Functional Distribution of the Peripheral Cardiac Sympathetic Pathways

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ABSTRACT

The functional peripheral cardiac sympathetic pathways of the dog were delineated in isovolumetric left ventricle preparations. In unpaced hearts, supramaximal stimuli at 2 cycles/sec to the right stellate ganglion caused the heart rate to increase 2.8 times more than stimuli applied to the left side. In paced hearts, such stimuli evoked rises in left ventricular systolic pressure which were 2.3 times greater when applied to the left than to the right stellate ganglion. Tonic cardiac sympathetic impulses appear to funnel through the stellate ganglia. Decentralization of the stellate ganglia abolished the ventricular responses to stimulation of the carotid baroreceptors and cephalic ischemia. Approximately two thirds of the tonic and baroreceptor reflex influences to the left ventricle entered the stellate ganglia from lower segments of the thoracic paravertebral chain, and one third entered from the communicating rami of the stellate ganglia. The ratio of the effects of complete decentralization of the left to those of decentralization of the right stellate ganglion was 1.6, both with respect to eliminating sympathetic tone and abolishing the baroreceptor reflex.

ADDITIONAL KEY WORDS heart rate ventricular contractility cardiac innervation carotid sinus baroreceptors cardiac control carotid-ventricular reflex stellate ganglia cardiac sympathetic nervous tone cephalic ischemia thoracic sympathetic chain anesthetized dogs

Numerous studies have been conducted to delineate, from the functional standpoint, the peripheral pathways of the sympathetic nerve fibers to the heart. Such studies have yielded valuable information concerning the chronotropic, dromotropic, and inotropic influences of sympathetic nervous activity upon the heart. Furthermore, the relative influence of certain of the major components of the peripheral sympathetic system upon specific cardiac functions has been assessed, but no serious attempt has been made so far to analyze this problem quantitatively. Previous experiments have been carried out upon animals with more or less intact cardiovascular systems. In intact systems changes in heart rate, impulse conduction, and contractile force induced by sympathetic stimulation evoke changes in arterial and central venous pressures which in turn modify cardiac performance mechanically or reflexly. These secondary alterations in cardiac activity complicate any attempt to analyze quantitatively the primary effects of the major components of the sympathetic nervous system upon the heart.

To avoid such difficulties, the experiments to be described were performed upon isovolumetric preparations of the left ventricle of dogs. By this expedient, arterial pressure could be held constant, and venous return to the left ventricle consisted only of a trivial volume of Thebesian drainage. The principal aim of the present series of experiments was to evaluate quantitatively the
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major peripheral pathways conducting tonic sympathetic impulses and conducting the efferent sympathetic impulses for the carotidodecidual reflex. The role of the stellate ganglia in mediating the cardiac component of the cephalic ischemia response was also assessed. The preparation permitted measurement of changes in heart rate and ventricular contractile force in response to electrical stimulation of right and left stellate ganglia.

Methods

Experiments were conducted upon 27 mongrel dogs which were anesthetized with morphine sulfate, 2 mg/kg iv, followed 30 min later by an intravenous infusion of urethane, 800 mg/kg, and chloralose, 60 mg/kg. Through a midline incision in the neck, both vagosympathetic trunks were transected, and a tracheal cannula was inserted. Intermittent positive-pressure respiration was instituted, and the chest was opened transversely at the level of the fourth intercostal space. Iso-
volumetric left ventricle preparations were made essentially as previously described. After heparin (3.4 mg/kg) was administered intravenously to prevent blood coagulation, the coronary vasculature and the cephalic portion of the animal were perfused by gravity from an overflow reservoir set at a fixed height above the animal, equivalent to a hydrostatic pressure of about 100 mm Hg. This reservoir was connected to a cannula in the proximal segment of the ligated left subclavian artery. The descending thoracic aorta just distal to the origin of the left subclavian artery was ligated just above the diaphragm. The overhead reservoir was continually filled with blood from a rotating-disc oxygenator by means of a roller pump. Blood to prime this system was obtained from donor dogs that had been anesthetized with thiamylal sodium (12.5 mg/kg iv). Blood from the superior vena cava and from the lingual artery drained any slight leakage of blood to the venous end of the oxygenator. To produce an abrupt elevation of intrasinusal pressure, the drain was occluded, and the tubing from the elevated reservoir was unclamped. Intrasinusal pressure was registered by means of a strain gauge.

