium was sutured. Third, the lateral and superior walls of the right and left atria were crushed circumferentially with a thin-bladed clamp, one jaw of which was inserted into the atrial chambers. The line of crushing began with the incision in the free atrial wall of the right atrium, continued across the superior aspect of the atria behind the aorta and the pulmonary trunk, and proceeded along the inferior margin of the left atrium to the medial junction of the inferior vena cava. This circumferential line of division was placed as close as possible to the atrioventricular groove. Fourth, the ventricles were exposed, the ventricles were removed. The left and right pulmonary arteries were ligated below the entrance of the costocervical vertebral veins. The venous return from the upper body was drained via T-shaped cannulas placed in the external jugular veins. This procedure allowed preservation of nerve fibers entering the heart along the superior vena cava (12). Second, the right coronary artery was mobilized and cannulated in a distal direction upstream from the sinus node artery. The right coronary artery then was tied distal to the sinus node artery. The latter vessel thus served to perfuse the atria with oxygenated blood from the extracorporeal circuit. Third, the ascending aorta and the pulmonary trunk were divided between ligatures, and the left circumflex artery and the first part of the left anterior descending coronary artery were dissected free and removed. Fourth, the heart was opened, the ventricular septum and the fossa ovalis were excised, and a plug-shaped cannula (diameter 4 cm, depth of plug 1 cm) was inserted into the common atrial chamber. The plug was secured in position by encircling ligatures placed in the dissected atrioventricular groove. The ventricles were removed, leaving a common atrial pouch perfused with oxygenated blood through the sinus node artery.

The blood draining from the coronary sinus into the atrial pouch was returned to the reservoir by a large catheter inserted via the right atrial appendage. The atrial pressure was adjusted to the required value by changing the height at which this catheter was raised or lowered until the mean pressure in the pulmonary trunk was about 10 mm Hg. At this pressure, the baroreceptors at the bifurcation of the pulmonary trunk and along the pulmonary arteries do not exert a vasomotor inhibition (10, 11).

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nervation of the atria, both atria and ventricles were paced electrically at 120 beats/min and at an atrioventricular delay of 100 msec.

Cervical vagal block was performed initially after the division of the interatrial septum and the lateral walls of the right atrium. At this point, the left atrium and the dorsal wall of the right atrium were innervated, the dog was on extracorporeal oxygenation, and the in situ heart supported the systemic circulation. Vagal block was repeated after denervation of the atria and, finally, after the denervation was extended to the ventricles by stripping the aorta and the pulmonary trunk and painting with 5% phenol solution. At the end of the experiment, the reactivity of the dog was shown as described previously.

That the preceding technique had denervated the atria was shown in four dogs by loss of the atrial response to supramaximal electrical excitation of the cardiac ends of the divided cervical vagus nerves (20 v, 40 cps, 1 msec) and the anterior poles of the decentralized stellate ganglia (10 v, 10 cps, 3 msec). This approach was chosen because control responses were sharply defined and of considerable magnitude and because volume distention of the right atrium only produced small and inconstant changes in peripheral resistance; also this procedure can cause tachycardia in the totally denervated heart (13). The control decreases in heart rate during stimulation of the right and left vagus nerves ranged from 67 to 79 beats/min and from 26 to 57 beats/min, respectively. Control increases in heart rate during stimulation of the right and left stellate outflows ranged from 90 to 121 beats/min and from 26 to 78 beats/min, respectively. Stimulation of either stellate ganglion caused a two- to threefold increase in the right and left atrial pressure pulses. Stimulation of the right and left vagus decreased the right atrial pressure pulse by a mean of 47% and 52%, respectively. After atrial denervation, stimulation of the cardiac efferent autonomic nerves was without effect on heart rate or atrial pressure. The reactivity of the denervated atria was demonstrated by the increases in heart rate and right and left atrial pressure pulses that immediately followed an injection of l-norepinephrine (5-30 µg) into the aortic root.

Results

ROLE OF RECEPTORS IN THE LUNGS

Cold block of the cervical vagi in five dogs increased aortic pressure before and after removal of the heart (Fig. 2). Before heart removal, the control mean aortic pressure was 167 ± 12 (se) mm Hg, and the increase caused by vagal block was 62 ± 11 mm Hg. After heart removal, the values were 166 ± 9 mm Hg and 25 ± 2 mm Hg, respectively. In these two conditions, the peak inspiratory pressures were 13 ± 0.6 cm H_{2}O, and 14 ± 1 cm H_{2}O, respectively.

