Study of the Sympathetic Vasoconstrictor Nerves to the Vessels of the Dog Hind Limb

By David E. Donald, Ph.D., and David A. Ferguson

ABSTRACT

In anesthetized dogs, the vasoconstrictor nerves to the vessels of the hind limb left the spinal cord in the anterior spinal nerve roots from T-10 through L-4 levels. Maximal vasoconstrictor responses occurred on stimulation of the T-12, T-13, and L-1 roots; none occurred on stimulation of roots caudal to L-4. The nerves first entered the lumbar paravertebral chain at or above L-1; the last point of entry was at the L-4, L-5 level. No fibers left the lumbar chain at L-1, L-2, and L-3 levels. The first point of exit was at L-4, and nerves continued to leave as far distal as S-1, the most caudal point examined. The maximal response to stimulation of the lumbar chain was at the L-4, L-5 level. With a single exception, the responses to electric stimulation of the anterior spinal nerve roots, lumbar chain, or sympathetic ganglia were confined to the vessels of the ipsilateral limb. In 22 dogs with unilateral sympathectomy (L-2 through L-7), changes in hind-limb vascular resistance were induced reflexly or by electric stimulation of the anterior spinal roots. The results indicated that sympathetic control of the resistance vessels of the hind limb was still absent 77 days after sympathectomy.

ADDITIONAL KEY WORDS canine limb perfusion limb blood flow sympathetic outflow reflex vasoconstriction vasomotor nerves anterior spinal nerve roots peripheral resistance sympathectomy

I. FUNCTIONAL ANALYSIS OF THE ROUTES TAKEN BY SYMPATHETIC VASOCONSTRUCTOR NERVES FROM THE SPINAL CORD TO THE HIND LIMB VESSELS OF THE DOG

To investigate sympathetic nervous control of vascular resistance in actively contracting skeletal muscle in the dog, it is necessary to interrupt the sympathetic nerves to the vessels of the hind limb in a manner that permits study of the conscious exercising animal shortly after the vascular denervation. Essential to such a technique is a knowledge of the pathways taken by the sympathetic vasomotor fibers from the spinal cord to the hind limbs, particularly where they enter and leave the lumbar sympathetic chain. This information is also necessary to assess the degree to which the sympathectomy has interrupted neurogenic control of the peripheral vasculature. The precise information needed was not available in previous studies (1-5).

Methods

Studies were conducted in 22 dogs in which anesthesia was induced by sodium thiopental, 20 mg/kg, and α-chloralose, 60 mg/kg, and maintained by α-chloralose, 10 mg/kg/hour. Atropine was given in an initial dose of 0.2 mg/kg and repeated at intervals of 1 hour. Heparin was given in an initial dose of 3 mg/kg and at hourly intervals in a dose of 1.5 mg/kg. Gallamine triethiodide (Flaxedil) was given in a dose of 50 mg at intervals of 1 hour to prevent muscle movement. These drugs were given intravenously. The animals were artificially ventilated with oxygen at a rate of 15 cpm and a peak inspiratory pressure of 12 cm H₂O.

Exit of Vasoconstrictor Fiber from the Spinal Cord.—The spinal cord was exposed from the T-9 through the L-7 level. At each segmental level, the anterior spinal nerve roots were isolated and the intervening spinal cord was removed. The femoral arteries were exposed bilaterally and
ligated 2 cm distal to the inguinal ligament. A Mayo-Gibbon, 360°, single roller pump was used to perfuse each hind limb separately at constant flow with the dog's own blood through cannulas placed in the femoral arteries, cranial and caudal to the ligature. The cranial femoral and superficial circumflex iliac arteries were divided between ligatures. A heat exchanger in the perfusion system maintained the temperature of the blood at 38°C. The output of each pump was adjusted initially to obtain a perfusion pressure of 120 mm Hg, and after that the perfusion flow was not altered. Perfusion pressure was measured by a fine catheter introduced into the blood-flow line and advanced until its tip was just distal to the cannula through which blood was pumped into the hind limb. Central aortic pressure was measured by a catheter inserted into a brachial artery and advanced into the ascending aorta. Each catheter was attached to a strain gauge (Statham Model P23) whose output was led into an ultraviolet-light recorder to allow simultaneous observation of all three pressures.

