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Systemic and Regional Blood-flow Changes during Spinal Anesthesia in the Rhesus Monkey

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The radioactive microsphere technique was used to determine the distribution of cardiac output and regional blood flow in rhesus monkeys before and 10, 20, 40, and 80 minutes after induction of spinal anesthesia. Five monkeys were studied during low spinal anesthesia (sensory level T10) and five other monkeys were studied during high spinal anesthesia (sensory level T1). Each monkey served as its own control. There was no significant change in regional blood flow during T10 spinal anesthesia. During T1 spinal anesthesia, blood flow (per 100 g of tissue) to kidneys was significantly reduced at 20, 40, and 80 minutes, blood flows to liver and carcass were significantly reduced at 20 and 40 minutes and blood flows to miscellaneous organs (lymph nodes, salivary glands, etc.) were significantly reduced throughout anesthesia. Blood flows to heart, brain, and lower extremity during T1 spinal anesthesia showed only non-significant changes. Vascular resistance in the lower extremity was significantly reduced during both levels of spinal anesthesia, indicating arteriolar dilatation. Also, during both levels of anesthesia, the lungs received an increased proportion of the radioactive microspheres, suggesting increased peripheral arteriovenous shunting of microspheres due to the arteriolar dilatation. (Key words: Anesthetic techniques, spinal, hemodynamics: Brain, blood flow; Kidney, blood flow; Heart, blood flow; Liver, blood flow; Spinal anesthesia.)

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PREVIOUS STUDIES of regional blood flow during spinal anesthesia in experimental animals and in man have been limited to a few organs of the body, made at different times, in different subjects.[1-5] The various methods for the determination of regional blood flow, *viz.*, placement of flowmeter or plethysmograph around the vessel supplying the organ or the organ itself, or measurement of clearance rates of known indicators, are too cumbersome to be employed for simultaneous measurement of blood flows to various organs in the same experimental subject. This has resulted in a lack of knowledge of how cardiac output may be redistributed during spinal anesthesia other than in the organ then under study.

A different technique for simultaneous measurement of blood flows to all organs and tissues of the body has been employed in recent years.[6,7] It consists of injecting non-recirculating carbonized microspheres labeled with different gamma-emitting isotopes into the left ventricle of the heart via a chronic indwelling catheter and later measuring the amounts of radioactivity in the various organs and tissues. Using different isotopic labels at various intervals permits repeated, simultaneous measurements of blood flows to all organs of the body, uncomplicated by recent surgery or anesthesia. This technique has been satisfactorily validated in unanesthetized, restrained monkeys.[7,8]

This technique was employed by us to study systemic and regional blood flow changes in rhesus monkeys during spinal anesthesia under two different dermatome levels of sensory block.

Materials and Methods

The subjects of the study were ten male monkeys (*Macaca mulatta*) weighing 4.9 to 8.4 kg. Under ketamine anesthesia hemato-

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crit determinations were made, polyvinyl catheters were placed in the abdominal aorta and inferior vena cava via the left femoral vessels, and a Teflon catheter was placed in the left ventricle of the heart via the left common carotid artery. The catheters were continuously flushed with heparinized saline solution with a Harvard infusion pump at the rate of 0.5 ml/hour. After operation, the monkeys were placed in primate restraining chairs modified to allow tilting to a supine position. The monkeys were permitted 3–5 days to recover from surgery.

The monkeys were fasted overnight before the experiment. On the day of the experiment, under methohexital sedation (30–60 mg), 20-gauge Teflon catheters were placed in the subarachnoid space via the L3–4 interspace. After insertion of the spinal catheters, the restraining chairs were tilted to a supine position and the monkeys placed in sound-protected booths. Room air with a 3–5 l/min flow of oxygen was continuously pumped into the booths to maintain Pa_{O_2} around 80–120 mm Hg. All catheters were brought outside the booths and connected to Statham strain gauges placed at mid-thoracic levels; thus, all measurements, infusions, and blood sampling could be performed without disturbing the animal. After sufficient time for recovery from methohexital (1–1½ hours), baseline measurements were carried out. Arterial, central venous, and left ventricular pressures were continuously recorded on a Sanborn 150 recorder. Cardiac outputs were determined in duplicate by the indicator dye-dilution technique. Indocyanine green dye was injected into the left ventricle; the blood was withdrawn at a constant rate from the catheter placed in the abdominal aorta and passed through a Gilford densitometer, and output curves were recorded on a Gilford recorder. All blood was returned to the animal after the dye-dilution curve had been obtained. Immediately after each cardiac output determination and injection of radioactive microspheres, arterial blood was analyzed for *p*H, P_{CO_2} and P_{O_2}. Rectal temperature was continuously monitored with a Yellow Springs thermistor probe.

