ABSTRACT

Human Splanchnic and Forearm Vasoconstrictor Responses to Reductions of Right Atrial and Aortic Pressures

By John M. Johnson, Loring B. Rowell, Manfred Niederberger, and Martin M. Eisman

Regional vascular responses to gradual reductions in right atrial pressure and aortic pressure were investigated in nine men. In each study, lower body negative pressure was applied in a ramp of −1 mm Hg/min for 40-50 minutes. During the range from control to −20 mm Hg, right atrial pressure (4 studies) fell from 4.2 mm Hg to −0.6 mm Hg; heart rate was slightly reduced (2 beats/min), and aortic mean pressure and pulse pressure (6 studies) were unchanged. The maximal rate of rise of aortic pressure showed no consistent trends. Forearm blood flow (30 studies) fell with the onset of lower body negative pressure and reached 67% of the control value by −20 mm Hg. Splanchnic blood flow (14 studies) was significantly reduced by −7 mm Hg and fell to 89% of control by −20 mm Hg. During the range from −20 to −50 mm Hg, right atrial pressure continued to fall. Aortic mean pressure fell slightly or was unchanged in four subjects and fell dramatically at −35 mm Hg in two subjects. Aortic pulse pressure began to fall at about −20 mm Hg and fell linearly thereafter. Heart rate paralleled aortic pulse pressure (r = −0.86 to −0.93). Forearm blood flow fell to 55% and splanchnic blood flow fell to 65% of control at −50 mm Hg. Thus, significant vasoconstriction occurred without measurable change in arterial blood pressure. We concluded that low-pressure baroreceptors, presumably in the cardiopulmonary region, initiate splanchnic and forearm vasoconstriction with more pronounced vasoconstriction occurring in the forearm.

KEY WORDS peripheral circulation heart rate splanchnic blood flow
blood pressure regulation forearm blood flow low-pressure baroreceptors

A major cardiovascular adjustment to moderate hemorrhage is increased sympathetic outflow to the heart and various vascular beds. Traditionally this adjustment has been associated with carotid sinus and aortic baroreceptors (1) which clearly play important roles in regulating arterial blood pressure. More recently, however, stretch receptors in the cardiopulmonary region (low-pressure baroreceptors) have been implicated in the mediation of reflex responses to hemorrhage in dogs, cats, and rabbits (2-4). In humans, simulation of hemorrhage by mild degrees of lower body negative pressure can evoke marked forearm vasoconstriction without significant changes in heart rate, aortic mean pressure, aortic pulse pressure, or maximal rate of rise of aortic blood pressure (dP/dt max) (5). It appears that reduced pressures in the cardiopulmonary region accompanying lower body negative pressure must be the stimulus for the reflex vasoconstriction. Previous studies in man (6, 7) have implicated low-pressure baroreceptors in the reflex release of forearm vasoconstrictor tone accompanying increases in thoracic blood volume induced by postural or respiratory maneuvers.

We attempted to determine whether the vasoconstriction seen in forearms during a mild degree of lower body negative pressure, which was insufficient to measurably affect arterial blood pressure, was evident in other vascular beds. Earlier work in this laboratory on the splanchnic vascular responses to large (−50 mm Hg) step changes in lower body negative pressure has shown that, although forearm blood flow always falls immediately with the drop in right atrial pressure, the response of splanchnic blood flow is more variable. Increments in splanchnic vascular resistance appear to be more closely associated with the fall in aortic pulse pressure while aortic mean pressure is constant than they are with the fall in right atrial pressure. These results suggest that the splanchnic vascular bed is less sensitive to reflexes arising from the cardiopulmonary region than it is to those arising from arterial baroreceptors (8). Thus, we attempted to describe the relative responses of
splanchnic and forearm vasculatures to gradual stimulation of the cardiopulmonary receptors. We used a slow ramp of lower body negative pressure that yielded a gradual decline in right atrial pressure with stable arterial blood pressure followed by a period in which both arterial blood pressure and right atrial pressure were reduced. This approach also gave information concerning threshold characteristics of these two major regions.