ROLE OF RECEPTORS IN THE HEART

In five dogs, cervical vagal block increased aortic pressure before and after removal of the lungs (Fig. 3). Before lung removal, the control mean aortic pressure was 169 ± 12 mm Hg, and the increase caused by vagal block was 40 ± 5 mm Hg. After lung removal, the values were 176 ± 7 mm Hg and 34 ± 3 mm Hg, respectively. Left and right atrial pressures, respectively, were 5 ± 0.9 mm Hg and 3 ± 0.8 mm Hg before and 5 ± 0.9 mm Hg and 2 ± 0.8 mm Hg after lung removal.

Six other dogs were studied only after removal of
Increase in aortic blood pressure during cervical vagal cold block with only the atria in situ. Data are from seven dogs with their carotid sinuses denervated, their aortic nerves cut, their vagi divided at the diaphragm, and their lungs removed. In each section of the figure, the vertical lines on the left represent increases with vagal block when the entire heart was in situ, and the vertical lines on the right represent increases obtained after removal of the ventricles, with the atria only in situ. B = before removal of the ventricles and A = after removal of the ventricles.

ROLE OF RECEPTORS IN THE ATRIA

In seven pneumonectomized dogs, vagal block increased aortic pressure when the total heart was in situ and when the ventricles were removed, leaving only the atria (Fig. 4). With the total heart in situ and supporting the systemic circulation, the control mean aortic pressure was 162 ± 4 mm Hg, and the increase caused by vagal block was 28 ± 3 mm Hg. With only the atria in situ, the values were 193 ± 7 mm Hg and 29 ± 2 mm Hg, respectively. In the first situation the pressures in the left and the right atrium were 10 ± 0.5 mm Hg and 2 ± 0.2 mm Hg, and in the second situation the pressure in the common atrial pouch was 9 ± 1 mm Hg. In each of these dogs, the atria were eventually removed. After this procedure, the control mean aortic pressure was 206 ± 8 mm Hg, and vagal block caused no change. An example of the effect of vagal block before and after removal of the atria is shown in Figure 5.

In six dogs with only the atria in situ, a dose of veratridine (0.5 μg/kg in 0.5 ml of saline) was injected into the artery that supplied the sinus node; this injection resulted in an immediate decrease in aortic pressure (33 ± 6 mm Hg). Injection of a similar amount of saline caused no change in aortic pressure.

ROLE OF RECEPTORS IN THE VENTRICLES

In five pneumonectomized dogs in which the systemic circulation was maintained by the in situ heart, vagal block increased aortic pressure both before and after denervation of the atria (Fig. 6). Before atrial denervation, the control mean aortic pressure was 130 ± 7 mm Hg, and the increase produced by vagal block was 30 ± 3 mm Hg. After atrial denervation, the values were 148 ± 6 mm Hg and 29 ± 7 mm Hg, respectively. Left and right atrial pressures, respectively, were 9.5 ± 0.5 mm Hg and 2.9 ± 0.2 mm Hg before and 9.8 ± 0.4 mm Hg and 3.0 ± 0.2 mm Hg after atrial denervation. In each of these dogs, after denervation was extended to the ventricles, the control mean aortic pressure was 138 ± 9 mm Hg, the left atrial pressure was 10.7 ± 0.8 mm Hg, and the right atrial pressure was 4.4 ± 0.4 mm Hg. In no
Denervation of Atria:

FIGURE 6

Increase in aortic blood pressure during cervical vagal cold block with the heart in situ and only the ventricles innervated. Data are from five dogs with their carotid sinuses denervated, their aortic nerves cut, their vagi sectioned at the diaphragm, and their lungs removed. In each section of the figure, the vertical lines on the left represent increases with vagal block when the intact heart was in situ, and the vertical lines on the right represent the increases after denervation of the atria, with only the ventricles innervated. B = before denervation of the atria and A = after denervation of the atria.

instance did vagal block cause a change in aortic pressure after ventricular denervation. An example of the effect of vagal block before and after denervation of the ventricles is shown in Figure 7.

After atrial denervation alone, a dose of veratridine (1 μg/kg body weight) was injected into the left heart in all five dogs; this injection resulted in an immediate decrease in aortic pressure (47 ± 1 mm Hg). This response was abolished when the pulmonary trunk, the aortic root, and the main left coronary artery were stripped and painted with phenol. This test afforded further evidence of afferent denervation of the ventricles.

