Dynamic Changes in Splanchnic Blood Flow and Blood Volume in Dogs during Activation of Sympathetic Nerves

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ABSTRACT
Arterial inflow to and venous outflow from the vascularly isolated splanchnic region were measured simultaneously by using cannulating electromagnetic square-wave flow transducers in 25 anesthetized dogs with intact abdomens. Change in splanchnic blood volume (SBV) was calculated as the instantaneous difference between inflow and outflow. Addition of the respective increases and decreases in SBV induced by changing carotid sinus pressure from the control value (mean, 136 mm Hg) to 200 or to 40 mm Hg gave an average total volume of 93 ml of blood (SD, ±21.7) that could be mobilized reflexly from the splanchnic circulation. Electrical stimulation (15 v, 2 cps, 3 msec duration) of the left thoracic splanchnic nerve for 2 to 10 minutes decreased SBV by 159 ml of blood (SD, ±84). These mean values represent 32 and 48%, respectively, of the calculated total SBV. In both situations the translocation of blood was rapid, 50% of the total change in splanchnic blood flow and SBV occurring within 30 seconds. During a 2-minute hemorrhage (7.2 ml/kg), a mean volume of 56 ml of blood (SD, ±16) was mobilized from the splanchnic region. This represented 54% of the total volume bled. Under these circumstances, blood moved out of the splanchnic circulation at a fairly constant rate.

KEY WORDS carotid sinus splanchnic nerves hemorrhage superior mesenteric artery stimulation viscera celiac artery

The relative magnitudes of splanchnic blood flow (1, 2) and blood volume (3-5) indicate that control of splanchnic vascular resistance is essential to the maintenance of systemic arterial blood pressure and that the uptake or release of blood by this region has important effects on the total cardiovascular system.

Kramer and Luft (6) recorded weight changes in the canine spleen during anoxia and estimated that splenic contraction could liberate a volume of blood equivalent to 16-20% of the normal blood volume. Barcroft and associates (7) reported that splenic contraction could increase circulating blood volume by 6-15% in the dog. Greenway et al. (8) found that maximal electrical stimulation of the hepatic nerves in the cat could result in expulsion of more than 50% of the blood in the liver, thus making it an important blood reservoir. Using qualitative instrumentation, Guntheroth and Mullins (9) found that in the dog the spleen, but not the liver, functions as a blood reservoir. Finally, the mesenteric circulation of the cat has been reported (10) to
decrease its capacity by 30–40% with maximal electrical stimulation. Little is known about the ability of the stomach or pancreas to mobilize blood.

A complete description of the splanchnic reservoir function, however, should include the total splanchnic circulation.

Combinations of indicator dilution and clearance techniques have allowed investigation of changes in total splanchnic blood volume in man and in animals (4, 5, 11–18). However, these methods do not permit continuous observations and require steady-state conditions of some duration. The present study was undertaken to examine (1) the amount of blood which could be taken up by or expressed from the total splanchnic circulation under various states of activity of the sympathetic adrenergic nerves and (2) the time course of the changes in inflow, outflow, and volume of blood in the splanchnic circulation. Three experimental situations were used: (1) alteration of sympathetic activity by change in pressure within the carotid sinus; (2) hemorrhage; and (3) electrical stimulation of the thoracic splanchnic nerve.

**Methods**

Male dogs averaging 14.6 kg body weight (sd ± 2.2) were used. The animals were fasted for 24 to 48 hours before the experiment. Anesthesia consisted of an initial dose of sodium thiopental (25 mg/kg, iv) and alpha-chloralose (50 mg/kg, iv). Supplemental doses of chloralose (10 mg/kg) maintained an even plane of anesthesia. Artificial respiration with oxygen was provided during the experiments at a frequency of 12 to 14 cycles/min, and a peak inspiratory pressure of 12 cm H₂O. When the chest was opened, inflation of the lungs was assisted by immersing the expiratory line in water to a depth of 3 to 5 cm.