**Methods**

The subjects were nine normal healthy men (20–42 years old). After institutional review of the project, each subject was given a thorough physical examination and acquainted with all procedures before informed consent was obtained.

Each subject was subjected to 1 or 2 trial runs of the experiment without catheters. These trials consisted of measuring forearm blood flow and heart rate throughout a ramp of lower body negative pressure applied at a rate of −1 mm Hg/min. End points for the ramps varied between −40 and −50 mm Hg, and they were usually followed by 5–8 minutes of lower body negative pressure sustained at the final level of the ramp. In ten studies, suction was returned to control via 5-minute steps to −20 mm Hg. and then to −10 mm Hg. A total of 15 trial runs was carried out on the nine subjects; Table 1 specifies which procedures were carried out on each subject.

Aortic pulse pressure, aortic mean pressure, splanchnic blood flow, forearm blood flow, and heart rate were measured in six subjects by procedures similar to those used in the trial runs. Right atrial pressure was measured at the same time in four of these subjects. In addition, splanchnic blood flow was measured in two other subjects without arterial catheterization. Duplicate experiments to measure splanchnic blood flow changes were performed on six of the subjects; measurements of arterial or central venous pressures were not repeated. In three studies, skin temperature was controlled at a fixed, neutral level to eliminate any possible cutaneous vasoconstriction due to the cooling effect of air flowing through the suction box at lower pressures. The effects of changes in skin temperature were eliminated in two additional studies in which skin blood flow to one forearm was arrested by epinephrine iontophoresis. Total forearm blood flow was measured in the contralateral forearm, and forearm muscle blood flow was estimated as the flow to the arm which had been subjected to iontophoresis.

Forearm blood flow was measured by venous occlusion plethysmography with a double-stranded mercury-in-Silastic strain gauge designed by Whitney (9). Circulation to the hand was occluded at the wrist for 3 of every 4 minutes throughout each study. The venous occlusion cuff around the upper arm was inflated to 40 mm Hg for 15 seconds of every 30 seconds while the wrist cuff was inflated. Forearm blood flow was calculated using a laboratory computer to convert slopes of plethysmographic recordings to flows as previously described (10).

Arterial blood pressure was recorded from a 20-gauge Teflon catheter inserted into a brachial artery using a modified Seldinger technique. The catheter tip was positioned under fluoroscopic guidance in the aorta at the junction of the left subclavian artery. Methods for the optimal electronic registration of aortic pulse pressure and aortic mean pressure from the same catheter and a Statham P23Gb transducer have been previously described (11). As a check, at the conclusion of one study the catheter was quickly removed from the brachial artery of the subject and immediately sealed in a piston-phone chamber; the response of the entire manometric system to a sinusoidal pressure input which varied from 1 to 150 Hz was determined as previously described (11). The manometric system had flat response characteristics to over 30 Hz.

To calculate beat-to-beat values for aortic mean pressure, aortic pulse pressure, and dP/dt max, we used a laboratory computer to sample analog pressure signals at 500 samples/sec and computed arterial systolic pressure, diastolic pressure, aortic mean pressure, and aortic pulse pressure for each cardiac cycle. Also, dP/dt

| TABLE 1 |
|Specification of Procedures Used with Each Subject|

<table>
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<th>Subject</th>
<th>Forearm blood flow - heart rate</th>
<th>Aortic pressure</th>
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<th>Iontophoresis</th>
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Entries in each column indicate the number of experiments in which the specified parameter was measured or the specified procedure was used with each subject. Forearm blood flow-heart rate column includes the two iontophoresis experiments.
max was estimated as the maximal rate of rise of aortic pressure over any 20-msec interval within a cardiac cycle, i.e., the greatest pressure rise over ten successive samples. Arterial blood pressure, heart rate, and right atrial pressure data were smoothed by computing average values for every 30 seconds throughout a study. Right atrial pressure was recorded with a Statham P23BB transducer and a catheter percutaneously inserted at the antecubital fossa and placed under fluoroscopic guidance at the junction of the superior vena cava and the right atrium.