At each segmental level of the cord, first one and then the other anterior spinal nerve root was stimulated electrically with monophasic rectangular pulses of 10 v, 15 cps, and 3-msec duration. An increase in limb perfusion pressure occurring within 3 seconds of the onset of stimulation was accepted as a valid vasomotor response. A constant infusion of fresh heparinized donor blood was used to sustain mean systemic blood pressure above 100 mm Hg throughout the experiment.

**Entrance of Vasoconstrictor Fibers into the Lumbar Paravertebral Chain.**—Midline laparotomy was performed, and the lumbar sympathetic chain was exposed bilaterally. Division of the phrenico-abdominal artery and vein, and mobilization of the pars lumbalis of the diaphragm allowed the exposure to be carried to the level of L-1. A cannula was inserted into the terminal portion of the aorta, just distal to the origin of the external iliac arteries, and the blood was directed into two separate pump-perfusion systems. Each hind limb was perfused separately through a cannula inserted into the distal portion of the external iliac artery. The deep circumflex iliac, internal iliac, median sacral, and deep femoral arteries were divided between ligatures to reduce collateral inflow into the perfused hind limb. Perfusion and central aortic pressures were measured as previously described in this section, the output from each pump having been adjusted initially to obtain a perfusion pressure of 120 mm Hg.

In two dogs of this group, both hind limbs were perfused from a common constant head of pressure, and the flow to each limb was measured by cannulating square-wave electromagnetic flow transducers (300 Series). The activity of the sympathetic vasoconstrictor nerves to the vessels of the hind limbs was altered reflexly by varying the pressure in both carotid sinuses which had been vascularly isolated by the Moissejeff technique (6). Ligation of the occipital artery at its origin from the common carotid artery ensured that only the baroreceptors of the carotid sinus were stimulated. Observations were made at steady intrasinus pressures of 30 and 200 mm Hg. Both vagi were sectioned in the neck. After a series of control observations, the right or the left lumbar sympathetic chain was divided at the level of L-1, just anterior to the most cephalad ganglion that could be exposed. The pressure changes in the carotid sinuses were then repeated.

The communicating rami of the most cephalad ganglion (usually opposite L-2) were severed next, and the pressure changes in the carotid sinuses were repeated. This procedure—section of the communicating rami and exposure of the carotid sinuses to static pressures of 30 and 200 mm Hg—was repeated with each succeeding ganglion until reflex vascular responses were no longer observed. Both the right and the left lumbar sympathetic chains were treated as previously described. During the procedure, a silk stitch was placed in the lumbar muscles adjacent to each ganglion to allow later identification of the ganglion with reference to the lumbar vertebrae.

Finally, the ability of each decentralized chain to transmit impulses was tested by electrically stimulating the chain (10 v, 15 cps, 3 msec) at its most cephalad exposed point.

One animal was prepared as previously described in this section, and the anterior spinal nerve roots were isolated from the level of L-3 to the sacrum. In addition, the abdomen was opened through an incision in the left flank, and the left lumbar paravertebral sympathetic chain was exposed from the L-2, L-3 level through the L-6 level. The anterior spinal nerve roots on either side were stimulated electrically at each segmental level (L-3 through L-7) before and after selected communicating rami connected to the left lumbar chain ganglia were cut.

**Exit of Vasoconstrictor Fibers from the Lumbar Sympathetic Chain.**—Each hind limb was perfused separately at constant flow, and perfusion and aortic pressures were measured as described previously in this section. The animals were eviscerated to allow easy access to each lumbar paravertebral chain. A spinal needle was inserted between C-1 and the base of the skull to permit the introduction of a fine polyethene catheter into

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the subdural space. Four milliliters of 10% procaine hydrochloride were infused through this catheter to produce a high spinal block. This procedure served to prevent reflex vasomotor effects and to obtain maximal dilatation of the vessels of the perfused hind limbs.

The right or the left lumbar sympathetic chain was stimulated electrically for periods of 30 seconds with monophasic rectangular pulses of 10 v, 15 cps, and 3-msec duration. An increase in perfusion pressure within 3 seconds of the onset of stimulation was accepted as a valid vasomotor response. The lumbar chain was stimulated first on the cranial side of the most cephalad ganglion that could be exposed. This point was usually beneath the diaphragm at the level of L-2. The lumbar chain was then crushed immediately distal to the ganglion, and the stimulation was repeated. Finally, the electrodes were applied to the communicating rami and the stimulation was repeated once more. Next, the ganglion immediately caudal to the first was exposed, and the electrodes were applied to the lumbar chain between the first and second ganglia. The sequence of stimuli previously described was carried out. This entire procedure was repeated at each succeeding caudal ganglion up to and including the first sacral ganglion. During the procedure, markers were placed at each point of stimulation, and on completion of the experiment, the points were identified with reference to the immediately adjacent vertebra.