The 8th, 11th, and 12th ribs were removed on the left side. The distal thoracic aorta was mobilized and ligated distal to the superior mesenteric artery. A cannulating (4-inch i.d.) electromagnetic flow transducer was inserted into the aorta upstream from the celiac artery, together with a 40-liter windkessel and a heat exchanger to maintain constant arterial blood pressure and temperature. A similar flow transducer was inserted into the inferior vena cava (IVC) (Fig. 1). The cannulations required a 4- to 5-minute interruption of splanchnic blood flow. Isolation of the splanchnic vascular bed was completed by doubly ligating the esophagus in the thorax at the diaphragm and dividing the phrenic vessels. A long catheter balloon was inserted up the femoral artery into the aorta as far as the distal aortic ligature. Inflation of this balloon completely occluded the abdominal aorta as far distally as the iliac bifurcation. A second balloon was passed up the femoral vein to occlude the abdominal vena cava at the hilus of the liver. Heparin was given in an initial dose of 3 mg/kg before cannulation and at hourly intervals in a dose of 1.5 mg/kg. In all of the experiments a recovery period of 30 minutes was allowed after completion of the surgical and cannulation procedures.

The perfusion circuit allowed for continuous monitoring of splanchnic arterial and venous blood flows in the closed abdomen. Bypass lines around both flow transducers allowed recording of zero flow before and after each intervention without interrupting flow to the gut. The signals from the flowmeters were electronically integrated to give continuous total forward flow over 3- or 6-second intervals. The reset time of the integrator from a full-scale deflection was 25 m sec. Volume changes in control and test situations were computed as the differences between arterial inflow and venous outflow over these periods.

Arterial and venous pressures were measured with Statham P23 strain gauges connected to needles inserted into the arterial and venous perfusion lines. All data were recorded on an ultraviolet oscillograph (Honeywell Visicorder).

Inflow and outflow of blood to the splanchnic region (SBF) and change in splanchnic blood...
volume (SBV) were observed under the conditions detailed below. In those experiments in which splanchnic arterial perfusion pressure was controlled through the windkessel system initially was set equal to the mean systemic arterial pressure of the dog, measured prior to cannulation of the aorta and IVC. The 140 ml of the dog’s own blood used to fill the windkessel system was replaced by an equal volume of dextran (average molecular weight 40,000).

On completion of the studies in each dog, the flow transducers were placed in series in a blood pump-reservoir circuit, primed with the dog’s own blood, for calibration over the range of experimental flows by timed volume collection. The extent of tissue perfused was delineated by injecting colored starch solution into the splanchnic arterial inflow line. The spleen, liver, pancreas, and stained intestines were removed and weighed en masse after removal of the gut contents.

The preparation was modified to study alterations of blood flow separately in the celiac and mesenteric arteries. The central end of the thoracic aorta was cannulated as before. The blood was passed through the heat exchanger and windkessel into the celiac and the superior mesenteric artery which were cannulated separately from within the aortic lumen. Flow transducers in each line permitted separate, simultaneous measurement of blood flow in each artery. The venous flowmeter was omitted, as were the balloons in the aorta and abdominal vena cava. Retrograde perfusion of the abdominal aorta was achieved through the femoral arteries, which were cannulated and connected to the aortic perfusion system.

Changes in Carotid Sinus Pressure.—Changes in SBF and SBV consequent to changes in pressure within the vascularly isolated carotid sinuses (Moissejeff technique) (19) were studied in 19 dogs. Both vagi were divided in the neck to eliminate reflexes originating from the aortic arch and cardiopulmonary system.

In the first group (five dogs), carotid sinus pressure was either increased or decreased from the control systemic blood pressure in a single step, was maintained at the selected value for 1 minute, and then was returned to the control value. Increments of 20 mm Hg were used until the carotid sinus had been exposed to a range of intrasinus pressures between 40 and 200 mm Hg.

In the second group (seven dogs), the carotid sinus pressure was decreased to a static pressure of 40 mm Hg and held there until stability of arterial and venous flows was achieved. The intrasinus pressure was then changed in a single step to a higher pressure, held at the selected pressure for 1 minute, and then decreased to 40 mm Hg. Increments of 20 mm Hg were used until the carotid sinus had been exposed to pressures from 40 to 200 mm Hg.

This last method also was used in a third group (seven dogs) in which changes in blood flow were measured separately in the celiac and in the superior mesenteric artery.