Splanchnic blood flow was measured by the constant dye-infusion technique as previously described (12, 13). After a 12.5-mg priming injection, indocyanine green dye, which is extracted solely by the liver, was infused into a superficial vein at a rate of 0.5 mg/min throughout the experiment. After a 20-minute dye equilibration period, blood samples were taken from either the arterial catheter or another forearm vein distal to the infusion site at 2- or 3-minute intervals throughout the control, the experimental, and the recovery periods; arterial and venous dye concentrations were equal. Since previous studies have shown that the hepatic extraction of indocyanine green dye remains constant throughout lower body negative pressure (8), hepatic veins were not catheterized, and the hepatic arteriovenous dye difference was estimated as 85% of the systemic dye concentration (13-15). Thus, the removal rate (R) of indocyanine green dye was calculated from the infusion rate (I) minus the rate of change of dye concentration in systemic blood (\(C_s\)) times the plasma volume (PV) (\(R = I - C_s \cdot PV\)). Plasma volume was estimated as 45 ml/kg body weight (16). Splanchnic blood flow (SBF) was calculated as SBF = R/0.85C_s(1-Hematocrit). Individual differences in extraction or errors in the estimation of plasma volume can lead to slightly inaccurate estimates of absolute splanchnic blood flow levels, but changes in splanchnic blood flow should be followed with the degree of accuracy previously specified (8). Changes in blood volume due to sampling or to extravasation of plasma can produce, at most, a very small error (8).

Skin temperature was calculated as the electrical average from ten copper-constantan thermocouples placed over the body (17). The subject was dressed in a water-perfused suit as described previously (17, 18) and the temperature of the water perfusing the suit was controlled to maintain skin temperature at a fixed, subjectively neutral level. The technique used for iontophoresis of epinephrine into the skin of one forearm has been described previously (19). A continuous ramp of lower body negative pressure was achieved by manual control of a valve on the suction box using a wide-scale display of box pressure (Statham P23Db transducer) on a 12-inch strip-chart recorder for visual feedback. Pressure was reduced in 0.25-0.5-mm Hg steps over any 15-30-second period. The pressure ransducer was calibrated between 0 and —50 mm Hg before each study using a graduated 100-cm water nanometer.

Statistical analysis was performed with a grouped t-test (20) that compared all splanchnic blood flow data or each 3-minute interval during the ramp with all control data as percents of each individual's average control splanchnic blood flow.

**Results**

In this investigation not all of the parameters were measured in each of the 30 studies. Nevertheless, results for aortic mean pressure, aortic pulse pressure, and right atrial pressure were similar among the different subjects, especially over the early (0–20 minutes) portion of a ramp. Responses of heart rate and forearm blood flow were likewise similar among the subjects and during repeated measurements. Splanchnic blood flow showed a more variable response that was largely due to the lower signal-to-noise characteristics of the measurement (±10% SD) and to the lower sampling rate (0.33-0.5/min compared with 2/min or greater for other variables). To determine the pattern of the splanchnic blood flow response to lower body negative pressure, we averaged all studies in which splanchnic blood flow was measured over the range of control to —40 mm Hg. Splanchnic and forearm blood flow data from duplicate studies on a given subject were averaged and treated statistically as a single observation. Splanchnic blood flow data from studies terminated prior to —40 mm Hg were eliminated, leaving 10 to 14 experiments (seven subjects). Thus, the protocol performed in all remaining experiments was identical up to a lower body negative pressure of —40 mm Hg. Other data from the four studies terminated at —40 mm Hg were not included in the averages. Values for splanchnic and forearm blood flow are percents of each individual's average control splanchnic and forearm blood flow. Figure 1 shows the averaged responses for arterial blood pressures in six subjects, right atrial pressure in four of these subjects, splanchnic blood flow in seven subjects, and forearm blood flow and heart rate in nine subjects. These averages results corresponded well to the individual responses, especially with aortic mean pressure, aortic pulse pressure, heart rate, right atrial pressure, and forearm blood flow, but interindividual responses of splanchnic blood flow were slightly more variable (Figs. 2, 3, and 5). Figure 2 shows responses of a subject in which all of the measurements were taken simultaneously over the full 50 minutes of lower body negative pressure.