After baseline measurements had been obtained, tetracaine without epinephrine was injected through the spinal catheter: 0.5–1.0 mg for low spinal anesthesia (sensory level approximately T10) and 2–4 mg for high spinal anesthesia (sensory level approximately T1). Five monkeys were studied at each level of anesthesia. Level of anesthesia was confirmed by response of the animal to towel-clip application to consecutive dermatomes and correlated with decrease in arterial blood pressure and paralysis of lower extremity; in high-level blocks, upper-extremity weakness was the end point. Four subsequent sets of blood flow measurements were made 10, 20, 40, and 80 minutes after tetracaine had been injected. No fluid was administered other than that used in flushing the catheters between measurements.

The distribution of blood flow to various organs was determined at each time interval by injecting a suspension of one of the five gamma-emitting nuclide-labelled microspheres (50 microns in diameter) into the left ventricle after each cardiac output determination. The suspensions containing 5,000–15,000 microspheres are mixed with blood in the left ventricle and are distributed to each organ in proportion to its blood flow. The microspheres are trapped in organ arterioles. Validation studies[8] have estimated that only about 0.1 per cent of arteriolar tree is embolized per infusion, without significant effect on systemic cardiovascular dynamics. The following nuclide labels were used: ^{46}scandium, ^{95}niobium, ^{85}strontium, ^{51}chromium, and ^{141}cerium.

At the end of the experiment, the animals were exsanguinated under sodium thiopental anesthesia and the organs and tissues removed, weighed to 0.1 g, and placed in plastic vials. Radioactivity in each vial containing part or all of the organ was measured in a Packard NaI scintillation counter. All of the tissues from the major organs were counted to determine the radioactivity in these organs. However, only 20 per cent representative samples of skin, muscles, bones, and fat were counted, and these counts were multiplied by 5 to give the total counts for these tissues.

Energy distribution patterns were recorded on a pulse-height analyzer set to divide the output of the scintillation counter into 1,024 channels of 1 kev each. Since the five nuclides used in an experiment emitted

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TABLE 1. Systemic Hemodynamic Values during T10 Spinal Anesthesia in Five Monkeys (Mean ± SE)

	Control	10 Minutes	20 Minutes	40 Minutes	80 Minutes
Heart rate (beats/min)	167 ± 9.4	179 ± 17.4	170 ± 14.9	175 ± 9	160 ± 6.7
Mean arterial pressure (mm Hg)	109 ± 8	97 ± 10.7*	100 ± 7.2*	103 ± 8.1	107 ± 2.6
Cardiac output (ml/kg/min)	210 ± 22.9	222 ± 35.4	214 ± 29.8	222 ± 36.5	225 ± 34
Total peripheral resistance (mm Hg/l/min)	77.7 ± 12.7	68.1 ± 12.7	69.9 ± 8.5	70.8 ± 10.1	70.6 ± 10.8
*p*H	7.52 ± 0.01	7.52 ± 0.01	7.52 ± 0.02	7.51 ± 0.01	7.49 ± 0.01
P_{O_2} (mm Hg)	95 ± 7	96 ± 9	95 ± 6	99 ± 5	102 ± 11
P_{CO_2} (mm Hg)	37 ± 2	37 ± 2	36 ± 3	36 ± 2	37 ± 4

* $P < 0.05$.