Pressure in the aortic arch was measured by means of a Radiometer pH meter, type TIT 1c. The pH of the blood leaving the arterial end of the oxygenator was continually measured by means of a Radiometer pH meter, type TTT lc. The pH of the blood was maintained at 7.4 by means of a slow drip of a saturated solution of NaHCO3. The outputs of the pH meter and of the strain gauges were recorded on a Beckman S-II Dynograph and were also stored on magnetic tape (Honeywell, LAR 7400). The Pco2 of the perfused blood were not measured; in other experiments using this apparatus, the Pco2 of the reservoir blood was 48 mm Hg and the oxygen saturation was 100%.

Results

STELLATE GANGLION STIMULATION

The typical changes in heart rate and left ventricular performance were assessed by measuring the pressure generated within a balloon, and the pH of the blood leaving the arterial end of the oxygenator was continually measured by means of a Radiometer pH meter, type TTT lc. The typical changes in heart rate and left
The changes in heart rate and left ventricular pressure in response to electrical stimulation of the right (RS) or left (LS) stellate ganglion, or of both ganglia simultaneously (RS + LS). Each stimulus was applied for precisely 1 min, as indicated by the bars between the heart rate and ventricular pressure tracings. Stimuli consisted of square-wave pulses of 15 v, 2 msec, 2 cycles/sec. In the upper half of the figure, the heart was beating spontaneously; in the lower half, the heart was paced at a rate of 230 beats/min.

FIGURE 2
Composite data from 8 experiments in which identical stimuli of 1-min duration were applied sequentially to the right and left stellate ganglia. The increments in left ventricular pressure above control (left half of figure) were measured in paced hearts. The increments in heart rate above control (right half of figure) were measured in spontaneously beating hearts. The height of each bar represents the mean change. The vertical line through the top of each bar in this and other figures represents the standard error of the mean.
duration, 2 msec, and train duration, 1 min. The spontaneous mean heart rate in these animals prior to stimulation was 170 ± 7.6 (SE) beats/min. As the right half of the figure shows, the rate increased by 55.8 ± 6.4 beats/min when the right stellate ganglion was stimulated. The increase amounted to only 19.9 ± 6.6 beats/min when the left stellate ganglion was stimulated; in 2 of the 8 experiments there was no detectable change. In no instance did stimulation of the left stellate ganglion elicit a greater response than equivalent stimulation of the right stellate ganglion.

The relative effectiveness of right and left stellate stimulation upon ventricular contractility is just the reverse of their relative influence upon heart rate. In the experiment shown in the lower half of Figure 1, right stellate stimulation evoked a rise in left ventricular systolic pressure (LVSP) of 60 mm Hg in the paced heart, whereas left stellate stimulation elicited a rise of 160 mm Hg.

The left half of Figure 2 displays the left ventricular pressure from the same 8 experiments from which the heart rate data in the figure were derived. In all cases, the same stimuli were employed before and after pacing. The control LVSP just prior to stimulation was 99.1 ± 9.0 (SE) mm Hg. The mean increase in LVSP evoked by right stellate stimulation was 45.3 ± 8.7 mm Hg. Left stellate stimulation elicited an elevation of 105.5 ± 9.0 mm Hg. In none of these 8 experiments did right stellate stimulation yield a greater response than equivalent stimulation of the left stellate ganglion.