The major findings, represented by the average responses from all subjects in Figure 1, may be summarized according to the responses seen prior to a lower body negative pressure of —20 mm Hg and those seen during a lower body negative pressure in the range of —20 to —50 mm Hg.
Right atrial pressure fell in a nearly linear fashion from $4.2 \pm 0.7$ (SE) mm Hg (control) to $-0.6 \pm 0.6$ mm Hg; this response was similar in the four subjects in whom right atrial pressure was measured. Average heart rate was $62 \pm 1.6$ beats/min during control; it fell to $60 \pm 1.6$ beats/min at $-10$ mm Hg and returned to $62 \pm 1.6$ beats/min at $-20$ mm Hg. Some subjects showed no change in heart rate during this period, others showed a slight fall ($5$–$7$ beats/min), and one subject showed a rise of $10$ beats/min. Aortic pulse pressure and aortic mean pressure were both constant over the interval from 0 to $-20$ mm Hg in three of six subjects and increased in one subject. In the other two subjects, aortic pulse pressure began to fall below control values at a lower body negative pressure of $-15$ mm Hg, but aortic mean pressure remained at control levels. No consistent trends in aortic $dP/dt$ max were observed. In four of these subjects, $dP/dt$ max initially rose above control levels but returned to control between $-20$ and $-30$ mm Hg. In another subject there was a small, initial fall from $650$ mm Hg/sec to $600$ mm Hg/sec after which $dP/dt$ max was essentially unchanged. In the remaining subject, $dP/dt$ max initially fell below control values, rose to above control at $-10$ mm Hg, and fell below control again at $-20$ mm Hg. Forearm blood flow in all subjects began to fall with the onset of lower body negative pressure and on the average, had fallen to $67 \pm 2.4\%$ of control at $-20$ mm Hg. Control of skin temperature did not reduce the forearm blood flow response. Iontophoresis did not prevent the sudden reduction in forearm blood flow at the onset of lower body negative pressure, and it did not affect the time course of the forearm blood flow response. Forearm blood flow to the arm subjected to iontophoresis was $2.9$ ml/100 ml min$^{-1}$.
Simultaneously measured responses of each variable depicted in Figure 1 from one subject. Abbreviations are the same as in Figure 1. Also shown is the maximal rate of rise of aortic pressure (dP/dt).

During control, and it had fallen to 1.8 ml/100 ml min⁻¹ at -20 mm Hg. Forearm blood flow in the control arm fell from 4.7 ml/100 ml min⁻¹ during control to 3.1 ml/100 ml min⁻¹ at -20 mm Hg. Thus, the forearm muscle was vasoconstricting; forearm vasoconstriction was not due to skin vasoconstriction caused by cooling. Splanchnic blood flow showed a less marked and a more variable response than did forearm blood flow, making it difficult in any one experiment to estimate the time at which splanchnic blood flow began to fall. Part of the variability was analytical (8), i.e., it arose from the technique used, and part was undoubtedly due to real fluctuations in splanchnic blood flow. The variability of the measurement during control periods forced us to use the average response for all subjects as percents of each individual's average control splanchnic blood flow to determine the time at which splanchnic blood flow was first significantly reduced below control values. We then calculated the average percent splanchnic blood flow for each 3-minute interval over the control period to -49 mm Hg. In seven subjects splanchnic blood flow, on the average, was significantly reduced at -7 mm Hg (95.5 ± 1.4% of control, \( P < 0.05 \)) and fell to 89.5 ± 4.1% of control by -21 mm Hg. Although splanchnic blood flow was reduced significantly below control at -7 mm Hg and -10 mm Hg, the reduction at -13 mm Hg (98.3 ± 2.8% of control) was not significant \( (P > 0.05) \). This result was largely due to an unexplained transient increase in splanchnic blood flow in one subject at this time. Splanchnic blood flow was significantly reduced below control at all times after -13 mm Hg, and calculated splanchnic vascular resistance showed a corresponding rise.

