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Peridural Anesthesia and the Distribution of Blood in Supine Humans

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To determine the effects of vasomotor tone on intrathoracic and splanchnic blood volume, the distribution of radioactively (^{99m}Tc) labeled erythrocytes was recorded by whole body scintigraphy before and during peridural anesthesia (PDA) in eight supine men. The radioactivity was recorded with a gamma camera and its distribution determined in the thorax, abdomen, and limbs. Arterial and central venous pressure, heart rate, and calf volume and flow also were measured. During PDA with a sensory block up to $T_{4/5}$, radioactivity increased only in the denervated legs (+9.9 ± 2.3% SE), whereas it decreased in all other regions, *i.e.*, in the thorax (−8.1 ± 1.2%), the innervated upper limbs (−10.6 ± 4.0%), and in the splanchnic vasculature (−5 ± 1.7%). However, in two of the subjects, after an initial decrease, splanchnic blood content increased while intrathoracic blood volume decreased further. The effects of PDA on thoracic and splanchnic filling could be duplicated by the sequestration of about 500–600 ml of blood in both legs. In supine humans, high peridural anesthesia evokes the same decrease in intrathoracic blood volume as orthostasis. This seems to be counteracted by a reflex decrease in filling of the denervated muscle and skin areas and also by a constriction of the splanchnic vasculature by an unknown mechanism. Potential circulatory collapse may ensue when the vasoconstrictor response fails in the splanchnic circulation. (Key words: Anesthetic techniques: epidural. Hemodynamics: epidural anesthesia. Sympathetic nervous system: epidural anesthesia. Veins: capacitance vessels, splanchnic, thoracic.)

NUMEROUS PUBLICATIONS deal with the effects of major conduction block on vascular flow/resistance relationship.[1,2] In contrast, little is known about the response of the capacitance vessels to the loss of vasomotor tone, which is likely to disturb the relationship between vascular capacity and blood volume, thereby jeopardizing cardiac filling.[3,4] In supine humans, peridural anesthesia (PDA) or spinal anesthesia lead to blood pooling in the denervated lower extremities and a reflex vasoconstriction in the innervated arms.[5–7] That this may evoke a redistribution of blood at the expense of cardiac filling is at least suggested by an accompanying decrease in central venous pressure,[6,8–10] but how a loss of efferent sympathetic drive affects thoracic and also splanchnic blood volume is unknown. We therefore looked at the distribution of blood before and after peridural anesthesia by means of whole body scintigraphy in combination with plethysmography. We found that peridural anesthesia caused a redistribution of blood into the denervated musculature and skin at the expense of cardiac filling. We also speculate that the splanchnic vasculature plays a crucial role in cardiovascular stability during peridural anesthesia through an unknown mechanism.

Methods

Eight healthy male volunteers (four physicians and four medical students) between the ages of 22 and 42 yr (average 33 yr) with a body weight of 77 kg (range 60–92 kg) gave their informed consent to participate in the study. They had fasted overnight, but fluid intake was not restricted until 2 h before the start of the experiments. The subjects were studied in the horizontal decubitus position at a room temperature of 27° C between 9:00 A.M. and 12:00 A.M. (six experiments) and between 1:00 P.M. and 5:00 P.M. (two experiments). No fluids were given intravenously.

Before each study, 5 ml of venous blood was withdrawn for erythrocyte labeling with technicium (^{99m}Tc, 5 mCi = 5 × 37 MBq) and reinjected intravenously.

A peridural catheter was inserted through an 18-gauge Crawford® needle at the L2–3 interspace. Both brachiocephalic veins were cannulated, one for drug infusion, the other for recording the central venous pressure (CVP) via a catheter that was advanced into the superior caval vein as judged from the pressure contour. The ECG was monitored by standard leads using surface electrodes. For measuring calf volume and blood flow, mercury-in-rubber gauges[11] fixed around both calves allowed the measurement of calf volume and blood flow by occlusion plethysmography. Two blood pressure cuffs (width 20 cm) were loosely fixed around the thighs. When pressurized to 100 mmHg, about 500–600 ml of blood could be pooled below the cuffs (leg congestion test). The placement of the various catheters and transducers took about 30 min, an interval sufficient to allow for equilibration of the labeled erythrocytes.