In all of the experiments, 15 minutes was allowed for recovery between tests, and during this time the carotid sinus was exposed to the systemic arterial blood pressure.

Hemorrhage.—Six dogs were bled through the catheterized costocervical trunk at the rate of 50 ml/min until 7.2 ml/kg had been withdrawn. The drawn blood was held out for 30 seconds and then rapidly reinfused through the cannulated external jugular vein. The windkessel was omitted in these experiments, and systemic arterial blood pressure was allowed to vary.

Electrical Stimulation of Left Thoracic Splanchnic Nerve.—In 19 dogs, both thoracic splanchnic nerves were cut (12 of these dogs had been used in the carotid sinus study). The left splanchnic nerve was mounted across two platinum electrodes and electrically stimulated by square-wave monophasic impulses (15 v, 3 msec duration, and frequencies between 1 and 20 cps; Grass Stimulator Model SD5). Generally, the period of stimulation was limited to 18 seconds. Longer periods of stimulation also were used. Stimulation at high frequency (15 cps) in seven dogs and at low frequency (1.5 to 2 cps) in another six dogs was maintained for 1 to 10 minutes.

Results

The preparation used in these studies proved to be stable, although characteristically the arterial inflow showed small, rhythmic fluctuations about a constant average flow. If no experimental disturbances were undertaken, blood flow would remain unchanged for 1 to 2 hours. In none of the experiments was hemorrhage or ascitic fluid found within the abdomen. The mean (±sd) SBF in the 25 dogs studied was 24.6 ± 7.4 ml kg⁻¹ min⁻¹. This represented a total flow of 361 ml/min or 28 ml min⁻¹ 100 g⁻¹ gut weight. The mean splanchnic weight was 1,230 ± 226 g or 85 ± 12.1 g/kg body weight. The systemic arterial blood pressure, controlled through the windkessel system, ranged from 105 to 150 mm Hg.
In the steady state prior to commencing an experiment, splanchnic arterial inflow was equal to venous outflow in 2 dogs, exceeded outflow in 11 dogs (mean difference, 8.7 ± 4.2 ml/min) and was less than outflow in 12 dogs (mean difference, 15.1 ± 9.6 ml/min). In any one dog, the difference between inflow and outflow was essentially constant throughout the experiment. Although small relative to the total flow (mean without regard to sign, 2.8%), if disregarded this difference would constitute a serious source of error in the computation of change in volume. The following correction, therefore, was used when inflow differed from outflow by more than 2%. Each was averaged over a 2- to 3-minute pretest period. Change in each flow was calculated as the difference between the averaged control flow and the recorded test flow. Changes in SBV were taken to be the differences between the calculated changes in arterial flow and the calculated changes in venous flow. Also, changes in SBV were calculated over periods not greater than 5 minutes and preferably 1 to 2 minutes.

**Effect of Change in Carotid Sinus Pressure.**—The time course of the changes in splanchnic arterial inflow, venous outflow, and SBV for two dogs is shown in Figure 2. These results, representative of the group, were selected since they most clearly illustrate the different characteristics of a pressor (left) and a depressor (right) response. The abrupt decrease in carotid sinus pressure was accompanied by a sharp decrease in inflow which reached a stable value in 12 seconds. Inflow and outflow again were equal and stable, although at lower values, 45 seconds from the onset of the change in pressure within the carotid sinus.

The expression of blood from the splanchnic circulation also occurred rapidly. Eighty percent of the total volume which left the splanchnic circulation did so in the first 24
Increases and decreases in splanchnic blood volume and flow in response to graded isolated changes in pressure in carotid sinus in five dogs. Each point indicates response to increase or decrease, in carotid sinus pressure, from constant (windkessel controlled) systemic arterial pressure. Control pressure for each dog is listed. Δ volume is total change with each change in carotid sinus pressure; Δ flow is change observed in the steady state.

When the carotid pressure was returned to the level of the control systemic pressure, there was an immediate increase in inflow, and 78% of the blood mobilized from the splanchnic circulation during the period of the carotid sinus hypotension was taken up by this region in the first 24 seconds after the carotid sinus pressure was returned to control levels.