The contralateral sympathetic chain was studied in a similar fashion.

**Results**

**Exit from Spinal Cord.**—In the seven animals studied, significant vasoconstrictor responses were obtained from the T-10 through the L-4 segmental level, with the major increases in perfusion pressure being observed on stimulation of T-12, T-13, and L-1 anterior spinal nerve roots of either side. Constrictor responses were not obtained below the L-4 segmental level even when stimulation was continued as far distal as the L-7 anterior spinal nerve roots. Individual responses are shown in Figure 1. The mean maximal increase in perfusion pressure was observed at the T-13 level for both the right and left sides and was 116 (± 38) and 122 (± 51) mm Hg, respectively. In one animal, stimulation of the left anterior root at L-3 resulted in an increase in perfusion pressure of 58 mm Hg in the left leg and of 19 mm Hg in the right leg. The rise in pressure began simultaneously on both sides. When stimulation was stopped, perfusion pressure decreased immediately on the left side, whereas on the right it was unchanged for 15 seconds and then decreased slowly. This pressure response in the right leg was not present after section of the right sciatic nerve. Apart from...
Vasodilatation in Perfused Hind Limb during Stimulation of Anterior Spinal Nerve Roots of the Dog

<table>
<thead>
<tr>
<th>Dog</th>
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<td></td>
<td></td>
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</tr>
<tr>
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<td>+7</td>
</tr>
<tr>
<td></td>
<td>Right</td>
<td>+13</td>
</tr>
</tbody>
</table>

*Values for dogs 1-5 show decrease in perfusion pressure (mm Hg) from control. Values for dogs 6 and 7 show increase in flow (ml/min) from control at constant aortic pressure; values shown are the maximal ones for each dog.

†Left nerve roots and left hind limb. ‡Right nerve roots and right hind limb.

In six of the seven animals, stimulation at the L-5, L-6, and L-7 levels resulted in a definite vasodilatation, which was often more pronounced on one side than the other (Table 1). This vasodilator response had the following features.

1. The pressure or flow did not return to the control level for several minutes after stimulation was stopped.
2. If a second stimulus was given 2 to 3 minutes after the first, the magnitude of the response was reduced. Ten to 15 minutes was required for full recovery.
3. The response was not inhibited by intravenous dosage with atropine sulphate, 0.2 mg/kg, with 1-(isopropylamino)-3-(1-naphthyloxy)-2-propanol hydrochloride (propranolol), 1 mg/kg, or with diphenhydramine hydrochloride (Benadryl), 2.5 mg/kg.
4. The response was present after removal of the paravertebral lumbar sympathetic chain from L-2 through L-7.
5. The response was not associated with visible movement of the hind limb muscles and could be obtained immediately after treatment with gallamine (50 to 100 mg iv).

Figure 2 shows reproductions of tracings obtained by stimulating the anterior spinal nerve roots at L-6 in dogs 1 and 7. These...
SYMPATHETIC VASOCONSTRICTOR OUTFLOWS IN DOG

responses were among the most pronounced in this group of dogs.

Entrance into the Lumbar Paravertebral Chain.—The individual data from seven dogs (Fig. 3) show that the magnitude of the reflex changes in limb perfusion pressure decreased progressively as the level at which the connecting rami were sectioned was moved toward the sacrum; there were no changes after section of the rami at the L-4, L-5 level. In each animal, the average of the responses obtained before section of the lumbar chain or connecting rami was taken as the control, and the subsequent responses were expressed as percent of this value. At least ten changes of pressure within the carotid sinus were used to obtain the control value, and each subsequent point represented the mean of the responses to four changes in sinus pressure.

A temporary reduction in the response of central aortic pressure was associated with the manipulations required to expose the point of section of the lumbar chain at L-1, but apart from this instance, the response of central aortic pressure to carotid sinus stimulation was relatively constant throughout each experiment. The mean changes in central aortic, right-leg, and left-leg perfusion pressures during the control period were, respectively, 59 (± 4), 72 (± 13), and 76 (± 13) mm Hg. In each animal, direct electric stimulation of the decentralized lumbar chains at the L-2 level resulted in an increase in limb perfusion pressure.