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Measurements

Radioactivity was recorded continuously by whole-body scintigraphy with the use of a gamma camera (Searl LFOV®, equipped with a parallel hole, high-resolution collimator) in the anterior-posterior direction. The subjects remained supine throughout the study, with their torso and extremities fixed by appropriate cushions. The counting sequence employed started on the left side from foot to head and in the reverse order on the right side.[12] Each counting cycle took 6 min (=1 scan) repeated without interruption over the duration of each study for a total of 96 min, *i.e.*, 16 scans. The counts were acquired by a computer (DEC PDP 11/15), stored, processed, and whole body images reconstructed for each scan. Average counting rates (counts per scan = cps, corrected for the physical decay of ^{99m}Tc) were printed for the following regions: 1) skeletal muscle (arms and legs; 2) splanchnic region (abdomen and liver plus spleen); 3) thoracic region (heart and lung). Additionally, the following variables were measured continuously: central venous pressure (CVP) electromanometrically (Statham D23db®); heart rate with an ECG-triggered cardiotachometer and calf circumference (Whitney gauge)[11] for deriving flow (occlusion plethysmography) and volume. The calf circumference was determined by tape initially and the mercury-in-rubber strain gauge then was calibrated at frequent intervals by an in-series micrometer. The arterial blood pressure was measured sphygmomanometrically by the same person at the end of each scan. The zero reference point for CVP was set at half the ventro-dorsal thickness of the chest.

Experimental Protocol

All experiments were started about 1 h after the injection of the tracer. The following scheme was adopted to each subject: 1) control for 18 min (= scans 1–3) with a leg congestion test during scan 2; 2) peridural anesthesia over a period of 24 min (= scans 4–7).

For peridural anesthesia, lidocaine 20 ml of a 2% solution was injected at the start of scan 4. The sensory block (tested by pin prick) reached T_5 in all of the subjects, in some of the smaller subjects T_4 within 18 min, and remained at these levels until the end of the recording period.

Data Analysis

Data are expressed as averages (±SE). The Wilcoxon rank test was employed for testing differences. P values less than 0.05 were considered significant.

Results

Blood Distribution

The distribution of the radioactivity before and during peridural anesthesia is shown in a representative example in figure 1. There is an increase in radio activity in the denervated legs, particularly the feet and calves, representing skin and muscle. By contrast, radioactivity decreased in the thorax, with the heart image being distinctly smaller during PDA than before.

The time course of these effects is shown in figure 2. After the peridural injection of lidocaine, there was a gradual increase of radioactivity in the denervated legs by about 10%, whereas the radioactivity decreased to a similar degree in the innervated arms and the thorax. Note also the paradoxic decrease in radio activity in the *denervated* abdominal region.

Scintigraphy provided only information on the relative distribution of blood content. To obtain an estimate of the actual volume shifts, the leg congestion test was applied in each case during scan 2 in the control period. Radioactivity increased strongly in the congested legs, while at the same time it decreased in both the thorax and abdomen to almost the same degree as during subsequent peridural anesthesia. Because about 500–600 ml of blood was sequestered in the legs during the pooling period (see below), we concluded that the thoracic and splanchnic filling must have been reduced by a similar volume.

To illustrate the response of special organs such as the heart, liver, and spleen, figure 3 contrasts the maximum changes in counting rates in the various regions. During peridural anesthesia the counting rates decreased in all regions except in the legs, where increases were noticed. The effects of PDA in the thoracic and splanchnic vasculature were duplicated by the leg congestion test. It should also be noted that the changes in counting rates in the restricted liver–spleen region was only slightly less than in the total abdomen.

There were uniform effects of peridural anesthesia in the thorax and the various muscle regions (table 1). However, the behavior of the splanchnic bed was not uniform. Here, the radioactivity decreased without exception during the onset of peridural anesthesia (up to scan 5), but after this initial decrease it started to increase in two subjects (subjects 4 and 6). These cases are noteworthy because in both the thoracic counting rates and arterial pressure exhibited the greatest decreases. In fact, incipient vasovagal syncope (heart rate decrease from 65 to 48 beats/min and blood pressure from 130/100 to 90/50 mmHg in combination with sweating, nausea, and dizziness) occurred in subject 4, who happened to have the highest body weight (92 kg) and a sensory block at T_5.