gamma rays at different but definite energy levels in the 0–1,000-kev range, the amount of radioactivity from each isotope in each vial could be determined. The count from each channel of the pulse-height analyzer was transferred to magnetic tape and the composite emission spectrum from each vial, representing five isotopes, was processed by PDP-15 digital computer to give individual counts of all five isotopes in each vial. After all organs and tissues had thus been counted and processed to give the amount of each of the five nuclides present, the total body count of each nuclide was derived by summation of counts of each nuclide from individual organs and tissues. The percentage of cardiac output to each organ was calculated as the amount of radioactivity of each nuclide in that organ divided by the total body count of that nuclide. Flow to each organ was the percentage of cardiac output times the cardiac output calculated from the dye-dilution curves obtained immediately prior to injection of that particular nuclide. Right-leg

TABLE 2. Systemic Hemodynamic Values during T1 Spinal Anesthesia in Five Monkeys (Mean ± SE)

	Control	10 Minutes	20 Minutes	40 Minutes	80 Minutes
Heart rate (beats/min)	205 ± 13.1	192 ± 15.8	176 ± 16.9	179 ± 16.8	187 ± 12
Mean arterial pressure (mm Hg)	114 ± 6.1	90 ± 9†	88 ± 8.5*	88 ± 8*	102 ± 7.9
Cardiac output (ml/kg/min)	268 ± 25.8	251 ± 29.3	216 ± 27.1*	209 ± 23.8†	215 ± 14
Total peripheral resistance (mm Hg/l/min)	67.5 ± 8.1	55.9 ± 4.9	62.3 ± 6.5	66.7 ± 6.2	73.7 ± 7.1
*p*H	7.55 ± 0.02	7.58 ± 0.02	7.56 ± 0.02	7.55 ± 0.03	7.52 ± 0.02
P_{O_2} (mm Hg)	82.4 ± 5.9	85.2 ± 8.3	90.4 ± 3.9	99.4 ± 7.4	98 ± 10.7
P_{CO_2} (mm Hg)	36.7 ± 2.38	36.4 ± 3.12	36.9 ± 2.98	37.4 ± 4.47	39.2 ± 4.14

* $P < 0.05$.
† $P < 0.01$.

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blood flow was taken as indicative of lower-extremity blood flow, since the left leg had been rendered ischemic in the process of placing the arterial catheter.

The baseline regional blood flow to each organ was expressed as the percentage of cardiac output received by that organ and as the absolute blood flow through it (ml blood/100 g tissue/min). The changes in systemic hemodynamic measurements and the changes in regional blood flow values in each monkey were compared with the baseline values for the same animal, using Student's t test for paired observations. Changes were considered significant when P was less than 0.05.

Results

The systemic hemodynamic values and pH, P_{O_2} and P_{CO_2} of arterial blood during low (T10) and high (T1) spinal anesthesia are shown in tables 1 and 2 and figure 1. Hematocrits ranged from 30 to 40 per cent. Since each animal served as its own control, the effect of any significant variation in hematocrits among the animals was minimized. During T10 spinal anesthesia, the only significant changes were an 11 per cent decrease in mean arterial pressure (MAP) 10 minutes after the injection of tetracaine and an 8 per cent decrease at 20 minutes.

During T1 spinal anesthesia a significant 22 per cent reduction in MAP, lasting 40 minutes, and a significant 20 per cent reduction in cardiac output (CO) at 20 and 40 minutes were found. Total peripheral resistance (TPR) showed no significant change.

Regional-blood-flow alterations during T10 spinal anesthesia were minimal and not consistent (table 3 and figs. 2 and 3). Absolute blood flow to the major organs was not altered significantly. Blood flow to the lungs (bronchial artery) showed only initial significant increases.

Regional blood flow values during T1 spinal anesthesia are shown in table 4 and figures 2 and 3. There was no significant alteration in blood flow to the heart or brain throughout T1 spinal anesthesia. Twenty minutes after the injection of tetracaine, kidneys, liver, and carcass showed significant

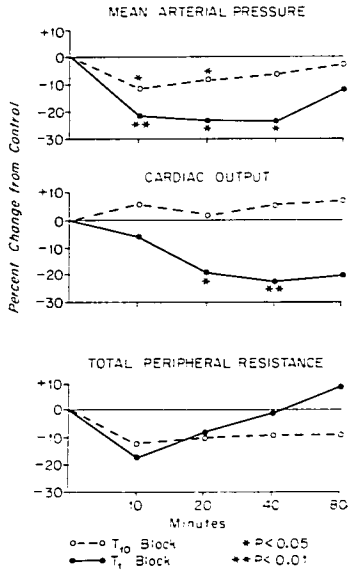

FIG. 1. The effects of T10 and T1 spinal anesthesia on mean arterial pressure, cardiac output, and total peripheral resistance. The values at each time interval are the mean percentage changes from control of the mean from five monkeys. Statistical significance compared with control is indicated at the bottom.