In the dog whose anterior spinal nerve roots were isolated, perfusion pressure increased 62 and 36 mm Hg on stimulation of the left anterior roots at L-3 and L-4, respectively. Stimulation of the right anterior roots gave increases in perfusion pressure of 54 and 30 mm Hg, respectively, at the L-3 and L-4 levels. No vasoconstrictor responses were obtained from stimulation of the L-5, L-6, and L-7 anterior roots on either side. The vasoconstrictor response obtained at L-3 and L-4 was confined to the ipsilateral limb, with no change in pressure in the contralateral limb. After section of the communicating rami to the left lumbar chain ganglion at the level of L-4, there was no response to stimulation of the left L-3 anterior nerve root, but an increase in perfusion pressure of 40 mm Hg resulted from stimulation of the left anterior root at L-4. The communicating rami to the next caudal left lumbar chain ganglion (L-4, L-5 level) were then sectioned. Stimulation of the left anterior root at L-4 now gave no response. The electrodes then were applied to the left lumbar chain at the L-2, L-3 level. Stimulation resulted in an increase in left-leg perfusion pressure of 94 mm Hg.

Exit from Lumbar Paravertebral Chain.—The changes in limb perfusion pressures in response to electric stimulation of the right and left lumbar sympathetic paravertebral chains at each segmental level are shown in Figure 4. In one dog (open square in Fig. 4), only the left chain was stimulated. Maximal responses were obtained at the L-4 and L-5
levels, after which the magnitude of the pressure change declined rapidly. However, pressor responses were still elicited at the sacral level. At the L-4 and L-5 levels, the mean increase in perfusion pressure in this group of seven dogs was 99 (sd 55) and 102 (sd 52) mm Hg in the right and the left leg, respectively.

A similar rise and fall was seen in the pattern of increase in limb perfusion pressure in response to stimulation of the communicating rami and of the lumbar chain after the chain had been crushed distal to the segmental ganglion. No increases in perfusion pressure were obtained at the L-2 and L-3 levels in any of the seven dogs. A small response was obtained at the L-4 level in four of the seven dogs, both from the lumbar chain and the communicating rami. The mean increase in perfusion pressure for both hind limbs at each segmental level was 2 (sd 4), 11 (sd 4), 24 (sd 9), 17 (sd 12), and 15 (sd 15) mm Hg at L-4, L-5, L-6, L-7, and S-1, respectively. In calculating these averages, the greater of the responses to the two modes of stimulation was used.

In this group of seven dogs, the vasoconstrictor response was confined to the vessels of the hind limb of the side stimulated, irrespective of whether the stimulus was applied to the lumbar chain, ganglia, or communicating rami.

Discussion
As in most studies involving the sympathetic nervous system, there was considerable variation from animal to animal (7), and while part of this variability in response undoubtedly had an anatomic basis (1), some at least resulted from injury to the nerves either during dissection or subsequent handling. However, we believe the data describe the
SYMPATHETIC VASOCONSTRICTOR OUTFLOWS IN DOG

Vasoconstrictor responses in dog hind limb from stimulation of sympathetic nerve fiber at each segmental level from T-9 through S-1. Averaged data from studies described under Methods I: Solid circles represent the exit of vasoconstrictor fiber from the spinal cord; X's show entrance of vasoconstrictor fibers into the paravertebral chain; and open circles and open triangles show exit of vasoconstrictor fibers from the lumbar sympathetic chain (open circle represents stimulation of lumbar chain intact distal to point of stimulation, open triangle represents stimulation of isolated communicating rami). Seven dogs were used in each of the three studies. Figure gives average route of sympathetic vasoconstrictor fibers from spinal cord to hind-limb resistance vessels.

Vasoconstrictor responses in dog hind limb from stimulation of sympathetic nerve fiber at each segmental level from T-9 through S-1. Averaged data from studies described under Methods I: Solid circles represent the exit of vasoconstrictor fiber from the spinal cord; X's show entrance of vasoconstrictor fibers into the paravertebral chain; and open circles and open triangles show exit of vasoconstrictor fibers from the lumbar sympathetic chain (open circle represents stimulation of lumbar chain intact distal to point of stimulation, open triangle represents stimulation of isolated communicating rami). Seven dogs were used in each of the three studies. Figure gives average route of sympathetic vasoconstrictor fibers from spinal cord to hind-limb resistance vessels.

general course of the sympathetic vasoconstrictor outflow to the hind limb of the dog. This is summarized in Figure 5, in which the data from each section of the study were averaged. The exit of vasoconstrictor fibers from the T-10 through the L-4 level of the spinal cord with the greatest response to stimulation at T-12, T-13, and L-1 is in agreement with the earlier studies in the dog by Bayliss and Bradford (8) and by Sheehan and Marrazzi in the cat (9) and in the monkey (10).