Cardiovascular Measurements

As can be seen in figure 4, the central venous pressure and calf volume mirrored the changes in blood content in the thorax and legs. Interestingly, there was only a small decrease in central venous pressure of 1.3 mmHg during peridural anesthesia, as compared with the 3.5

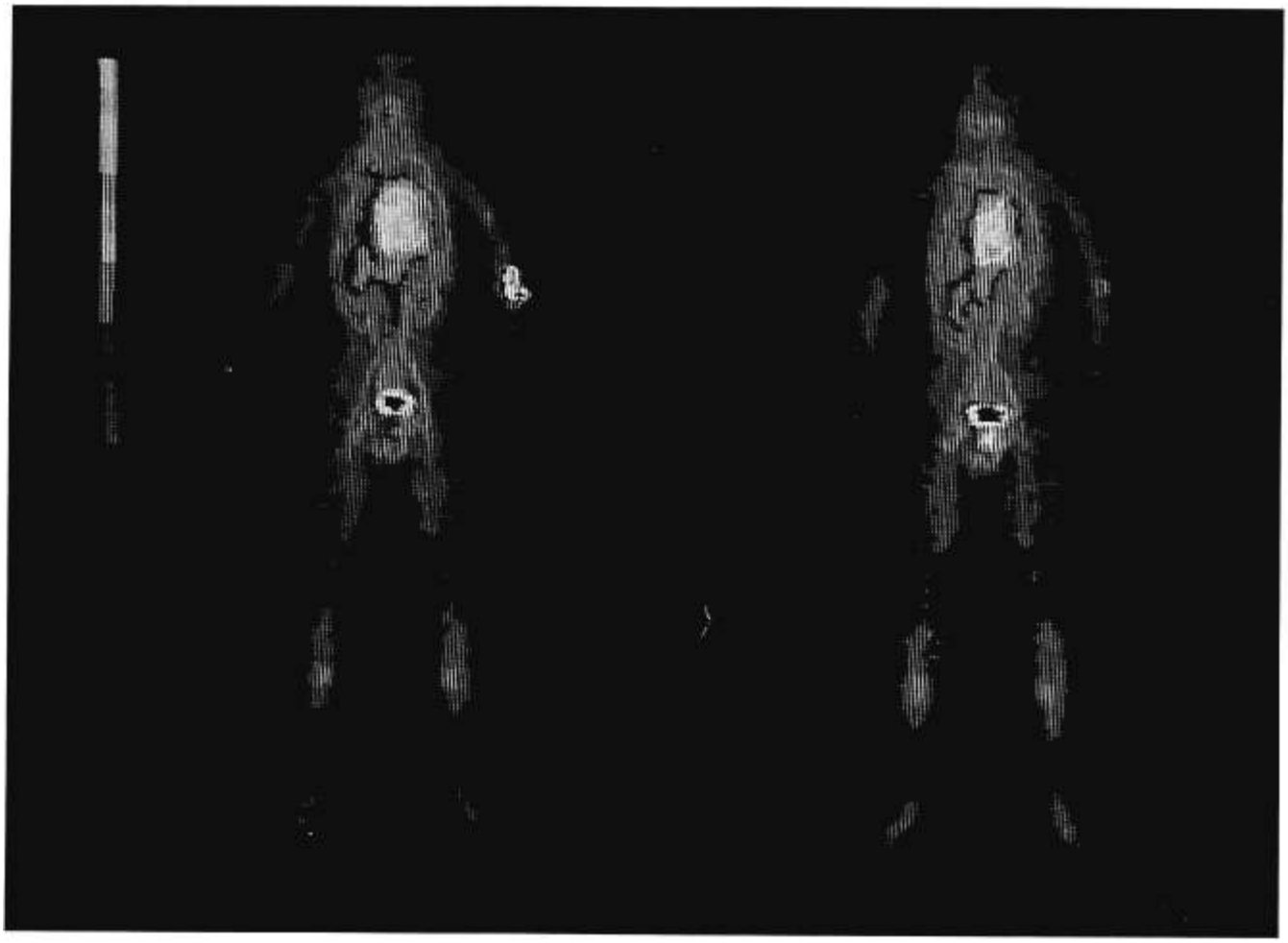

FIG. 1. Distribution of technicium-labeled erythrocytes before and during PDA. Subject 4, 92 kg, sensory block T_5, whole body image from scan 3 on the left and scan 7 during PDA on the right, activity increases from dark blue to white according to the color scale. (The radioactively polluted sponge in the cubital fossa subsequently was removed.) During PDA, the areas of high activity (lighter colors) are smaller than before in the cardiac region but larger in the denervated legs, particularly in the feet (mainly skin) and also the calves (mainly muscle tissue).

