Responses of abdominal vascular capacitance to stimulation of splanchnic nerves

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KARIM, F., AND R. HAINSWORTH. Responses of abdominal vascular capacitance to stimulation of splanchnic nerves. Am. J. Physiol. 231(2): 434-440. 1976. — In chloralose-anesthetized dogs the abdominal circulation was vascularly isolated without opening the abdominal cavity. The region was perfused at constant flow through the aorta and drained at constant pressure from the inferior vena cava. Changes in resistance were calculated from changes in perfusion pressure and changes in capacitance were calculated by integrating changes in venous outflow. Stimulation of both splanchnic nerves at 20 Hz increased resistance by 135% and reduced capacitance by 7.2 ml kg⁻¹. The capacitance responses at 1 and 2 Hz (3.42 and 5.43 ml kg⁻¹) were 48 and 67% of the responses at 20 Hz. However, the resistance responses at 1 and 2 Hz (14 and 31% increase) were only 12 and 26% of the responses at 20 Hz. After occlusion of the splenic pedicle, capacitance responses were reduced by about 40%. Although changes in inferior vena cava pressure changed the volume of blood in the abdomen by 0.92 ml kg⁻¹ cmH₂O⁻¹, the responses to stimulation were relatively constant in any one animal at constant venous pressures between 5 and 15 cmH₂O.

The technique described by Brooksby and Donald (2), in which part of the splanchnic circulation of the dog was perfused at constant pressure.

The present investigation was undertaken to determine capacitance responses in the total abdominal circulation from stimulation of efferent sympathetic nerves with use of a preparation in which the abdominal circulation was vascularly isolated and arterial flow and venous pressure were held constant, thus ensuring that abdominal capacitance responses were due solely to active constriction of blood vessels.

METHODS

Dogs weighing 17.5–28 kg were anesthetized with chloralose (0.1 g/kg; Etablissement Kuhlman, Paris) infused through a catheter which had been passed, under local anesthesia (amethocaine hydrochloride, 2%) through a saphenous vein so that its tip lay in the inferior vena cava. The chloralose was dissolved to make a concentration of 1 g/100 ml in a solution of sodium chloride (0.9 g/100 ml). A state of light surgical anesthesia was maintained during the experiment by further infusions of chloralose (about 10 mg/kg every 15 min). The neck was opened in midline, the trachea was cannulated, and positive-pressure ventilation was started by means of a Starling Ideal pump, with 40% oxygen in nitrogen humidified at room temperature. The rate of the pump was 18 strokes/min and the stroke volume was approximately 17 ml/kg body wt. When the pleura was opened a resistance to expiration was inserted equivalent to 3 cmH₂O.

All structures immediately above the diaphragm, with the exception of the sympathetic nerves, were cut or tied. The thoracic wall was divided by cutting through the sternum and the 8th to 13th ribs on both sides. The dorsal spinal muscles were cut transversely and a strong nylon cord around the spine and the anterior muscles was mechanically tightened using a lever-and-ratchet system. Circulation through the spinal canal was prevented by packing the canal tightly with surgical gauze through a hole drilled in the first lumbar vertebra. The esophagus, the attachments of the pericardium to the diaphragm, and the phrenic and vagus nerves were tied and cut. The lowest four pairs of intercostal arteries were cut between ties close to the aorta.

Temporary bypasses were connected between the proximal ends of a carotid and a femoral artery and an external jugular and a femoral vein to allow some circulation to the abdomen during the cannulation proce-
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The animal was given suxamethonium chloride (0.5 mg/kg every 15 min) and heparin (500 IU/kg followed by 50 IU/kg every 30 min). The circuit (Fig. 1) was filled with dextran solution (Dextraven 150, Fisons Pharmaceuticals Ltd.) or a mixture of dextran and blood obtained from a second dog. The aortic cannulas were inserted and the abdomen was perfused for about 5 min before the perfusion was stopped and the inferior vena cava was cannulated. The time of each cannulation was less than 4 min, during which time abdominal aortic pressure remained above 40 mmHg. Blood from the abdomen was passed through a stainless steel cannula (11 mm ID) and drained into a reservoir from which it was pumped into an external jugular vein. Strong nylon cords were tightened at the proximal ends of the hindlimbs, leaving the area investigated to be that from the diaphragm down to the limb ties. The abdomen was not opened except in experiments in which the splenic pedicle was tied. The bypasses were clamped and the arterial perfusion pump was set before the sympathetic nerves were crushed so that abdominal aortic pressure was approximately the same as systemic arterial pressure. The level of the venous reservoir was set so that pressure in the inferior vena cava was approximately the same as it was before cannulation. Systemic arterial pressure was maintained by use of an arterial reservoir which had a constant pressure of air above the blood. The reservoir enabled large changes in abdominal vascular volume to occur with little change in systemic arterial blood pressure. The volume of blood in the circuit was about 200 ml plus the variable volume in the reservoirs (up to 1 liter).