In the study of the regions of entry of vasoconstrictor fibers into the lumbar chain, we were concerned that all of the fibers leaving the spinal cord might not be activated by the carotid sinus reflex, since the response to electric stimulation of the lumbar chain usually exceeded that elicited from the sinus. However, the experiment in which stimulation of the anterior spinal nerve roots was combined with section of appropriate communicating rami confirmed the L-4, L-5 level as the last point of entry into the lumbar chain of the sympathetic vasoconstrictor outflow to the hind limb. This last experiment also suggested that all of the vasoconstrictor outflows to the limb were activated during maximal stimulation of the carotid sinus. The upper limits of entry were not defined because of technical difficulties in exposing the thoracic ganglia from the abdomen, but the combined data of Figure 5 suggest that the general pattern of entry into the paravertebral sympathetic chain reflects the pattern of exit from the spinal cord.
The first point of exit of vasoconstrictor fibers from the chain was at the L-4 level, and fibers continued to leave at successive caudal levels as far as S-1, the lowest level studied. Thus, the lumbar chain contained the greatest number of fibers at the L-4 and L-5 levels because at this point all of the fibers had entered the chain but relatively few had left. In an earlier study, Cloninger and Green (5) reported that stimulation of the lumbar chain at L-5 gave vasoconstrictor responses that exceeded those obtained at other segmental levels.

The data presented here are in accord with an earlier study by Randall and associates (4) in that vasoconstrictor responses were obtained from stimulation of the lumbar sympathetic chain as far caudal as the sacral level. A difference, however, is that in the present study the preganglionic inflow to the lumbar sympathetic trunk of vasoconstrictor fibers terminated at L-4, L-5, whereas these authors indicated inflows as far caudal as L-7. A possible explanation may lie in the relative magnitude of the vascular regions examined in the two studies, the central foot pad of the dog in the study by Randall and associates and the vascular bed of the external iliac artery in the present instance. The vessels of the foot pad may represent such a small proportion of the iliac artery distribution that we were unable to detect alterations in the caliber of the former.

The dilatation observed on stimulation of the L-5, L-6, and L-7 anterior spinal nerve roots was puzzling, and so far no reasonable explanation can be offered. Although movements of the muscles of the hind limb were not observed, the possibility that the vasodilatation was due to fine fasciculations deep in the muscles cannot be ruled out.

II. PERSISTENCE OF LOSS OF SYMPATHETIC CONTROL OF HIND-LIMB RESISTANCE VESSELS AFTER UNILATERAL LUMBAR SYMPATHECTOMY (L-2 THROUGH L-7) IN THE DOG

To study the extent to which blood flow in active muscles is influenced by the sympathetic nervous system, the blood flow to the hind limbs was measured during graded exercise in trained conscious dogs whose lumbar paravertebral chain had been removed on one side from the L-2 through the L-7 level. Because these animals were studied over a period of several weeks, it was necessary to determine if sympathetic vascular control was still absent in the operated limb 1 to 2 months after the surgical procedure. Studies in man (11-14) and dog (15) have suggested that there are functionally significant pathways that do not traverse the sympathetic chain and that are not interrupted by sympathetic trunk extirpation. The present study was undertaken to determine the persistence of sympathetic denervation because it permitted more rigorous testing of such denervation than was possible in the trained animals referred to above.

Methods II

In each of 22 dogs, the right or the left lumbar sympathetic chain was removed intact from the L-2 through the L-7 level by sterile surgical procedures. In three dogs, a bilateral lumbar sympathectomy was performed. The persistence of sympathetic denervation was studied 50 to 77 days after operation, either by inducing reflex changes in sympathetic vasomotor activity by changing pressure in the isolated carotid sinuses (16 dogs), or by electric stimulation of the anterior spinal nerve roots (9 dogs). Blood flow to the hind limb either was allowed to vary or was held constant. Anesthesia and other techniques employed and the measurements of flow and pressure were as described in Methods I.