mmHg decrease during the leg congestion test, even though peridural anesthesia and the leg congestion test decreased the thoracic blood content by the same degree.

Calf volume, which represents primarily the behavior of the capacitance vessels of the striated musculature, increased by about 4 ml/100 ml tissue during leg congestion and by 1 ml/100 ml during PDA. The former figure allows one to roughly estimate the blood volume pooled in the legs. If the leg is regarded as a cylinder 60 cm in length (average distance between the distal edge of the cuff and the heel in our subjects) having a circumference that corresponds to the average calf circumference, namely 38 cm, one can calculate a tissue mass of approximately 14 kg for both legs. Using the above figure of 4 ml/100 ml tissue, both legs therefore must have accommodated on the average a total of about 560 ml blood during the leg congestion test. The increase in calf volume during peridural anesthesia also was accompanied by a significant increase in calf blood flow, which almost doubled and was maintained until the end of the recording period.

On the average, heart rate and arterial blood pressure changed little during peridural anesthesia, but it is important to note that some of the individual responses were quite different. As is seen in table 2, blood pressure decreased most in those three subjects who happened to have the highest values (>100 mmHg) during the control (scan 3). In two of these subjects (subjects 4 and 6), one of whom (subject 4) almost fainted, blood pressure remained well below the controls also during scans 6 and 7.

Discussion

By using 20 ml of a 2% solution of lidocaine it was possible to achieve sensory block to at least T_5, in some

of the smaller subjects up to T_4. Because peridural lidocaine in a dose of 400 mg should not achieve arterial blood levels greater than 3 μg/ml, no direct depressant effects on either the vascular smooth muscles or the myocardium should be seen.[13,14] The significance of a recent report showing lidocaine concentrations of the order of 3 μg/ml to decrease arteriolar diameter by about 10% in rats is difficult to tell.[15] Generally, nerve block elicits profound vasodilation,[1,2] which appears to exclude direct effects of lidocaine as an important factor for the vascular response to sympathetic block. Consequently, in all likelihood the loss of sympathetic drive determined the principal effects in the present study. The blood content increased in the legs but decreased in the arms. Since the sympathetic supply to the arms originates from the same segments as that to the heart,[16] the cardiac sympathetic innervation presumably remained by and large intact as evidenced also by the absence of statistically significant heart rate changes.[1] Thus it appears safe to assume that peridural anesthesia interrupted the sympathetic outflow below T_5 in all of our subjects.