The results of simultaneous stimulation of the right and left stellate ganglia are illustrated in the lower right section of Figure 1. When both ganglia were stimulated for 1 min, LVSP increased by 210 mm Hg. The increases elicited by the same stimuli applied to the right and left ganglia individually were 60 and 160 mm Hg, respectively, as stated above. Thus, the sum of the individual effects (220 mm Hg) was slightly greater than the magnitude of the response to simultaneous stimulation of both ganglia. In most of the experiments in which similar stimuli (15 V, 2 msec, 2 cycles/sec) were employed, the sum of the individual effects exceeded the magnitude of the response to combined stimulation. The disparity was usually greater than that illustrated by Figure 1. When frequencies above 2 cycles/sec were employed, the individual responses were greater, and an even wider disparity existed between the sum of the individual responses and the responses to combined stimulation.

When smaller responses were evoked, usually by employing frequencies less than 2 cycles/sec (especially to the left stellate ganglion), the sum of the individual responses was not significantly different from the magnitude of the response to combined stimulation. In 4 of 6 such experiments, the frequency of stimulation applied to the left stellate ganglion (mean, 1.6 cycles/sec) was less than that applied to the right side (mean, 3.0 cycles/sec). In the other two experiments, the same frequencies were used on each side. The mean sum of the individual responses (47 + 77 = 124 mm Hg) was approximately equal to the magnitude of the response to combined stimulation (122 mm Hg). The mean value of LVSP in the absence of stimulation was 93.5 ± 10.2 (SE) mm Hg. The ratio of the combined response to the sum of individual responses was computed for each experiment; it ranged between 0.87 and 1.10. The mean ratio was 0.98 ± 0.034, which does not differ significantly from 1.00. It was therefore concluded that the responses to weak and moderate stimuli summate linearly; the responses to stronger stimuli do not. The calculations described in the succeeding sections of this paper are based upon this conclusion.

CARDIAC SYMPATHETIC TONE AND THE CAROTID BARORECEPTOR REFLEX

In the paced left ventricle, the reductions in tonic sympathetic activity and the changes in response to a standard baroreceptor stimulus were determined after successive interruptions of the cardiac sympathetic pathways. Figure 3 illustrates the results of such an experiment. To the left of arrow 1, the sympa-
The changes in left ventricular pressure (LVSP) evoked by standard elevations of carotid sinus pressure (CSP) and by transections of sympathetic nerve fibers. At arrows 1 and 2, the paravertebral chains were sectioned at the inferior poles of the left and right stellate ganglia, respectively. At arrows 3 and 4, the rami communicantes of the left and right stellate ganglia, respectively, were transected. The strips at both ends of the figure were recorded at fast paper speed to show the left ventricular pressure contours.

thetic pathways were intact, except that the vagosympathetic trunks had been sectioned in the neck earlier in the experiment. The pressure was elevated twice in the isolated right carotid sinus, from 0 to 160 mm Hg, for 1 min each time. Reductions in LVSP of 69 and 75 mm Hg were evoked by these abrupt increments of intrasinusal pressure, from a control level of LVSP of 230 mm Hg.

At arrow 1, the left sympathetic chain was sectioned at the inferior pole of the stellate ganglion. This was attended by a rapid diminution in LVSP from a level of 230 mm Hg just prior to section to a value of 178 mm Hg shortly after section. A subsequent standard elevation of intrasinusal pressure (midway between arrows 1 and 2) evoked a maximum decrease in LVSP of 41 mm Hg.

At arrow 4, sectioning the communicating rami to the right stellate ganglion was begun. There followed a prompt and considerable reduction of LVSP to a level of 115 mm Hg, from a previous level of 160 mm Hg. Repeated rises of carotid sinus pressure (to the right of arrow 4) thereafter failed to evoke any detectable change in LVSP.

In the 10 experiments included in this series, the magnitudes of the alterations which followed interruption at any specific point in the cardiac sympathetic pathways were quite variable. In all cases, however, after both stellate ganglia had been decentralized by sectioning the sympathetic chains and cutting the communicating rami, elevations of intrasinusal pressure evoked no detectable alterations of LVSP. The composite data are summarized in Figures 4 and 5.