Results

Reflexly Induced Changes in Vasomotor Nerve Activity.—The averaged data for each of the 16 dogs are given in Tables 2 and 3. Dogs studied both under conditions of varied and of constant flow are identified by the same number in each table. In the studies in which the dog perfused its own limbs (Table 2), blood flow in the denervated limb passively varied directly with reflexly induced changes in aortic blood pressure. Flow in the innervated limb either changed very little or
TABLE 2

Changes in Limb Blood Flow Induced by Hypertension and Hypotension in Isolated Carotid Sinus of the Dog

<table>
<thead>
<tr>
<th>Dog</th>
<th>No. of tests</th>
<th>Aortic pressure (mm Hg)</th>
<th>Blood flow (ml/min)</th>
<th>Aortic pressure (mm Hg)</th>
<th>Blood flow (ml/min)</th>
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<tbody>
<tr>
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</table>

Values represent the average change from control levels in limb flow and aortic blood pressure in each dog.

varied inversely to that seen in the sympathectomized limb and to the change in aortic pressure. These opposing changes in blood flow are illustrated in Figure 6. When the limbs were perfused at constant flow (Table 3), the changes in perfusion pressure in the innervated limbs were similar to those recorded from the aortic arch. In nine sympathectomized limbs (dogs 1, 3, 7, 8, 11-13, 15, and 16), there was a pressure response with small oscillations that were synchronous with the aortic pressure pulses. This slow pressure rise was absent after ligation of collateral arterial inflow to the leg. In three dogs (9, 10, and 14) the pressure response persisted after ligation but was reduced or absent after removal of the remaining lumbar sympathetic chain. An example of each class of response is shown in Figure 7.

Stimulation of the Anterior Spinal Nerve

![Figure 6](image-url)

**Figure 6**

Blood flow in normal and sympathectomized hind limb of dog during changes in carotid sinus pressure. **Left:** Pressure in carotid sinus was abruptly changed from 200 to 30 mm Hg. Blood flow in sympathectomized limb (S) passively varied directly with changes in aortic pressure (A), while flow in normal limb (N) varied inversely. **Right:** Carotid sinus pressure was reduced in steps from 160 to 30 mm Hg. Flow in normal limb was relatively unchanged, while flow in sympathectomized limb passively varied directly with changes in aortic pressure.
TABLE 3

Increase in Aortic Pressure and in Limb Pressure (Perfused at Constant Flow) when Pressure in Isolated Carotid Sinus of the Dog was Decreased from 200 to 30 mm Hg

<table>
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<tr>
<th>Dog</th>
<th>Days after operation</th>
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<th>No. of tests</th>
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<td>Left</td>
<td>11</td>
<td>105</td>
</tr>
<tr>
<td>14*</td>
<td>67</td>
<td>Right</td>
<td>9</td>
<td>63</td>
</tr>
<tr>
<td>15</td>
<td>67</td>
<td>Left</td>
<td>7</td>
<td>58</td>
</tr>
<tr>
<td>16</td>
<td>68</td>
<td>Right</td>
<td>9</td>
<td>85</td>
</tr>
</tbody>
</table>

Mean | 82       | 85       | 18       |
SD   | 30       | 30       | 10       |

*Response in sympathectomized limb still present after ligation of arterial collateral but reduced or absent after contralateral L-2 through L-7 acute sympathectomy.

Roots.—Stimulation of the anterior spinal nerve roots from the T-9 through the L-7 level failed to evoke any vasoconstrictor responses in the hind-limb vessels of the three dogs with bilateral lumbar sympathectomy.

Figure 8 shows the changes in perfusion...
pressure in the four dogs with unilateral lumbar sympathectomy in which the hind limbs were perfused at a constant flow. The changes in blood flow in the self-perfused normal and sympathectomized hind limbs during anterior root stimulation are shown in Figure 9. No constrictor responses were obtained on the sympathectomized side by stimulating the ipsilateral or contralateral anterior nerve roots.

Discussion

In this study the presence or absence of sympathetic vascular control was assessed initially by comparing, in the normal and the surgically sympathectomized hind limb, the vascular responses evoked by reflex excitation of the sympathetic vasoconstrictor nerves. The following were accepted as evidence of vasoconstriction: an increase in perfusion pressure in a limb perfused at constant flow, a decrease in flow in a limb perfused at constant pressure, or an unchanged flow in the presence of a sudden increase in perfusion pressure. With these criteria, sympathetic vasoconstriction was judged to be absent in 13 of the 16 dogs studied 50 to 77 days after surgical removal of the lumbar sympathetic chain from the L-2 through the L-7 level.