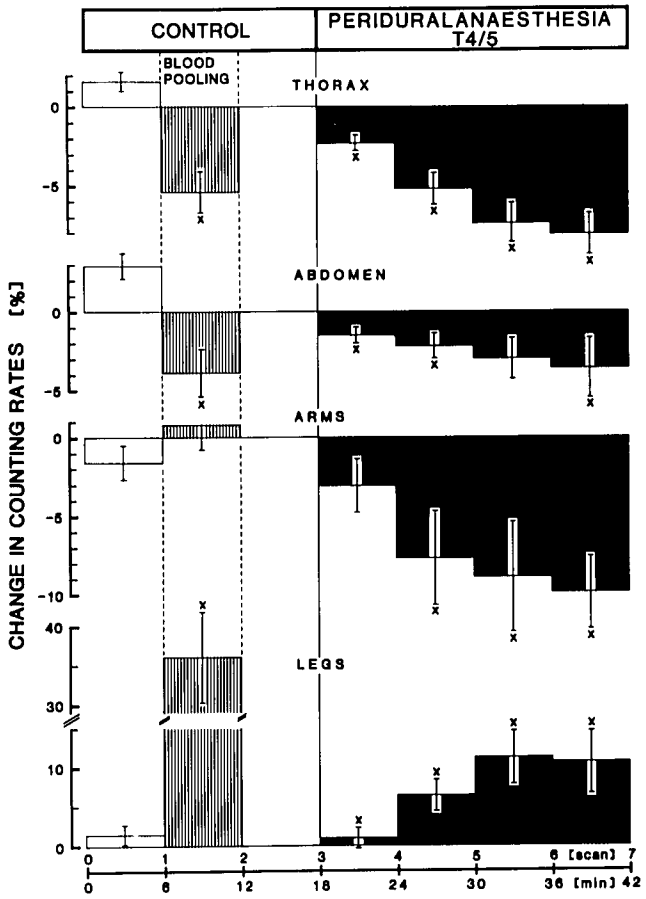

FIG. 2. The effects of PDA on the distribution of technicium-labeled erythrocytes in supine man. Activity was sampled for 6 min (=1 scan). Changes are expressed as a percentage of the third scan. Blood was sequestered in both legs (= blood pooling) during scan 2. Averages (±SE) from eight experiments. Wilcoxon rank test, x = $P < 0.05$. During PDA radioactivity decreased in all regions except the denervated legs. Note also that the decrease in intrathoracic and splanchnic blood content corresponded to that during the blood pooling test.

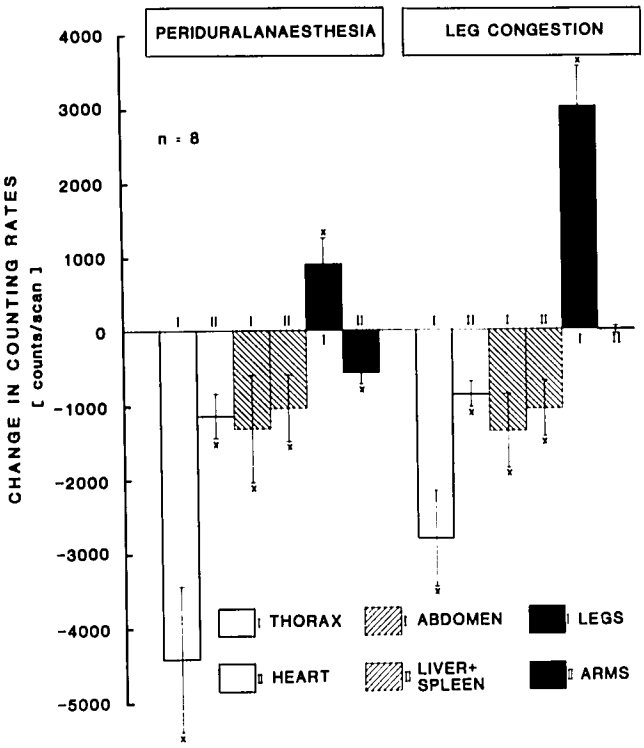

FIG. 3. Effects of peridural anesthesia ($T_{4/5}$) and blood sequestration in both legs (leg congestion test) on regional blood volume distribution in supine humans. Maximum changes in counting rates (averages ± SE). Wilcoxon rank test, x = $P < 0.05$. PDA evoked almost the same decrease in counting rates in the thorax and abdomen and, specifically, the cardiac and upper abdominal region (liver and spleen). Note also that the changes in counting rates were almost identical for the total abdomen when compared with the more limited liver and spleen region.

Given that the greater proportion of the blood volume is contained within the capacitance vessels[4,17] and that the radioactivity reflects the erythrocyte concentration in any given region, the experiments show the importance of vasomotor tone in controlling the distribution of blood within the capacitance vessels, particularly in relation to intrathoracic filling. The foregoing results support previous plethysmographic observations that also demonstrated the pooling of blood in denervated limbs and vasoconstriction in the innervated limbs.[2,5,6,8] Not previously demonstrated, however, was the decrease in intrathoracic and cardiac blood volume and, of special interest, the decrease in splanchnic filling.