The changes in LVSP in the absence of carotid baroreceptor stimulation, which were produced by each interruption of the sympathetic pathways, are displayed in Figure 4. The mean value of LVSP for these 10 experiments prior to any nervous transection was 140.9 ± 13.1 (SE) mm Hg. At the completion of all transections, the mean level of LVSP was 68.4 ± 8.9 mm Hg. The per cent of this change induced by any given transection is a measure of the fraction of the tonic sympathetic influence mediated by that pathway. Section of the paravertebral chains just inferior to the right and left stellate ganglia accounted for 28.0 ± 5.3 and 42.0 ± 4.0%, re-
The mean changes in tonic sympathetic nervous activity evoked by cutting the right (R) and left (L) paravertebral chains at the inferior poles of the stellate ganglia (left half of figure) and by transecting the communicating rami to the right and left stellate ganglia (right half of figure). The total change represents the difference between the control left ventricular pressure and the pressure after complete decentralization of both stellate ganglia. The height of each bar represents the mean change for the 10 experiments in this series. In half the experiments, the right paravertebral chain and rami communicantes were sectioned before the left; in the other half, the sequence was reversed.

Subsequent completion of decentralization of the right and left stellate ganglia by sectioning their communicating rami accounted for 11.8 ± 3.5 and 19.9 ± 3.9%, respectively, of the total change in LVSP.

Prior to any sympathetic nervous transections, standard pressure elevations in the isolated carotid sinus induced a mean reduction of LVSP of 38.2 ± 5.9 (SE) mm Hg (from the control level of LVSP of 140.9 ± 13.1 mm Hg stated above). In each experiment, the per cent reduction in response evoked by baroreceptor stimulation was computed for each stage of denervation. The composite data for these 10 experiments are presented in Figure 5. Section of the sympathetic chain inferior to the right stellate ganglion decreased the response to baroreceptor stimulation by 21.8 ± 4.5% (SE). Transecting the left chain at the equivalent location diminished the response by 40.3 ± 3.9%. Completion of the decentralization of the right and left stellate ganglia further curtailed the responses to baroreceptor stimulation by 16.1 ± 3.4 and 21.8 ± 5.5%, respectively.

Cephalic Ischemia

The changes in response of the paced left ventricle to cephalic ischemia associated with interruptions in the sympathetic pathways are illustrated in Figure 6. At A, the brachiocephalic artery and the aorta just beyond the origin of the left subclavian artery were clamped for 90 sec. The first transient on the aortic pressure tracing indicates the time of clamping. Although the aorta was perfused via the left subclavian artery from a reservoir at a constant hydrostatic level, there was a pressure drop in the tubing from the reservoir to the aorta equal to the resistance times the flow. When flow was abruptly curtailed by brachiocephalic and aortic clamping, the pressure drop in the tubing diminished; hence, aortic arch pressure rose. A screw clamp on the tubing from the reservoir was immediately adjusted to return aortic...
The left ventricular response to cephalic ischemia in a representative experiment. Between the upward transients at A, B, and C in the aortic pressure tracing, cephalic ischemia was produced for precisely 90 sec by occluding the brachiocephalic artery and descending thoracic aorta. Between arrows RS, the right stellate ganglion was decentralized; between arrows LS, the left stellate ganglion was decentralized. The bottom half of the figure is a continuation of the top half. The short strip in the upper left-hand portion was recorded at a faster paper speed (100 mm/sec) to display the contour of the left ventricular pressure curve under control conditions.

Arch (and therefore coronary artery) pressures to the control level. After 90 sec of cephalic ischemia, this screw clamp was released at the same time that the clamps on the brachiocephalic artery and descending aorta were released. This was associated with a momentary rise in pressure, followed by a more prolonged fall. The pressure reduction is undoubtedly due to the greatly accelerated flow associated with reactive hyperemia. Similar transients are evident during the subsequent occlusions at B and C.