In the three dogs (9, 10, and 14) in which reflex excitation of the sympathetic vasoconstrictor nerves still elicited increases in perfusion pressure in the sympathectomized limb after interruption of collateral arterial inflow to the limb, the pressure changes were abolished or reduced by removing the contralateral lumbar sympathetic chain. However, when each dog perfused its own hind limbs, blood flow in the iliac artery of the sympathectomized limb passively varied directly

Changes in limb perfusion pressure in response to electric stimulation of isolated anterior spinal nerve roots from T-9 through L-7 levels. Dogs 1 and 2 had chronic right lumbar sympathectomy; dogs 3 and 4 had chronic left lumbar sympathectomy. Vasoconstrictor responses were not obtained in sympathectomized limbs, and stimulation of anterior nerve root on one side did not evoke response from contralateral limb vessels. Note slight vasodilatation on stimulation of anterior nerve roots in L-5 through L-7 levels in both normal and sympathectomized limbs.
with induced changes in aortic blood pressure and always varied inversely with the change in flow seen in the normal limb. In the course of the experiments on dog 9, there was a period when large spontaneous fluctuations in aortic blood pressure were observed. Blood flow in the sympathectomized limb passively varied directly with these changes in aortic pressure, but flow in the innervated limb was either unchanged or varied inversely. A reproduction of one of the records made during this phase is shown in Figure 10.

Thus, although it seemed that reinnervation from the remaining sympathetic chain was taking place in these three dogs, the data showed that this was not sufficiently complete to allow the same degree of autonomic control of the peripheral vasculature as was observed on the normally innervated side. Barcroft and Hamilton (16, 17) have suggested that the new pathways resulting from regeneration of the sympathetic fibers are functionally less efficient than the original.

In studies in which the dog perfused its own leg, the fact that flow in the sympathectomized leg passively varied directly with changes in aortic pressure did not preclude the possibility that these seemingly wholly passive changes were modified to some degree by the sympathetic nerves. The end point selected for study was the overall response of the hind-limb resistance vessels. Since no attempt was made to study particular regions of the hind limb—for example, skin, isolated muscles, or the foot pad—the data discussed thus far cannot eliminate the possibility that some limited region may not have been denervated by L-2 through L-7 sympathectomy. However, the different patterns of response in perfusion pressure and blood flow in the normal and the sympathectomized limb would indicate that if such a region did exist,
its vascular bed must be small in relation to that of the whole limb.

In Part I it was shown that the sympathetic vasoconstrictor nerves to the vessels of the dog hind limb left the spinal cord in the T-10 through the L-4 anterior spinal nerve roots. The most decisive proof of completeness of sympathectomy thus would seem to be afforded by those studies in which the anterior spinal nerve roots were stimulated from the T-9 through the L-7 level. These nine experiments offered no evidence of neurogenically mediated vasoconstriction in the sympathectomized leg from either ipsilateral or contralateral anterior nerve roots. Also, since the roots were stimulated as far caudal as L-7, the data do not support the contention that intermediate ganglia in the spinal nerves contribute to sympathetic peripheral vascular control.

Similarly, in a study in man, Barcroft and Hamilton (16, 17) found no evidence of any sympathetic nervous connection between the central nervous system and the hands for 6 months after sympathectomy, although a year or more later, vasmotor and sudomotor reflexes had returned in many of the hands. In a study of circulatory changes in the foot after lumbar sympathectomy, Lynn and Barcroft (18) noted that the return of vascular tone was very rapid but that blood flow in the foot was still twice that of the control 3 months after sympathectomy.

The available data indicated that sympathetic control of the resistance vessels of the hind limb was largely abolished after removal of the lumbar paravertebral chain from L-2 through L-7, and that neurogenic vascular control was still absent 3 months after sympathectomy. Thus, within this 3-month period, a study of the normal and the denervated leg in the same animal during exercise should provide evidence of the degree to which blood flow in active muscles was being affected by the sympathetic nervous system.

References

Study of the Sympathetic Vasoconstrictor Nerves to the Vessels of the Dog Hind Limb
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Circ Res. 1970;26:171-184
doi: 10.1161/01.RES.26.2.171

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