When the carotid sinus pressure was increased abruptly from the resting systemic pressure, there was an immediate increase in SBF to a peak value in 12 seconds. In most cases arterial flows did not become stable but fluctuated for the duration of the increased pressure within the carotid sinus.

Outflow increased steadily throughout the period of carotid sinus hypertension but was not equal or nearly equal to inflow until the end of 1 minute. When the pressure in the carotid sinus was returned to the level of the control systemic pressure, expression of blood from the splanchnic circulation was rapid and, in the case illustrated, 61% of the volume taken up had been released by 30 seconds.

The results from the five dogs subjected to a sudden single increase or decrease in carotid sinus pressure from the resting systemic blood pressure are shown in Figure 3. The mean inflow in the control state throughout the experiments was $26.6 \pm 7.4\text{ ml kg}^{-1}\text{ min}^{-1}$, which represented a flow of 354 ml/min. Inflow exceeded outflow in three dogs and was less than outflow in two dogs. Neglecting sign, the individual mean inflow-outflow differences were $7.0 \pm 5.0$, $6.0 \pm 3.9$, $7.0 \pm 6.4$, $9.0 \pm 6.6$, and $12.0 \pm 11.8$ ml/min. The mean change in SBV when carotid sinus pressure was changed from control pressure to 40 or to 200 mm Hg was $44.8 \pm 16.5$ ml. The mean volume recovered by the splanchnic region within 2 minutes of return of carotid sinus pressure to control levels was $36.5 \pm 16.4$ ml or $79.4 \pm 19.5\%$ of the volume change induced initially. These differences were significant at the 0.01 level (Student’s t-test for paired observations) (20).

The curves relating the change in SBF and SBV to the pressure within the carotid sinus were sigmoidal. The steepest part of the flow and of the volume curve lay within $\pm 30$ mm Hg of the resting systemic blood pressure. In three of the five dogs, greater alteration of volume was seen with vasoconstriction than with vasodilatation, but in only two cases was this true of changes in blood flow. Addition of
be mobilized when the carotid sinus pressure was varied between 40 and 200 mm Hg. At a carotid sinus pressure of 40 mm Hg, SBF was decreased a mean of 115 ± 62 ml/min from the mean control flow; at 200 mm Hg, the mean increase in SBF above control was 114 ± 62 ml. Reflex vasoconstriction evoked the most stable responses in blood flow and volume. With reflex vasodilatation there was the same biphasic or oscillatory pattern in arterial blood flow as described.

Figure 4 shows the increases in SBV and SBF for the seven dogs in which isolated increments in carotid sinus pressure were made from a constant initial pressure of 40 mm Hg. In all instances, increase in carotid sinus pressure resulted in an initial transitory peak flow followed by a period of stable but decreased arterial inflow. At the control intrasinus pressure of 40 mm Hg, the mean inflow was 14.3 ml kg⁻¹ min⁻¹, which represented a blood flow of 209 ml/min. Inflow exceeded outflow in three dogs and was less than outflow in four dogs. Neglecting sign, the mean difference was 9.7 ± 6.6 ml/min. On increasing carotid sinus pressure from 40 to 200 mm Hg, a mean of 59 ± 14 ml was taken
up by the splanchnic bed; on returning sinus pressure to 40 mm Hg, a mean of 46 ± 12 ml was released, a percent recovery of 78 ± 14%. These differences were significant at the 0.05 level (Student's t-test for paired observations). The curve relating change in SBV to carotid sinus pressure was sigmoidal, with the steepest part lying between intrasinus pressures of 100 and 180 mm Hg. At an intrasinus pressure of 200 mm Hg, mean increase in SBF was 143 ml/min.

Measurements of blood flow separately in the celiac and superior mesenteric arteries revealed that the alterations of blood flow were similar in both vessels during a change of pressure within the carotid sinus (Fig. 5). During the reflexly induced vasodilatation, blood flow in each artery showed an initial increase which decreased to reach a steady state in 30 seconds. With an intrasinus pressure of 200 mm Hg, the mean maximal increase in blood flow was 81 ± 25 ml/min in the celiac artery and 69 ± 13 ml/min in the superior mesenteric artery, representing increases of 60 and 66%, respectively, from the mean control flows at an intrasinus pressure of 40 mm Hg (135 ± 58 ml/min in the celiac artery and 103 ± 68 ml/min in the superior mesenteric artery).