PATHWAYS OF VAGAL FIBERS FROM CARDIAC AND PULMONARY RECEPTORS

Studies in the cat (2) and the rabbit (1) have used blockade or section of the thoracic vagal trunks at specific anatomic locations as a means of distinguishing between the heart and the lungs as a source of tonic vasomotor inhibition. The interpretation of these studies was based on the assumption that most or all of the pulmonary afferent fibers joined the main vagus trunk at or distal to the pulmonary hilus (pulmonary plexus) and that those from the heart entered at a more cranial location (cardiac plexus). In the present experiments, the dogs with only their lungs in situ were eventually subjected to sustained inflation of the lungs at 40 cm H₂O. Lung inflation caused a reflex systemic hypotension (Fig. 8, left); the hypotensive response was almost as large after section of the vagi immediately cephalad to the pulmonary hilus and was significantly less after section of the vagi in the neck. In dogs with only their hearts in situ, the left and right atria were simultaneously distended by balloons filled with 30–40 ml of saline at
Effect of bilateral vagal section immediately cephalad to the pulmonary hilus (tracheobronchial junction) and in the neck on reflex systemic hypotension caused by inflation of the lungs (A) and by balloon distention of the atria (B). In A only the lungs were in situ, and in B only the heart was in situ. Responses to lung inflation and atrial distention are shown as changes from control.

37°C. The heart was excluded from the systemic circulation by delivering the arterial blood through the left subclavian artery and draining the venous blood from the venae cavae. Distention of the atria (Fig. 8, right) resulted in a reflex arterial hypotension that was greater before than after vagal section immediately cephalad to the pulmonary hilus; a small hypotensive response was still present after section of the vagi in the neck. Thus, in the dog, section of the intrathoracic vagus at different levels cannot be used to produce selective interruption of vagal afferents from the lungs and the heart.

Discussion

In dogs with their carotid sinuses denervated, their aortic nerves cut, and their vagi sectioned at the diaphragm, cervical vagal cold block increased aortic blood pressure owing to systemic vasoconstriction when (1) the ventilated lungs were in situ and the heart was removed, (2) the working heart was in situ and the lungs were removed, (3) the beating atria were in situ and the lungs and the ventricles were removed, and (4) the beating ventricles were in situ, the lungs were removed, and the atria were denervated. Thus, the present experiments demonstrate that vagally innervated receptors in the ventilated lungs and the beating heart both exert a continuous reflex inhibition on the vasomotor center. These experiments also demonstrate that the inhibition from the heart originates both in the atria and in the ventricles.

Previous studies, based on the fact that balloon distention of the atrial appendages (14), the pulmonary vein-left atrial junctions (15), and the superior vena cava-right atrial junction (16) caused no maintained systemic vasodilatation, have denied the atrial receptors a role in control of systemic vascular resistance (17). This position is refuted by the present observation that, at pressures close to normal, receptors in the isolated atria exert a tonic reflex restraint on the systemic resistance vessels.

Three points of evidence suggest that the lungs are a less important source of vasomotor inhibition than is the heart. After removal of the heart (Fig. 9A), the mean increase in aortic pressure during vagal block was 40% of that from both the heart and lungs. After removal of the remaining left lung (Fig. 9B), the mean pressor response to vagal block was 85% of that in the control situation, indicating that the contribution of the left lung was only 15% of the total. Also, the mean pressor response to vagal block with only the heart in situ was greater (34 mm Hg, Fig. 9B) than that with only the lungs in situ (25 mm Hg, Fig. 9A).

The data from the studies of the atria and the ventricles (Fig. 9C and D) suggest that normally these two areas exert equal degrees of vasomotor inhibition, because vagal block with only the atria or with only the ventricles caused the same mean increase in pressure. The increase in both cases also was similar to the increase obtained from the total heart. One possible interpretation of these
findings is that atrial and ventricular fibers have a degree of occlusion at the level of the vasomotor center.

In our opinion, any comparison of the relative importance of the different areas studied should be made with caution for the following reasons. (1) There was an interval of 40-60 minutes and considerable surgical interference between the control and the final test situation. (2) There was no certainty that the preparation of the isolated organ or area preserved all of its afferent fibers.