Blood pressures were recorded using Statham (P23Gb) transducers connected to cannulas in the abdominal aorta (passed through a femoral artery), the inferior vena cava (passed through a femoral vein), and a brachial artery (systemic arterial pressure). The pressure signals were amplified and mean pressures were obtained using RC networks with time constants of 2 s incorporated in the amplifiers (S. E. Laboratories, Feltham, England). Blood flows were recorded using a Biotronex flowmeter with cannulating transducers in the aortic inflow and vena caval outflow. Zero flows were recorded at intervals during the experiment. The flowmeters were calibrated using the animals’ blood at the end of the experiment. Pressures and flows were recorded on photographic paper using a direct-writing ultraviolet light recorder (S. E. Laboratories).

The sympathetic trunk and splanchic nerves were crushed at the T12 level and pairs of stimulating electrodes were placed around all the nerves distal to the crushed area. The electrodes were connected to a Grass stimulator (model S4).

Arterial blood gases and pH were measured frequently during each experiment by use of standard glass electrode systems (20). Since the dogs were ventilated with 40% oxygen, Pao2 was always greater than 150 mmHg. Arterial Pco2 and pH were maintained at 35–40 mmHg and 7.30–7.40, respectively, by adjustment of the stroke of the respiratory pump and intravenous infusion of molar sodium bicarbonate.

In three dogs we used a different perfusion system (13): the blood from the venous reservoir (Fig. 1) was pumped through an oxygenator (Temptrol Pediatric, Bentley Laboratories Inc., Santa Ana, Calif.) and back into the abdominal aorta. Thus the circulation to the abdomen was entirely separate from that to the rest of the body. The pH, Pao2, and Pco2 of the blood leaving the oxygenator were kept within the normal limits defined above by adjustment of the rate of the oxygen flow and by infusion of sodium bicarbonate.

The results were analyzed using paired t tests as described by Snedecor and Cochran (24).

RESULTS

Evidence for vascular isolation of abdomen. In all experiments the effects on outflow of stopping the aortic perfusion pump were recorded. Mean abdominal aortic pressure decreased from 75 (SE ± 5.0) to 4.9 mmHg (SE ± 0.87). Mean outflow decreased from 1,465 ± 112 to 43 ± 8.3 ml min−1 in less than 2 min; i.e., flow decreased to 2.9% of its initial value, indicating the effectiveness of the vascular isolation.

In the three dogs with entirely separate abdominal circulation the dye, Evans blue, was injected into the abdominal circulation and its rate of appearance in the systemic circulation over 30 min was measured by use of a spectrophotometer (Unicrom; Pye Ltd., Cambridge, England). In two dogs no dye at all was detected during this time and in one dog a trace of dye appeared in the systemic circulation. This amount of dye indicated that there was an outflow of blood from the abdominal circulation of 1.6 ml min−1.

FIG. 1. Experimental preparation. Blood from descending thoracic aorta collected and pumped at constant flow into aorta above diaphragm. Blood from inferior vena cava collected in reservoir and returned to external jugular vein. SG, strain gauge; P, roller pump; F, electromagnetic-flowmeter cannulating probe; S, snare-nylon cord tightened with ratchet.
Responses of abdominal circulation to bilateral stimulation of splanchnic sympathetic nerves. In all 26 dogs the mean values and standard errors of the measured variables after the sympathetic nerves were crushed, but in the absence of stimulation, were: systemic arterial pressure, 116 ± 6.0 mmHg; abdominal aortic perfusion pressure, 75 ± 5.0 mmHg; inferior vena caval pressure, 9.3 ± 0.70 cmH₂O; abdominal aortic inflow, 1,414 ± 113 ml min⁻¹; inferior vena caval outflow, 1,465 ± 112 ml min⁻¹.