Under control conditions, LVSP was approximately 155 mm Hg. The rhythmic fluctuations of LVSP which occurred at a frequency of 10/min were associated with perceptible movements of the entire rib cage. At the inception of cephalic ischemia, there occurred a pronounced rise of LVSP to 210 mm Hg. The fluctuations in LVSP during the last 30 sec of cerebral ischemia were associated with gasping movements of the rib cage.

Immediately after restoration of the cerebral circulation, there was a marked decline in LVSP to a minimal value of 85 mm Hg, and an absence of any perceptible respiratory movements. Left ventricular systolic pressure thereafter climbed steadily, and regained the control level approximately 5 min after restoration of cerebral blood flow. Respiratory movements began 30 sec after cessation of cephalic ischemia, as manifested by the oscillations of LVSP. The amplitude of the respiratory fluctuations of LVSP during recovery from cephalic ischemia varied inversely as the respiratory frequency, in confirmation of a previous observation.

Between the first pair of arrows (RS), the right stellate ganglion was decentralized. This was accompanied by a reduction in LVSP from a maximum value of 165 mm Hg to a value of 140 mm Hg just prior to the second
period of cerebral ischemia (B). The changes in LVSP during and after cephalic ischemia closely resembled those observed with the first period (A) of ischemia.

The second pair of arrows (LS) indicates the time during which the left stellate ganglion was completely decentralized. During this denervation procedure, LVSP dropped precipitously from a maximum value of 140 mm Hg to attain a stable level of 35 mm Hg. A third period of cerebral ischemia (C) evoked no detectable change in LVSP. However, the dog displayed the same changes in respiratory activity as were evident earlier in the experiment, with deep gasping movements during the last half of the 90-sec period of occlusion.

A total of 6 such experiments were conducted. With the cervical vagosympathetic trunks sectioned, but with all other sympathetic pathways intact, LVSP increased by $81 \pm 15$ (se) mm Hg during cephalic ischemia, from a mean control level of LVSP of $138 \pm 18$ mm Hg. After decentralization of the first stellate ganglion, the mean response to cephalic ischemia was reduced by only 8.3%, which was not significant statistically. In 3 of these experiments, the left stellate ganglion was inactivated first; the subsequent response to cephalic ischemia was less by 20, 43, and 44%. In the other 3 experiments, the right stellate ganglion was inactivated first; the subsequent response to cephalic ischemia was less by 11% in 1 experiment, but greater by 18 and 50% in the other 2 experiments.

After the second stellate ganglion had been decentralized, cephalic ischemia had no perceptible effect upon LVSP in any of the 6 experiments. In all cases, cephalic ischemia elicited characteristic gasping movements involving all the ribs, indicating that the brain stem and spinal cord, at least to the lowest thoracic levels, were still active.

**Discussion**

**STELLATE GANGLION STIMULATION**

Numerous investigators had previously noted the prepotent influence of the right sympathetic nerves upon heart rate, and of the left sympathetic nerves upon left ventricular performance. However, because these earlier experiments were conducted upon animals with relatively intact cardiovascular systems, the over-all response of the heart to nervous stimulation actually represented the result of direct, reflex, and humoral influences. The substantial rise in arterial pressure evoked by left-sided stimulation undoubtedly evokes carotid sinus and aortic arch baroreceptor reflexes, which would tend to decrease heart rate. Thus, in some of the experiments reported by Randall and Rohse and by Freis et al., left-sided stimulation actually resulted in a diminution of heart rate. The experiments of Hosoda illustrate the critical role played by the baroreceptors in the over-all heart rate response. In his experiments, when the baroreceptor reflexes were intact, left-sided stimulation accelerated the heart rate by only 4 beats/min. After section of the carotid sinus nerves, the same stimulus evoked a rise in heart rate of 20 beats/min. Arterial baroreceptor activity would also reflexly modify ventricular performance. The occasional depression of ventricular contractility during cardiac sympathetic nervous stimulation reported by Randall, Priola, and Ulmer might be ascribable to the carotido-ventricular reflex, which would be evoked by any concomitant elevation of arterial pressure. In the present study, in which arterial pressure was held constant, depression of left ventricular contractility was never observed during stellate ganglion stimulation.