Effect of Hemorrhage.—A total of 90 to 130 ml of blood was removed over 2 minutes from each of six dogs. The mean control blood flow was 29.3 ± 9.2 ml kg⁻¹ min⁻¹ which represented a flow of 431 ml/min. The mean splanchnic weight was 1,176 ± 161 g. Inflow exceeded outflow in two dogs and was less than outflow in four dogs. Disregarding sign, the mean inflow-outflow difference was 16.3 ± 12.1 ml/min. The mean volume of blood expressed from the splanchnic circulation during hemorrhage was 56 ± 16 ml and a mean of 54 ± 18 ml was taken up in the first 2 minutes after the blood was reinfused. The difference between the volumes released and taken up was not significant at the 0.05 level (Student's t-test for paired observations). Systemic arterial
Changes in splanchnic blood volume and flow with stimulation of splanchnic nerve

Pressure was well maintained throughout the period of bleeding (mean decrease, 9.8 ± 3.3 mm Hg). Systemic arterial flow decreased by a mean of 92 ± 28 ml/min or 21% of the control flow. There was no obvious relationship between the amount of blood expressed and the initial level of arterial flow or the extent to which inflow was decreased during hemorrhage. The time courses of events shown in Figure 6 for two of the dogs are representative of the group in that blood was released from the splanchnic circulation at a fairly constant rate over the whole course of the hemorrhage; following reinfusion, uptake of blood by the splanchnic bed initially was rapid.

Effect of Electrical Stimulation of Left Thoracic Splanchnic Nerve.—In every instance, supramaximal electrical stimulation of the left thoracic splanchnic nerve resulted in a rapid decrease in inflow and a brief but generally pronounced augmentation in outflow. The changes in blood flow were rapid; by 12 seconds from the onset of stimulation, arterial blood flow had stabilized at a new decreased level and venous flow had reached its peak value. The magnitude of these changes was related to the frequency of

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**FIGURE 8**

Relationship of time course of change in venous flow to total volume of blood mobilized from splanchnic circulation during electrical stimulation of the left thoracic splanchnic nerve at 15 cps. Left: Unusually rapid decrease in outflow with virtual cessation by 42 seconds; 7.5 ml/kg of blood or 33% of the calculated volume was mobilized. Right: Outflow decreased more slowly; by 42 seconds, 16.2 ml/kg of blood or 71% of the calculated volume had been expressed. In the first example, volume was thought to be subnormal prior to stimulation. This would explain the small volume of blood mobilized and the increase in inflow without change in outflow observed toward end of period of stimulation.

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stimulation but varied considerably from dog to dog. The relationships of the frequency of electrical stimulation to the decrease in total inflow and to change in SBV are shown in Figure 7. The curves representing change in blood flow in the celiac and in the superior mesenteric artery are not shown but were similar in contour. The duration of electrical stimulation was limited to 18 seconds, since arterial flows had become stable within this period. The brief period of stimulation also ensured that the responses were not influenced by circulating catecholamines liberated into the bloodstream from sympathetic nerve endings or, in the case of the separately perfused celiac and superior mesenteric arteries, from the adrenal glands. In addition, it avoided the long period of decreased SBF which followed prolonged stimulation at high frequencies. In this group, changes in SBV were not followed in the poststimulation period, since the induced changes in blood flow were large and limitation of the period of measurement to 18 seconds decreased the magnitude of possible error to less than 10%. The mean decrease of 33% in initial blood flow and change in SBV of 50 ml observed when carotid sinus pressure was decreased from control resting levels to 40 mm Hg were approximately similar to the effects of electrical stimulation at 2 and 4 cps, respectively.

In 13 dogs, electrical stimulation of the left thoracic splanchnic nerve was maintained for 2-10 minutes. This ensured that arterial and venous flows came into equilibrium, and thus a value could be obtained for the maximal volume of blood that could be expressed at the particular frequency. At 15 cps the mean volume expelled was 15.0 ± 2.4 ml/kg; at only 2 cps, 72% of this volume was mobilized.