The sympathetic nerves on both sides were stimulated at 10–15 V with pulses of 2–4 ms duration and different frequencies. An example of records obtained during stimulation at 1, 2, and 5 Hz in one dog is shown in Fig. 2. Abdominal aortic pressure increased, outflow increased transiently, and inflow and vena caval pressure remained constant. The reduction in abdominal vascular capacitance was determined by integrating the change in outflow from its control value with respect to time by use of a planimeter. In all dogs, the change in outflow was complete in 55 s (SE ± 2.5).

Responses to stimulation at 10 Hz were determined in all 26 dogs. This resulted in a decrease in capacitance of 132 ± 14.4 ml or 5.75 ± 0.61 ml kg⁻¹ and an increase in arterial perfusion pressure of 83 ± 10.7 mmHg from a control value of 70 ± 5.0 mmHg. The perfusion pressure change represented an increase in vascular resistance of 127 ± 17.3%.

Eleven of the dogs showed evidence of "autoregulatory escape" (6, 8). In those dogs, when stimulated at 5 Hz, there was a peak increase in resistance at 30 ± 6.8 s (mean ± SE) from the start of stimulation, but the steady-state response at about 120 s was 77 ± 6.2% of the peak value. In the other 15 dogs perfusion pressure reached a peak and remained unchanged during stimulation.

In 14 of the dogs the responses of capacitance and resistance were determined to stimulation of the sympathetic nerves at frequencies from 1 to 20 Hz. The results, summarized in Table 1, show that capacitance responses, unlike resistance responses, were large at low frequencies of stimulation. This is shown particularly in Fig. 2, in which the response to stimulation at 5 Hz was no greater than that to stimulation at 2 Hz. Resistance responses, however, increased progressively with increasing frequency. The differences in the responses of resistance and capacitance at various frequencies are seen in Fig. 3. In each dog, the responses of resistance and capacitance at 20 Hz have been expressed as 100%, and the responses at other frequencies have been calculated as the percent of this response. At a stimulus frequency of 1 Hz the average capacitance response was nearly half the response at 20 Hz, whereas the corresponding resistance response was only about 12% of the response at 20 Hz. At 1, 2, and 5 Hz the relative responses of resistance and capacitance were significantly different.

Effects of clamping splenic pedicle. In six dogs, the abdomen was opened through the diafragm and the

**TABLE 1. Capacitance and resistance responses to stimulation of splanchnic nerves at different frequencies**

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Capacitance Responses, ml kg⁻¹</th>
<th>Resistance Responses, % increase*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 Hz</td>
<td>2 Hz</td>
</tr>
<tr>
<td>1</td>
<td>1.97</td>
<td>3.19</td>
</tr>
<tr>
<td>2</td>
<td>3.13</td>
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<tr>
<td>3</td>
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<td>1.54</td>
</tr>
<tr>
<td>4</td>
<td>3.74</td>
<td>5.74</td>
</tr>
<tr>
<td>5</td>
<td>1.04</td>
<td>1.93</td>
</tr>
<tr>
<td>6</td>
<td>4.52</td>
<td>6.04</td>
</tr>
<tr>
<td>7</td>
<td>3.96</td>
<td>5.28</td>
</tr>
<tr>
<td>8</td>
<td>2.04</td>
<td>3.75</td>
</tr>
<tr>
<td>9</td>
<td>0.50</td>
<td>0.73</td>
</tr>
<tr>
<td>10</td>
<td>3.41</td>
<td>4.30</td>
</tr>
<tr>
<td>11</td>
<td>4.24</td>
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<tr>
<td>13</td>
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<td>10.49</td>
</tr>
<tr>
<td>14</td>
<td>9.30</td>
<td>9.93</td>
</tr>
<tr>
<td>Mean</td>
<td>3.49</td>
<td>5.43</td>
</tr>
<tr>
<td>± SE</td>
<td>0.64</td>
<td>1.92</td>
</tr>
</tbody>
</table>

Values listed are the averages obtained from each dog. * In these experiments, the average aortic perfusion pressure in absence of stimulation was 70 mmHg (SE ± 7.0).
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FIG. 3. Capacitance and resistance responses at different frequencies. In each dog changes in capacitance and resistance produced by stimulation at 20 Hz were expressed as 100% and responses at other frequencies were calculated as percentages of these responses. Values shown are means ± 1 SE. Probabilities calculated for paired comparisons: * at both 1 and 2 Hz, P < 0.001. Absolute values of capacitance and resistance responses in these animals are given in Table 1.