While scintigraphy only yields information about the relative distribution of the blood, using these measurements in conjunction with plethysmography and the effects of the leg congestion test enabled the actual volume

TABLE 1. Regional Counts per Second from Technicium Scintigraphy Expressed as a Percent of Scan 3 (=100%) during Control and Peridural Anesthesia

Region	Subject	Control			Peridural $T_{4/5}$			
		1 [%]	2 [%]	3 [cpx × 10^3]	4 [%]	5 [%]	6 [%]	7 [%]
Thorax	1	1.7	−10.1	23.9	−0.4	−2.4	−4.7	−9.0
	2	2.2	−1.7	25.9	−1.2	−2.0	−4.0	−3.8
	3	0.4	−5.0	71.9	−4.0	−7.2	−9.1	−10.4
	4	2.0	−6.6	67.4	−3.0	−8.2	−13.3	−12.9
	5	5.0	−7.2	61.9	−1.3	−3.9	−4.0	−5.0
	6	±0	−4.3	34.6	−3.3	−9.7	−11.3	−11.3
	7	−0.3	−5.0	85.1	−3.6	−5.1	−7.9	−7.6
	8	1.9	−9.0	60.4	−1.2	−2.7	−4.5	−4.5
Averages		1.6	−6.1		−2.3	−5.2	−7.4	−8.1
SE		±0.6	±1.0		±0.5	±1.0	±1.3	±1.2
P			x		x	x	x	x
Abdomen	1	1.2	−12.3	20.3	−2.4	−2.2	−12.4	−12.2
	2	3.9	−2.3	19.9	−1.6	−2.9	−4.3	−5.7
	3	5.8	−2.5	25.9	−1.4	−7.4	−7.5	−12.5
	4	3.1	−4.5	31.2	−0.4	−7.6	−1.2	−2.4
	5	4.6	+0.7	31.4	±0	−3.3	−4.3	−4.7
	6	±0	−2.9	13.6	±0	−6.3	+0.4	+0.4
	7	−3.7	−2.5	31.2	−2.8	−2.8	−3.9	−1.3
	8	−4.5	−8.0	33.4	−3.4	−0.3	−1.6	−1.6
Averages		1.3	−4.3		−1.5	−4.1	−4.4	−5.0
SE		±1.3	±1.4		±0.5	±0.9	±1.4	±1.7
P			x		x	x		x
Lower legs	1	+3.5	+21	5.8	+0	+3.0	+4.4	+4.4
	2	−3.6	+60	3.7	−6.0	+9.2	+12.3	+3.3
	3	+6.9	+45	5.2	+16.8	+13.2	+32.2	+36.1
	4	+2.9	+55	8.9	+3.0	+12.9	+16.0	+16.4
	5	+0.1	+20	12.7	−3.7	+1.6	+3.7	+4.0
	6	−1.0	+37	6.0	+4.0	+6.0	+4.4	+4.4
	7	−0.8	+15	15.9	−4.3	−0.1	+4.2	+5.7
	8	+4.5	+64	9.3	−2.6	+4.6	+10.9	+10.5
Averages		1.6	40		+0.9	6.3	11	10.6
SE		±1.2	±6.8		±2.6	±1.8	±3.4	±4.0
P			x			x	x	x
Arms	1	−2.0	+6	1.8	−1.1	+6.1	+7.7	−0.5
	2	−1.7	+8.1	1.3	−6.6	−0.5	−6.2	−2.1
	3	+2.6	+0.2	7.4	−1.3	−4.9	−0.9	−12.8
	4	±0	−2.4	8.3	−9.5	−22.2	−23.8	−7.3
	5	−1.2	−0.4	6.8	−4.8	−8.0	−7.0	−11.5
	6	±0	−1.3	3.2	−4.9	−11.5	−9.3	−9.3
	7	−5.2	−5.9	4.9	−3.1	−8.9	−12.4	−16.7
	8	−6.8	−1.9	6.4	−6.6	−11.7	−19.2	−19.1
Averages		−1.8	+0.3		−4.7	−7.7	−8.9	−9.9
SE		±1.1	±1.6		±1.0	±3.0	±3.5	+2.3
P					x	x	x	x

The numbers heading each column of data refer to the serial number of scans, each lasting 6 min. Peridural lidocaine (20 ml 2%) was injected at the end of scan 3 and a leg congestion test performed during scan 2. Wilcoxon rank test, x = <0.05 considered statistically significant.