Examination of the time course of the changes in splanchnic arterial and venous blood flows showed that the amount of blood expressed depended on the decrease in inflow, the initial increase in outflow, and the rate of the later decrease in outflow. At 15 cps, inflow generally was decreased to near zero in 12 to 15 seconds, and the amount of blood expressed depended mainly on the magnitude of the initial rapid increase in outflow and on the rate at which this subsequently decreased. An example is shown in Figure 8.

Three examples of results with the lower frequencies of stimulation are shown in Figure 9 and further illustrate the different patterns of change in SBF and SBV.

TIME COURSE OF CHANGES IN ARTERIAL AND VENOUS FLOW AND TOTAL VOLUME OF BLOOD EXPULSED DURING CONTINUED LOW-FREQUENCY (1.5-2 cps) ELECTRICAL STIMULATION OF LEFT THORACIC SPLANCHNIC NERVE (15 V, 3 msec). TOTAL CALCULATED VOLUME OF BLOOD MOBILIZED IS SHOWN FOR EACH CASE, COMPUTED FROM START OF STIMULATION UNTIL ARTERIAL AND VENOUS FLOWS WERE EQUAL OR THEIR DIFFERENCE WAS THE SAME AS IN CONTROL SITUATION.
Discussion

Measurement of change in blood volume from the summed difference in inflow and outflow demands complete vascular isolation of the region under study, absolute accuracy of flow measurements, and absence of exchange of fluid between intravascular and extravascular compartments during the period of observation. These criteria were only partly met in the present study. Vascular isolation of the splanchnic region was estimated to be 95% complete, and the difference in simultaneous measurement by the two transducers of a constant flow varied from 0 to 2%. Zero flow was recorded before and after each observation, and the period of observation was limited for the most part to 3 minutes and preferably to 1 minute. Since the outflow-inflow difference was constant in any one experiment, a correction was made for this. Recovery values for blood mobilized were 95% in the hemorrhage study and 78 and 79% in the carotid sinus studies. The most confidence could be placed in the results when the changes in flow were large and the period of observation was approximately 30 seconds.

The mean SBF of 23.8 ml kg^{-1}min^{-1} agreed with values of 22.2 and 25.5 ml kg^{-1}min^{-1} obtained by Chien (21) and by Smythe and associates (18), respectively, in intact dogs anesthetized with sodium pentobarbital; however, it was 26% lower than the value 37.5 ml kg^{-1}min^{-1} reported by Shoemaker (17) in unanesthetized dogs. The average value for blood flow in the superior mesenteric artery obtained in the present study was 12.3 ml kg^{-1}min^{-1}. Measurements in resting unanesthetized dogs by Fronek and Stahlgren (22) indicated an average blood flow of 14.6 ml kg^{-1}min^{-1} in this vessel. Thus, the changes in SBF and SBV observed in this study may underestimate those occurring in the unanesthetized animal. In addition, there is the possibility of adverse effect on splanchnic vascular reactivity of total interruption of arterial blood flow distal to the superior mesenteric artery. In the two studies in which alteration of pressure within the carotid sinus was used to evoke a reflex vasodilatation or vasoconstriction, the mean maximal volumes of blood mobilized from the splanchnic circulation were 3.7 and 7.1 ml/kg. Using the values reported by Grim (1) and Bradley (5), calculated total SBV was 22.7 ml/kg. The average volume of blood mobilized in the carotid sinus studies represented 16 and 32%, respectively, of this calculated total volume.

When electrical stimulation of the left splanchnic nerve was continued until arterial and venous flows equilibrated at the new level, the mean volume of blood expressed was 329 ml at 15 cps and 159 ml at 2 cps. These volumes represented 66 ± 29% and 48 ± 22% of the calculated total SBV. On three occasions at 15 cps, a value of 90% was obtained; it does not seem likely that even supramaximal long-continued nerve stimulation could so markedly decrease the vascular volume. With near-zero arterial inflow, capillary pressure would be greatly decreased and water would pass from tissues into the bloodstream. However, the changes in SBV calculated for the first 1–2 minutes of stimulation probably dominantly reflect change in intravascular volume.