Within limits, the response of the left ventricular myocardium to moderate stimulation of the right and left sympathetic nerves was additive. With stronger stimuli, which increased LVSP to values more than 2.3 times the control level, the response to simultaneous stimulation became appreciably less than the sum of the individual responses to unilateral stimuli. Such limiting, or saturation, probably accounts for the failure of unilateral stellate ganglion decentralization, especially on the right side, to reduce significantly the response to cephalic ischemia (Fig. 6). Under control conditions, this potent reaction is su-

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perimposed on the tonic level of sympathetic activity, and the magnitude of the total ventricular response is evidently limited. The total number of sympathetic impulses reaching the left ventricle during cephalic ischemia is probably substantially less after right-sided stellate ganglion decentralization than when both stellate ganglia are intact. Yet, because of saturation, the total response to cephalic ischemia is not appreciably diminished after right-sided decentralization. In fact, the response may even be greater, because it originates from a lower control level of sympathetic activity.

SYMPATHETIC TONIC INFLUENCES ON THE LEFT VENTRICLE

For the entire series of 10 experiments summarized in Figure 4, the mean control LVSP was approximately twice as great as the mean level after decentralization of both stellate ganglia. Sympathetic influences over this range summate linearly. Therefore, the fractional change in LVSP induced by a specific interruption of the sympathetic nervous pathways (computed as a fraction of the total change, to yield the data presented in Figure 4) would then also express the fraction of the sympathetic influences which were abrogated. The data included in Figure 4 indicate, therefore, that approximately two thirds of the tonic sympathetic influences reaching the left ventricle under the conditions of these experiments traverse the sympathetic chains at the inferior poles of the stellate ganglia. It is likely that such impulses are travelling rostrally, rather than caudally. In 4 of these experiments, the sympathetic chains just inferior to the site of transection were stimulated intensely; no cardiac responses were detectable. Conversely, stimulation of the stellate ganglia just superior to the site of transection never failed to elicit a substantial response. The lower thoracic segments of the spinal cord were viable in these experiments, as evidenced by the rhythmic respiratory motions of the lower rib cage. Anzola and Rushmer also observed that stimulation of the inferior end of the severed sympathetic chain below the stellate ganglion evoked no significant change in heart rate or in ventricular pressures or dimensions. On the basis of their anatomical studies, Perman and Mizeres report that, in the dog, no nerve fibers pass to the heart from sympathetic ganglia below the stellates.

Several investigators have observed cardiac acceleration and augmentation in response to stimulation of sympathetic fibers below the stellate ganglia, down to T5 or T6, when the sympathetic chains have been intact. Evidently such impulses ascend the chains and pass through the stellate ganglia before reaching the heart. The remaining tonic sympathetic impulses which reach the left ventricle via the stellate ganglia must enter these ganglia through their communicating rami. These rami conduct approximately one third of the tonic impulses (Fig. 4, right).

Regardless of whether the tonic sympathetic impulses to the left ventricle enter the stellate ganglia from their rami communicantes or from below via the paravertebral chain, more effective impulses, on the average, come through the left than through the right stellate ganglion (Fig. 4). However, definite deviations from this rule have been observed in individual experiments. For example, in the experiment depicted in Figure 3, sectioning of the rami to the right stellate ganglion (arrow 4) produced a much greater reduction in LVSP than did transection of the rami to the left stellate ganglion (arrow 3). Randall, Priola, and Ulmer also noted considerable variations in the distribution of sympathetic cardiac fibers among different dogs.