The splenic pedicle was occluded with a Potts clamp (Thackray Ltd., London) to prevent the spleen from participating in the response to stimulation. The results from these experiments have been summarized in Table 2. Clamping the splenic pedicle did not change resistance in the absence of stimulation. Perfusion pressure (mean ± SE) was 62 ± 6.1 before, and 63 ± 6.6 mmHg after clamping. The resistance responses to stimulation were also not significantly changed after clamping. Stimulation at 10 Hz increased resistance by 154 ± 32% before, and 145 ± 30% after clamping. Capacitance responses, however, were less when the pedicle was clamped. Stimulation at 10 Hz decreased capacitance before clamping by 5.28 ± 1.0 ml kg⁻¹ and after clamping by 3.0 ± 0.5 ml kg⁻¹ (means ± SE). This represented a reduction in the response of 39 ± 4.7%. The reduction in the capacitance response caused by clamping the splenic pedicle was a result of exclusion of the spleen rather than damage to the splanchic nerves or deterioration of the preparation, because, in two dogs, when the splenic clamp was removed the responses returned near to their original levels. The average capacitance responses at 10 Hz, before applying and after releasing the clamp, were 4.9 and 5.1 ml kg⁻¹, respectively.

The results, listed in Table 2, also show that when the splenic pedicle is clamped the relationship between the capacitance responses at low and high stimulus frequencies is not significantly altered. In six tests in three dogs, before clamping the splenic pedicle the capacitance response at 2 Hz was 59 ± 3.7% of the response at 10 Hz, and after clamping, the response at 2 Hz was 51 ± 5.4% of the response at 10 Hz.

Changes in abdominal blood volume resulting from changes in inferior vena caval pressure. The changes in abdominal blood volume in six dogs in which inferior vena caval pressure was changed are shown in Fig. 4. The slope of the pressure-volume plots provides a measure of the compliance of the vascular bed. The average compliance was 0.92 ml kg⁻¹ cmH₂O⁻¹ (range, 0.60-1.48).

Capacitance responses from stimulation of sympathetic nerves at different venous pressures. In seven dogs the sympathetic nerves were stimulated at different inferior vena caval pressures. The results, plotted in Fig. 5, indicate that over the range of venous pressures

<table>
<thead>
<tr>
<th>Dog and Test No.</th>
<th>Capacitance Responses, ml kg⁻¹</th>
<th>Resistance Responses, % increase*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clamp off</td>
<td>Clamp on</td>
</tr>
<tr>
<td>5</td>
<td>3.48</td>
<td>2.15</td>
</tr>
<tr>
<td>14</td>
<td>2.21</td>
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<tr>
<td>24</td>
<td>8.22</td>
<td>2.97</td>
</tr>
<tr>
<td>25A</td>
<td>3.01</td>
<td>0.95</td>
</tr>
<tr>
<td>25R</td>
<td>1.89</td>
<td>1.64</td>
</tr>
<tr>
<td>26A</td>
<td>3.22</td>
<td>1.32</td>
</tr>
<tr>
<td>26B</td>
<td>3.00</td>
<td>1.74</td>
</tr>
<tr>
<td>26C</td>
<td>3.41</td>
<td>2.21</td>
</tr>
<tr>
<td>Mean</td>
<td>3.79</td>
<td>1.80</td>
</tr>
<tr>
<td>± SE</td>
<td>0.91</td>
<td>0.29</td>
</tr>
</tbody>
</table>

Results from nine tests in six dogs are given. In dogs 25 and 26, A refers to the first test of stimulation before and during splenic pedicle clamping, B and C refer to subsequent tests—after removing and after reapplying clamp. * In these experiments, the aortic perfusion pressure in absence of stimulation was 62 mmHg (SE ± 6.1) before, and 63 mmHg (SE ± 6.6) after clamping splenic pedicle.

FIG. 4. Changes in volume in abdominal circulation produced by changing pressure in inferior vena cava in 6 dogs. Responses are given relative to lowest IVC pressure used in each dog. Slopes of lines give compliance of region; average compliance was 0.92 ml kg⁻¹ cmH₂O⁻¹.
tion, the hematocrit returned to its previous value turned to its control level, during continued stimula-
only 1.1% (range, +0.6 to +2.2). When flow had re-
tained from a separate dog in which sympathetic nerves were stimu-
stimulation corresponded to an increase in hematocrit of
(normal change, +0.06%; range, -0.08 to +0.20%).