There were interindividual differences in the time course of the splanchnic blood flow response (Fig. 3). One subject clearly showed an early fall in splanchnic blood flow, and another showed a delayed fall. In four subjects splanchnic blood flow fell before lower body negative pressure had reached -20 mm Hg; in three subjects this decrease was less clear. Each subject had the same general pattern of response from one study to the next.

**EFFECTS OF LOWER BODY PRESSURE FROM -20 to -50 MM Hg**

Right atrial pressure continued to fall, but it fell less steeply than it did during the first 20 minutes. At -50 mm Hg, right atrial pressure averaged -3.25 mm Hg (two subjects). Heart rate increased in a nearly linear fashion between -20 and -50 mm Hg; this linear increase consistently occurred in all studies. At -20 mm Hg, heart rate averaged 62 ± 1.6 beats/min and rose to an average of 82 ± 3.7 beats/min at -50 mm Hg. Aortic pulse pressure began to fall at about -20 mm Hg (range -15 mm Hg to -25 mm Hg), the fall in aortic pulse pressure paralleled the rise in heart rate. At -40 mm Hg, aortic pulse pressure averaged 29 ± 4.4 mm Hg. Figure 4 is a plot of aortic pulse pressure vs. heart rate for the duration of lower body negative pressure in a single subject, exemplifying the close correlation always found between these parameters. Correlation coefficients for heart rate vs. aortic pulse pressure varied between -0.86 and
Subject A shows the usual progressive fall in splanchnic blood flow and rise in arterial dye concentration during lower body negative pressure; three other subjects showed this response. Subject B shows the most delayed response. Two other subjects showed no clear change in splanchnic blood flow or arterial dye concentration prior to a lower body negative pressure of -15 to -20 mm Hg.

-0.93 for five of the six subjects in whom aortic pulse pressure was measured. The sixth subject had a lower correlation (-0.56) due to an abbreviated protocol during which heart rate and aortic pulse pressure were only slightly changed from control. After -20 mm Hg, aortic mean pressure remained unchanged in two subjects, fell slightly (4-5 mm Hg) in two subjects, and fell dramatically in two subjects. The latter two experiments were terminated to avoid syncope. Aortic dP/dt max continued to show no consistent trends. Forearm blood flow continued to fall as lower body negative pressure progressed, but it fell less steeply; between -20 and -50 mm Hg, forearm blood flow fell from

67 ± 2.4% to 55 ± 6.0% of control. Splanchnic blood flow continued to decrease as lower body negative pressure progressed. There was no apparent difference in the rate of decline of splanchnic blood flow between -20 and -50 mm Hg compared with that between 0 and -20 mm Hg. At -40 mm Hg, splanchnic blood flow averaged 80.1 ± 2.0% of control.