shifts to be estimated. During the leg congestion test some 500–600 ml blood were sequestered in both legs. While in the upright position, humans pool a similar volume in their legs up to the groin with 78% of this volume coming from the intrathoracic circulation and the rest being derived from the splanchnic circulation.[18] Furthermore, this estimate was supported by the decrease in central venous pressure of 3.5 mmHg during the leg congestion test. In normal supine humans central venous pressure changes approximately 7 mmHg for a change in blood volume of 1 l.[19] Since the intrathoracic–extrathoracic compliance ratio is normally unity,[20] however, half of such an induced blood loss will originate from the intrathoracic and the other half from the extrathoracic vasculature. Hence, a

change in central venous pressure of 7 mmHg for a change in blood volume of 1 l should reflect a change in intrathoracic blood volume of approximately 0.5 l. With the foregoing in mind, leg congestion and peridural anesthesia must have each effectively depleted the intrathoracic and splanchnic vasculature by about 250 ml each. Since both leg congestion and peridural anesthesia elicited about the same changes in counting rates in the thorax, PDA can be said to redistribute blood at the expense of cardiac filling, an effect resembling the blood volume shifts that occur during orthostases.[18]

The depletion of the intrathoracic vasculature induced by peridural anesthesia appears to be counteracted, in part, by the decrease in blood volume in the innervated arms[5,6,8] but, much to our surprise, in part, also by a decrease in splanchnic blood volume. The splanchnic vasculature is an important blood reservoir in humans,[21,22] whose filling is believed to be predominantly regulated by the sympathetic nervous system.[23] In fact, at least one-third of the blood can be mobilized from the liver of dogs and cats during maximal stimulation of sympathetic nerves.[24] Furthermore, in animals splanchnic volume varies by and large directly with splanchnic blood flow achieved either by electrostimulation of splanchnic nerves or by the injection of adrenergic substances.[23,25-27] In humans the effects of sympathetic drive on the splanchnic flow–volume relationship is less well documented. Splanchnic flow, however, decreases with maneuvers known to increase sympathetic tone.[28,29] Similarly, etilefrine, a catecholamine with alpha- and beta-adrenergic properties, conspicuously and preferentially reduced splanchnic filling in supine humans.[30] By inference, a loss of sympathetic drive then should result in an increase of splanchnic flow. Surprisingly, however, during high epidural block, splanchnic blood flow was found to decrease and flow resistance to increase in humans,[31] monkeys,[32] and dogs.[33] Hence, the decrease in splanchnic blood volume observed in the present experiments could readily be interpreted as a consequence of the reduced flow. Nevertheless, it remains puzzling that a loss of sympathetic tone should have a constrictor action in the splanchnic bed, whereas it acts in an opposite manner elsewhere, as in denervated muscle and skin regions.

It is more difficult to explain the *secondary* increases in splanchnic blood content in two of our subjects in whom sensory block had only reached T_5. Since sympathetic influences could hardly be responsible, an unknown neural or humoral mechanism must be considered for the control of splanchnic filling. It appears attractive to consider a vagal link, because the only subject who almost fainted had clear signs of vagal activation, at which time splanchnic filling increased significantly in parallel with a further decrease in intrathorac blood content. The tissue of cholinergic splanchnic vascular control is controver-

TABLE 2. Changes in Mean Arterial Blood Pressure Expressed as a Percentage of Scan 3 (=100%)

	Control			Peridural $T_{4/5}$			
Subject	1	2	3	4	5	6	7
1	+5	+7	93	±0	−5	−5	±0
2	+2	+13	90	+7	+7	+7	+3
3	−3	+7	103	+7	−18	−8	±0
4	±0	±0	110	−13	−17	−57	−23
5	±0	±0	93	−1	±0	−3	−3
6	±1	−9	107	−17	−14	−20	−20
7	±3	±3	87	±0	−9	±0	±0
8	±2	±3	90	±0	±0	±0	−2

The numbers heading each column designate the serial number of scans, each lasting 6 min. Peridural lidocaine (20 ml of 2%) was injected at the end of scan 3 and the leg congestion test was performed during scan 2. Arterial pressure was measured at the end of each scan.