It is questionable whether the tonic sympathetic activity observed in the present series of experiments represents a basal degree of sympathetic tone. The studies of Murphy and Wallace et al. upon resting, unanesthetized dogs indicate that the extent of tonic cardiac sympathetic activity ordinarily is negligible. In anesthetized animals subjected to a variety of experimental procedures, however, considerable tonic sympathetic activity may be manifest. It has been suggested by Glick and Braunwald that such increased cardiac sympathetic tone is probably
related to the surgical procedures employed rather than to anesthesia.

Baroreceptor Reflex

The efferent limb of the carotid-ventricular reflex includes both the sympathetic and the parasympathetic divisions of the autonomic nervous system. Since the cervical vagosympathetic trunks were sectioned in the present study, the reduction of LVSP evoked by carotid baroreceptor stimulation (Fig. 3) reflects a diminution of tonic sympathetic activity. The fractional change in response to carotid baroreceptor stimulation evoked by serial transections of sympathetic pathways (Fig. 5) were not significantly different statistically from the reductions in sympathetic tone evoked by these same interruptions of sympathetic pathways (Fig. 4). It may be deduced, therefore, that baroreceptor stimulation reduces cardiac sympathetic activity along any given pathway in proportion to the tonic sympathetic activity mediated by that pathway.

In the experiment displayed in Figure 3, after both stellate ganglia had been completely decentralized (to the right of arrow 4), baroreceptor stimulation failed to evoke any change in LVSP. The response was entirely abolished by this procedure in each of the 10 experiments in this series. Other investigators also have reported that the carotid-ventricular reflex is abrogated by bilateral stellate ganglionectomy. In the experiment illustrated in Figure 6, after decentralization of the right stellate ganglion, the response to cephalic ischemia (B) was similar to the control response (A). After the second stellate ganglion had been decentralized, then cerebral ischemia (C) evoked no detectable response. Similarly, in the 5 other experiments in this series, after both stellate ganglia had been decentralized, cephalic ischemia produced no detectable change in LVSP. It is probable, therefore, that all sympathetic impulses reaching the heart to evoke this response must traverse the stellate ganglia, although they do not necessarily synapse in these ganglia.

Fibers may arise in the brain stem and travel with the vagosympathetic trunks, which can exert a positive inotropic effect upon the ventricular myocardium or produce cardioacceleration. If vagal cardioaugmentor fibers do exist, they would have been interrupted in our experiments, since both vagi were sectioned in the neck. To test for the possibility of such cardioaugmentor fibers, in one additional experiment the vagi were not cut, but the efferent parasympathetic influences were blocked with atropine. In this experiment, after bilateral stellate ganglionectomy, cephalic ischemia evoked a definite increase in LVSP. This parallels the results of certain earlier studies, in which it was found that cephalic ischemia produced cardioacceleration after atropine, whereas it always evoked bradycardia prior to parasympathetic blockade. However, these experiments do not prove the existence of such hypothetical cardioaccelerator and cardioaugmentor fibers. It is well known that efferent parasympathetic stimulation characteristically elicits such responses after atropine. Acetylcholine is liberated at parasympathetic endings, and it has been shown that acetylcholine may release norepinephrine from cardiac tissue.

The present study demonstrates that stellate ganglion decentralization, combined with cervical vagosympathectomy, effectively blocks the efferent cardiac sympathetic pathways of the cephalic ischemia response and the carotid sinus baroreceptor reflex. Stellate
ganglionectomy and vagotomy also abolish the cardiac responses to increases in brachiocephalic arterial pressure, to epinephrine injections into the brachiocephalic artery, and to electrical stimulation of the septal nuclei of the brain. It has been demonstrated that bilateral excision of the sympathetic chains from the stellate ganglia down to T5 causes a relatively small reduction in the myocardial stores of norepinephrine. Nevertheless, it appears likely that such a procedure would effectively eliminate any reflex cardiac sympathetic influences, and probably would eliminate tonic cardiac sympathetic influences.

References