Discussion

The results of these experiments agree with those of Roddie et al. (7) and Zoller et al. (5) that forearm blood flow can be significantly altered without consistent, measurable changes in the parameters of arterial blood pressure which are thought to be important inputs to arterial baroreceptors. In our studies and those of Zoller et al. (5), aortic pulse pressure and aortic mean pressure usually remained at control levels until lower body negative pressure reached about -20 mm Hg. The rise in heart rate and the fall in aortic pulse pressure began at -20 mm Hg. Heart rate vs. aortic pulse pressure reveals the high correlation (r = -0.92) between these two variables.
mm Hg; however, forearm blood flow was consistently and markedly reduced. The maximal rate of rise of aortic pressure showed no consistent trends during this 20-minute period; it was unchanged or increased in four subjects and reduced in two subjects. The close correlation between aortic pulse pressure and heart rate suggests that increased sympathetic outflow evoked by arterial baroreceptors began when heart rate rose above control at a lower body negative pressure of about -20 mm Hg. Although we cannot report that absolutely no change in the arterial signal occurred during this early portion of the ramp, no change measurable by our techniques occurred in any subject except one. This one exception had increases in aortic pulse pressure, aortic mean pressure, and dP/dt max during the first 20 minutes of lower body negative pressure (Fig. 5). Nevertheless, the onset of lower body negative pressure and presumably the fall in right atrial pressure were associated with a rapid reduction in forearm blood flow and a less dramatic fall in splanchnic blood flow. The iontophoresis studies and the studies in which skin temperature was kept constant indicate that the fall in forearm blood flow was not caused by thermally induced cutaneous vasoconstriction.

A unique finding in this study was that splanchnic vasoconstriction can also occur without measurable or consistent changes in arterial blood pressure. On the average, splanchnic blood flow fell to 89.5 ± 4.1% of control (P < 0.001) before lower body negative pressure had reached -20 mm Hg or before there was any change in heart rate. Previous investigations (8) have suggested that splanchnic blood flow more closely parallels the fall in aortic pulse pressure than that in right atrial pressure when lower body negative pressure is applied in steps of -50 mm Hg (8). Reductions in aortic pulse pressure occurred too rapidly during these large steps to allow discrimination of the small reductions in splanchnic blood flow that accompanied a fall in right atrial pressure alone.

Our studies along with those of others suggest that the likely source of the stimulus for these vasomotor changes arises from low-pressure baroreceptors. To date such receptors have only been identified in the cardiopulmonary region of lower species, and reflex vasomotor responses originating from such stretch receptors have been implicated in several studies. For example, distention of the pulmonary vein–left atrial junction in dogs increases heart rate (21), but section of vagal afferent fibers arising from the heart in cats causes marked renal vasoconstriction and increased heart rate with less marked vasoconstriction in skeletal muscle (3). In dogs, selective reduction of carotid sinus and cardiopulmonary pressures has indicated that reflexes originating from low-pressure baroreceptors are preferentially directed toward the splanchnic and the renal circulations whereas...
First, both splanchnic and forearm blood flow responses may be minor. However, species differences may be important; for example, Scher et al. (22) have suggested that unanesthetized dogs rely much more on changes in heart rate induced by the vagus than does man, who relies more on increased sympathetic nerve activity in his response to arterial baroreceptor stimulation. On the average, 83% of the reduction in forearm blood flow seen at —40 mm Hg had occurred by —20 mm Hg, but only 53% of the reduction in splanchnic blood flow seen at —40 mm Hg had occurred by —20 mm Hg. An unresolved question is whether this difference in the pattern of the response of these vascular beds is due to different sensitivities to vasomotor outflow in individual vascular beds or to differing degrees of sympathetic outflow to the forearm and viscera.