The reflex hypotension produced by injection of veratridine (the Bezold-Jarisch reflex) has been attributed to receptors in the left ventricle (18, 19). In the present experiments, however, veratridine produced a reflex hypotension when it was injected into the sinus node artery in dogs with their lungs, atria as well as in the ventricles. In the present experiments, however, veratridine or area preserved all of its afferent fibers.

Produced by distention of the vascularly isolated and the final test situation. (2) There was no siderable surgical interference between the control ventricles, and major coronary vessels removed. Thus, the Bezold-Jarisch reflex can originate in the atria as well as in the ventricles.

In 1931, Koch (20) defined the aortic nerve in dogs, and his technique was used by Heymans and Bouckaert (21) in their studies of neurogenic hypertension in dogs. Studies from this and other laboratories have shown that section of the aortic nerves in the neck abolishes the reflex hypotension produced by distention of the vascularly isolated aortic arch, brachiocephalic trunk, and right subclavian artery (4-5) and also abolishes the hypertensive response to injection of cyanide into the aortic root (4-5). These studies showed that this procedure acutely abrogated aortic baroreceptor and chemoreceptor reflexes.

This conclusion recently has been challenged by Itô and Scher (22). In acute studies, these investigators recorded a decrease in aortic blood pressure in response to stimulation of the central segment of the sectioned “peripheral aortic nerves” after section of the cervical aortic nerves in one of three dogs and one of five cats. The hypotension was stated to be 10 mm Hg or less. It should be appreciated that two of the nerves that compose the peripheral aortic nerve—the dorsal and the ventromedial cervical cardiac nerves—supply filaments not only to the aortic arch but also to the pretracheal plexus, a primary area of nerve distribution for the heart (23).

Oberg and Thorén (24) have recorded a reflex decrease in blood pressure caused by electrical stimulation of nonmyelinated afferent fibers in the cardiac nerve in the cat. These fibers normally are silent or have a sparse irregular discharge and are not easy to identify by standard electrophysiological techniques (25). Kulak (26) has shown, in cats and rabbits, that afferent impulses traveling along the nonmyelinated fibers of the vagus and the aortic depressor nerves from cardiac receptors to the central nervous system are capable of exerting profound and varied effects on arterial blood pressure. Thus, the conclusion of Itô and Scher (22) that the reflex response they observed was due solely to excitation of aortic depressor fibers is not valid. In the current studies, using dogs with bilateral section of the cervical aortic nerves, the pressor responses to vagal block were present when the heart and the lungs were in situ and invariably absent after their combined removal. Since, in the latter situation, the arterial blood pressure was well above the level at which the aortic baroreceptors exert a tonic vasomotor inhibition (27-29), absence of the pressor response demonstrated abrogation of the aortic baroreceptor reflex.

Infrequently, no aortic nerve can be identified (5). When this situation occurs, the experiment has to be abandoned because the nerve probably lies within the vagal trunk. An example of this situation was encountered in the present series of experiments; on one occasion, no left aortic nerve was found. In this experiment, vagal cold block caused similar pressor responses (> 40 mm Hg) before and after removal of the heart and lungs. The pressor response was not modified by cervical section of the right vagus but was abolished by cervical section of the left vagus, implying that in this instant the aortic baroreceptor fibers traveled within the left vagus nerve. It should be emphasized that the evidence for loss of aortic baroreceptor and chemoreceptor reflexes after section of the cervical aortic nerves applies only to the acute situation.

Electrophysiological studies have shown that spontaneously active vagal fibers from the cardiopulmonary region are both myelinated and nonmyelinated, the latter having a sparse and irregular discharge (25, 30-35). The demonstration that each of the three regions studied exerted an inhibitory influence on the vasomotor center allows speculation as to the type of fiber involved in each situation. Selective blockade of afferent vagal traffic may provide the answer to this question.

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References

1. PILLSBURY HRC III, GUAZZI M, FREIS ED: Vagal afferent
16. Kappagoda CT, Lindsen RJ, Snow HM: Reflex increase in heart rate from distension of the junction between the superior vena cava and the right atrium. J Physiol (Lond) 220:177-197, 1972
30. Paintal AS: Vagal receptors and their reflex effects. Physiol Rev 53:159-227, 1973
31. Coleridge HM, Coleridge JCG, Kidd C: Cardiac receptors in the dog, with particular reference to two types of afferent ending in the ventricular wall. J Physiol (Lond) 174:323-339, 1964

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