As important as the quantity of blood made available was the speed at which alterations in volume occurred. Of the total volume of blood mobilized in response to a reflexly induced vasoconstriction, an average of 53% was expressed in 18 seconds and the changes in volume were relatively complete by 60 seconds. During continued stimulation of the left splanchnic nerve, approximately 40% of the total volume was expelled in a period of 18 seconds.

An idea of the intensity of the sympathetic adrenergic activity involved in the naturally evoked response was obtained by comparison with the response to electrical stimulation of the splanchnic nerve (23). In regard to splanchnic arterial inflow, the maximal response would be complete arrest of inflow. Whether or not this could be obtained through electrical stimulation seemed to depend to some extent on the absolute magnitude of blood flow; cessation of inflow was not seen when SBF was 500 ml/min (33 ml
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kg⁻¹min⁻¹) or more, even with stimulation frequencies of 20 cps. Complete arrest of arterial inflow did occur on occasion, in one animal at a frequency of 5 cps. Folkow and associates (10) reported maximal decrease in arterial inflow in cat mesenteric circulation at stimulation frequencies of 4 and 6 cps. Greenway and associates (24) and Celander (25) noted maximal decrease in splenic arterial inflow at a frequency of 3 cps. Greenway and associates (8) reported that in the cat the maximal response of hepatic artery and portal vein resistance usually was reached at 10 cps. The averaged data from the 19 dogs in this study showed that, with stimulation frequencies of 10 cps and above, there was an 80% decrease in arterial inflow.

The location on the appropriate stimulus response curve obtained during electrical excitation of the left thoracic splanchnic nerve (Fig. 7) was determined for the average maximal reflexly evoked changes in SBF and SBV. This comparison indicated that, in the present studies, the maximal reflex activity evoked in the splanchnic vascular nerves via the carotid sinus baroreceptors was the equivalent of electrical excitation at a frequency between 2 and 4 cps.

Lofving (26) commented on the wide differences in vascular reactivity of the skin, skeletal muscle, intestine, and kidney in cats in response to alteration of carotid baroreceptor activity. Generally, reflex vasoconstriction was pronounced in skeletal muscle, less intense in intestinal and cutaneous vessels, and present only to a small degree in kidney. Likewise, the present study indicated that the maximal vasoconstrictive response of the splanchnic circulation evoked reflexly through the carotid sinus could be equated to a low frequency of electrical stimulation. In a recent study of the influence of the carotid baroreceptor on different components of the cardiovascular system, Breder and Webb-Peploe (27) equated maximal splenic responses to an increase in splenic nerve traffic of 1 to 4 cps, whereas maximal responses of the limb resistance vessels corresponded to an increase in lumbar sympathetic traffic of 6 to 10 cps.

A mean volume of 3.9 ± 0.9 ml/kg of blood was mobilized from the splanchnic circulation during the hemorrhage. This represented 17% of the calculated total SBV and 54% of the total volume bled. Although minor fluctuations were sometimes observed in the rate at which blood was mobilized, there was no sudden large release of blood from the splanchnic circulation in the first moment of the hemorrhage. This was in sharp contrast to the findings with changes in carotid sinus pressure or electrical stimulation of the splanchnic nerve. A possible explanation might be that the stimulus to the sino-aortic baroreceptor system during hemorrhage occurred in small increments rather than in a single, large, step input. At the end of the hemorrhage, systemic arterial pressure had decreased on the average by 10 mm Hg, and the maximal decrease was 15 mm Hg. Figure 2 shows that, when the pressure in the carotid sinus was abruptly decreased by these amounts, the mean volume of blood mobilized was 16 ± 8 ml and 23 ± 11 ml, respectively. These values are significantly different at the 0.01 level from the volume mobilized during hemorrhage. However, it is possible that the activity of receptors in the atria or at the veno-atrial junctions was affected by the hemorrhage and evoked reflex constriction of mesenteric veins and of the spleen and liver (28). The volume of blood expressed from the splanchnic circulation then would represent the summed response to stimulation of both high- and low-pressure baroreceptor systems.

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