We have implied that the constriction in splanchnic and forearm vasculature is neurogenically mediated. Although this supposition is not directly supported in these experiments, it is supported by several studies in which the forearm vasodilation seen with passive leg raising (6) or the forearm vasoconstriction seen with upright posture (23, 24) or with lower body negative pressure (25, 26) could be blocked by surgical or pharmacological sympathectomy of the forearm. Similarly, Wilkens et al. (27) have found in hypertensive patients that the increase in splanchnic vascular resistance normally accompanying upright tilting is essentially absent following surgical sympathectomy of the forearm. Similarly, Wilkens et al. (27) have found in hypertensive patients that the increase in splanchnic vascular resistance normally accompanying upright tilting is essentially absent following surgical sympathectomy of the forearm. Several observations in our studies also suggest that the role of humoral agents in splanchnic and forearm responses may be minor. First, both splanchnic and forearm blood flow rapidly increase when suction is suddenly reduced from —50 to —20 mm Hg and then to —10 mm Hg; they rapidly return to control when suction is ended (Fig. 2). However, since the clearance rate of humoral agents is unknown, we cannot determine whether the return of flow is rapid enough to discount humoral contributions. Second, the beta-adrenergic actions of epinephrine on splanchnic blood flow opposed the vasoconstriction, corresponding effects on heart rate and forearm blood flow should have been obvious. The rapid recovery of forearm blood flow and heart rate at the end of suction and the failure to observe rising heart rate during the early stages of lower body negative pressure suggest that epinephrine is not a major factor in the responses. Nevertheless, the splanchnic vasoconstriction observed in the first few minutes of our experiment may not have been neurogenically mediated. Humoral factors have been implicated in the response to hemorrhage of the cutaneous bed of dogs (28), the renal bed of rabbits (29), and the intestinal bed of cats (30). However, the importance of species differences, anesthesia, and changes in arterial blood pressure in these respects is unresolved.

Our results cannot be ascribed to autoregulation, which implies a constant blood flow in the face of an altered perfusion pressure. Such a mechanism has been proposed for human skeletal muscle (31) only during sympathetic blockade. The changes we observed do not comply with autoregulatory behavior. If autoregulation were present in the forearm or the splanchnic beds, it would oppose the observed changes rather than account for them.

The sensitivity of the method for estimating changes in splanchnic blood flow requires comment. Changes in arterial dye concentration closely track changes in splanchnic blood flow during lower body negative pressure (8, 32). The maximal total error in estimating a change in splanchnic blood flow from tracking and measurement errors is 8% of any given change in splanchnic blood flow over a 2-minute sampling interval (8). To explore this problem further, we solved the equation for the behavior of arterial dye concentration for the following conditions: (1) if splanchnic blood flow had changed linearly from 1.5 to 1.0 liter/min over the time course of a ramp from 0 to —50 mm Hg or (2) if splanchnic blood flow had changed by the same amount only over the period from —20 to —50 mm Hg. The solutions for the predicted behavior of arterial dye concentration for these two conditions, and the average time courses of arterial dye concentration, and the corresponding time courses of splanchnic blood flow are shown in Figure 6. The measured time course of splanchnic blood flow and arterial dye concentration more closely approximated those in which splanchnic blood flow begins to fall with the onset of lower body negative pressure. Accepting the previous analysis (8) for the sources and potential magnitude of errors in measuring arterial dye concentration and estimating splanchnic blood flow, we cannot with certainty say whether splanchnic blood flow began to fall with the onset of lower body negative pressure or...
The degree of correlation between heart rate and aortic pulse pressure agrees with previous findings that the carotid sinus baroreflex is sensitive to aortic pulse pressure (33) and to the rate of change of pressure (34). In our studies, aortic dP/dt max showed no consistent changes. However, because aortic pulse pressure is a function of the average rate of rise of aortic pressure, a reduction in aortic pulse pressure would correspond to reduced stimulation of arterial baroreceptors.

In man, therefore, forearm and splanchnic blood flow, but not heart rate, are targets of increased sympathetic activity resulting from small displacements of blood from the central circulation. Moreover, these vascular beds constrict prior to any measurable fall in aortic mean pressure, aortic pulse pressure, or dP/dt max. The rise in heart rate above control signals the time and the degree of the fall in aortic pulse pressure. The data strongly suggest that reflexes arising from cardiopulmonary stretch receptors initiate the falls in both forearm and splanchnic blood flow but that greater vasoconstrictor effects occur in the forearm.

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