Hematocrit changes. In five dogs the electrical imped-
ance of the blood leaving the abdomen was recorded
using a 4,000-Hz AC current. Impedance is related to
hematocrit (5, 22). Impedance usually showed a small
transient increase during the rapid expulsion of blood
from the abdomen. However, the peak change for 10-Hz
stimulation corresponded to an increase in hematocrit of
only 1.1% (range, +0.6 to +2.2). When flow had re-
turned to its control level, during continued stimula-
tion, the hematocrit returned to its previous value
(mean change, +0.06%; range, -0.08 to +0.20%).

DISCUSSION

The area investigated extended from the diaphragm
to the site of the leg ties. Thus the tissues consisted
mainly of the abdominal and pelvic viscera, but also
some skin, muscle, and bone. The advantages of study-
ing the abdomen are that it is a large area and has a
relatively large proportion of its fluid volume within
blood vessels. It is unlike limb preparations, as studied
by Mellander (18), in which most of the volume is con-
tained outside blood vessels (21). There have been sev-
everal previous reports of capacitance responses in abdo-
ninal organs, for example, in the spleen (3, 10), the liver
(11, 12), and parts of the intestine (1, 6), but no previous
reports of responses of the entire abdominal vascular

normally used (5-14 cmH₂O), the volume expelled was
relatively independent of inferior vena caval pressure.
However, at very high venous pressures the responses
to stimulation were reduced, and in two dogs the re-
sponse was lower at low venous pressures. In most
experiments it was not possible to reduce venous pres-
ure below about 4 cmH₂O.

Several investigators have shown that absorption or
transudation of tissue fluid occurs when venous pres-
sure is changed (15, 16, 23), or when the activity of the
sympathetic nerves is increased (18, 21). In our experi-
ments, volume responses from stimulation of sympa-
thetic nerves were due almost entirely to a decrease in
the capacitance of blood vessels; since the responses
were rapid, the venous outflow returned to the original
level within 55 s of starting stimulation and the hemato-
crit never decreased during stimulation. The hematocrit
usually increased slightly during the phase of increased
outflow, indicating either that a greater number of red
cells were washed out (e.g., from the spleen) or that, as
outflow transiently increased (inflow constant), there
was a slight transient loss of intravascular fluid to the
tissues resulting perhaps from a transient increase in
capillary pressure. However, hematocrit changes were
very small (peak change at 10 Hz only about 1%), and
changes only occurred during the phase of increased
outflow.

Constant-flow perfusion, as used in the present experi-
ments, minimizes secondary metabolic vasomotor
changes which might contribute to the autoregulatory
escape described by Folkow et al. (6) and explain why
resistance responses in our experiments showed little or
no autoregulatory escape.

The sympathetic nerves were crushed and stimulated
immediately above the diaphragm, at the level of the
lowest thoracic vertebra. Most of the abdominal plex-
uses and ganglia are supplied with nerves leaving the
spinal cord above this level, although the lower ganglia
receive some of their innervation from below this level
(19). The lower ganglia supply mainly the hindlimbs
and the pelvic viscera. Because the spinal canal was
packed at L₁, the lower ganglia did not receive a nerve
supply from below this level. Stimulation of sympa-
thetic nerves above the diaphragm would not activate
the lower sympathetic nerves, and thus the response
obtained might be smaller than would have been ob-
tained if all the efferent sympathetic nerves were stimu-
ated.

The volume of the splanchnic circulation (22-kg dog)
is about 400 ml (4, 14, 17). On the assumption that
sympathetic nerve activity was present in the animals
in which the estimations were made, the volume in the
splanchnic circulation in our experiments was in the
absence of activity in the sympathetic nerves is about 150
ml greater than this, i.e., 550 ml; the volume in the entire abdominal circulation, which also includes the tissue of the abdominal wall, is somewhat greater. The response to stimulation (average at 10 Hz, 132 ml) represents a reduction in capacitance of about 20%.

The responses obtained were the result not only of constriction of capacitance vessels in the gut, but also of those in the spleen, abdominal wall, liver, kidneys, and suprarenal glands. We were able to confirm that, in the dog, the spleen has an important capacitance function. However, the method is not sufficiently sensitive to examine the effects of removal of other organs. Clearly the speed of onset of the response precludes any major effects from humoral agents from the kidney or suprarenal glands.