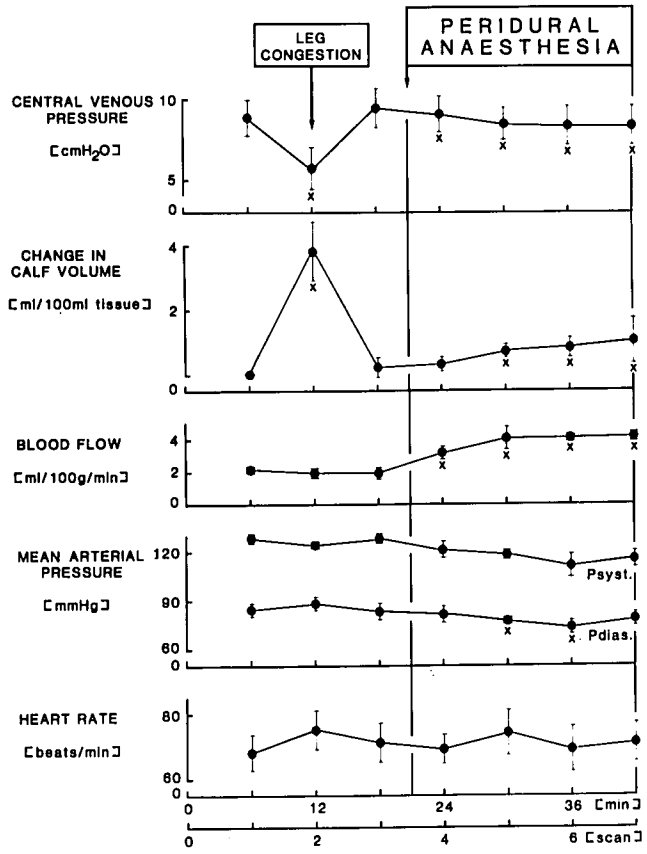

FIG. 4. The response of various cardiovascular variables to peridural anesthesia ($T_{4/5}$) and blood sequestration in both legs (leg congestion) in supine man. Averages (±SE) from eight experiments. Wilcoxon rank test, x = $P < 0.05$. During PDA, the small decrease in central venous pressure was accompanied by an increase of both calf volume and flow and by a small decrease in blood pressure without any change in heart rate. Leg congestion lead to a significant increase in calf volume and a marked decrease in central venous pressure.

sial.[34] There is evidence both for[36] and against such a link.[25,35] Using an isolated perfused liver preparation, the latter authors found acetylcholine to have venoconstrictor properties and to increase liver volume, particularly in the monkey. In humans there is at least circumstantial evidence for the existence of a venoconstrictor mechanism by which splanchnic volume can be affected independently of the sympathetic system. If one accepts the premise that splanchnic blood flow is regulated exclusively through the sympathetic nervous system,[23,28] it is of interest that splanchnic blood volume decreased with hemorrhage in humans, even though flow resistance did not change.[21] These authors concluded "that it (the splanchnic reservoir) can be preferentially depleted of blood by a mechanism which does not automatically increase vascular resistance, and that the ability of our subjects (which all showed signs of imminent fainting) to tolerate blood loss was attributable in large parts to this mechanism." The existence of a presumably vagally mediated vasoconstrictor mechanism may in fact explain why selective blockade of the coeliac ganglion, through which traverse numerous parasympathetic fibers, results in an increase in splanchnic blood flow.[37]

Irrespective of the speculation about the mechanism by which splanchnic filling is controlled, the response of the splanchnic vasculature to the loss of vasomotor tone seems to be of utmost importance for cardiovascular stability during major conduction block.

The importance of sympathetic drive for the distribution of blood, particularly for intrathoracic filling, has been demonstrated here. Elimination of the efferent sympathetic drive, which is associated with peridural anaesthesia, elicits a redistribution of blood into the denervated musculature and skin at the expense of cardiac filling. This is normally counteracted by a compensatory constriction of capacitance vessels in the remaining innervated muscle and skin areas by a reflex increase in sympathetic tone and also by a constriction of the capacitance vessels in the splanchnic area, the mechanism of which is presently unknown. Circulatory collapse seems to ensue when for whatever reasons the vasoconstrictor response by the splanchnic circulation fails.

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