It is assumed that the responses obtained were due predominantly to constriction of veins. Arterial resistance vessels are unlikely to make a major contribution to the overall volume response for the following reasons: 1) less than 20% of the blood is contained in these vessels (9), and therefore even complete closure would not expel sufficient blood. 2) Since resistance is inversely proportional to the fourth power of the radius and capacitance is proportional to the square of the radius, it can be calculated that, if the entire length of the arteries constricted, the volume change of the arteries in our experiments at 10 Hz would be about 37 ml, i.e., 24% of the total response, and at 1 Hz it would be 8 ml, i.e., 10% of the total responses. Since it is likely that arterial constriction occurs mainly or entirely at the arterioles and the bulk of the arteries do not change in diameter appreciably, the total volume change would be much less than the maximum changes calculated. Furthermore, at low stimulus frequencies capacitance responses were large although resistance responses were small, whereas at high stimulus frequencies resistance increased steeply with little further capacitance change.

The difference in stimulus frequencies required to elicit capacitance and resistance responses agree with the conclusions obtained from experiments on the cat's hindquarters by Mellander (18), although Mellander did not perfuse the area at constant flow and therefore the effects of changes in passive distension and in capillary filtration were not eliminated.

It must be considered whether the difference between the responses of resistance and capacitance vessels at low frequencies can be explained in terms of the changes in the geometry of blood vessels. If the radius of blood vessels, in absence of stimulation, is taken as 1 unit, resistance responses are proportional to \((1/r^4) - 1\) where \(r\) is the new radius; capacitance responses are \(1 - r^2\). This implies that, as \(r\) becomes small, \(1 - r^2\) gradually approaches 1, but \((1/r^4) - 1\) rises very steeply. Therefore, for a large decrease in radius, the percentage change in resistance will be much greater than the percentage change in capacitance. So if these maximum changes in resistance and capacitance are taken as 100%, for small changes in radius the percentage of the maximum resistance response would be very much less than the corresponding percentage of the maximum capacitance response. However, in the present work, in which the resistance response at 20 Hz was +135%, the relative change in radius would be from 1 to 0.8. The resistance response at 1 Hz was 14% of the response at 20 Hz. If the radii of capacitance vessels decreased in the same proportion, the capacitance response at 1 Hz would theoretically be 23% of the response at 20 Hz. However, the actual capacitance response obtained was twice this, 48% of the response at 20 Hz. This indicates that, at low frequencies, the radii of vessels responsible for the capacitance responses decrease by a greater proportion than the radii of the vessels responsible for resistance responses.

The compliance of the abdominal circulation is high. Changes in venous pressure resulted in changes in a volume of about 0.92 ml kg\(^{-1}\) cmH\(_2\)O\(^{-1}\). There was a rapid and a slower phase of the volume response similar to the responses described by earlier workers (e.g., 18, 21). It is assumed that the rapid phase is due predominantly to changes in distension of blood vessels and the slow phase mainly due to changes in capillary filtration. We did not attempt to completely separate the two effects. It was usually not possible to lower the pressure in the inferior vena cava below about 4 cmH\(_2\)O by lowering the reservoir. This was presumably due to the weight of the viscera since the animal was on its back. We were not able, therefore, to determine whether collapse of the veins at very low pressures affected responses. However, as shown in Fig. 5, the responses to stimulation were relatively independent of vena caval pressure over a wide range. There is a suggestion from Fig. 5 that at the highest and the lowest pressures studied the responses may have been less. The high compliance of the region and capillary filtration effects are reasons why venous pressure must be held constant if active capacitance responses are to be distinguished.

The importance of capacitance responses of the abdominal circulation in circulatory control has yet to be firmly established. We have shown that a large volume of blood is expelled when the nerves are artificially stimulated. Clearly if similar changes occur in responses to a more physiological activation of sympathetic nerves they must make a major contribution to the control of blood pressure and flow. However, about 50% of the maximum capacitance response of the abdominal circulation was obtained at 1 Hz; so if abdominal capacitance vessels are to have a major role in circulatory control, the range of impulse frequencies in the sympathetic nerves must vary mainly between 0 and 2 Hz. Since most of the resistance responses occur at higher impulse frequencies, if large responses are to occur in both resistance and capacitance vessels, the impulse frequencies in the sympathetic nerves supplying the two types of vessel must be different. Since records from sympathetic efferent nerves cannot distinguish fibers innervating capacitance vessels from those innervating resistance vessels the only method by which to determine whether there is differential activity in nerves to resistance and capacitance vessels would be to determine the responses to physiological stimuli.

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