reductions in absolute blood flow, while the percentages of CO received by these organs were not altered consistently. Both the percentage of CO and absolute blood flow to lungs (bronchial artery) were increased significantly at 10, 20 and 40 minutes. The right leg (non-ischemic) received a significantly increased percentage of the CO, but absolute blood flow to the right leg showed only non-significant increases. Organs and tissues grouped under "miscellaneous" (testes, penis, bladder, salivary glands, thyroid, adrenals, major vessels and nerves, trachea, esophagus, tongue, fat, lymph nodes, etc.) received significantly reduced percentages of the CO and absolute blood flow throughout spinal anesthesia.

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Regional vascular resistances per 100 g tissue for major organs and right leg are shown in table 3. The significant changes were a decrease in renal vascular resistance during high spinal anesthesia at 10 minutes and decreases in the vascular resistance in the right leg during both T10 and T1 spinal anesthesia.

Discussion

In designing the experiment, the main consideration was to simulate as closely as possible the induction and course of spinal anesthesia as usually administered to human patients in the clinical setting. Nonhuman primates were chosen for the study because of their close phylogenetic and physiologic resemblance to man.[9,10] The animals were awake and unpremedicated and, though restrained, were not overly excited. Marked variations in rectal temperature, hematocrit, and aterial blood *p*H, P_{CO_2} and P_{O_2} did not occur except in one animal whose P_{CO_2} values ranged from 21 to 30 mm Hg and *p*H from 7.58 to 7.68. The seemingly alkalotic *p*H values in other animals with near-normal P_{CO_2} values in awake restrained monkeys have been reported before.[7,11]

TABLE 3. Regional Blood Flow during T10 Spinal Anesthesia in Five Monkeys (Mean ± SE)

	Control	10 Minutes	20 Minutes	40 Minutes	80 Minutes
Heart					
Per cent CO	4.9 ± 0.4	4.9 ± 0.4	5.4 ± 0.5	5 ± 0.5	4 ± 0.5
Flow/100 g/min	297 ± 54.4	312 ± 77.9	309 ± 29.3	302 ± 48.2	256 ± 57.6
Brain					
Per cent CO	3.6 ± 0.22	3.5 ± 0.23	3.4 ± 0.27	3.6 ± 0.23	3.3 ± 0.36
Flow/100 g/min	61 ± 9.9	64 ± 15	59 ± 11.9	65 ± 14.9	60 ± 8.9
Kidneys					
Per cent CO	13.9 ± 1.23	12.4 ± 1.03*	12 ± 1.11	11.9 ± 1.25	11.1 ± 0.98
Flow/100 g/min	687 ± 98.2	632 ± 99.1	574 ± 46.5	596 ± 84.7	558 ± 24
Liver (hepatic artery and portal vein‡)					
Per cent CO	22 ± 3.3	20.2 ± 3.9	20.3 ± 4	21 ± 3.8	22.4 ± 4.5
Flow/100 g/min	200 ± 34.4	203 ± 56	193 ± 49.6	212 ± 59	221 ± 61.1
Lungs					
Per cent CO	0.6 ± 0.07	1.61 ± 0.27*	1.32 ± 0.38	1.13 ± 0.29	0.55 ± 0.15
Flow/100 g/min	24 ± 2.9	64 ± 3.9†	47 ± 10.6	40 ± 6.8	21 ± 3.3
Right leg (nonischemic)					
Per cent CO	4.7 ± 0.71	6.9 ± 1.9	6.9 ± 1.68	6.3 ± 1.48	5.4 ± 1.26
Flow/100 g/min	8 ± 0.7	10 ± 0.7	11 ± 1.6	10 ± 1.4	10 ± 0.6
Carcass§					
Per cent CO	34 ± 2.76	34.1 ± 2.88	35.1 ± 1.78	34.6 ± 2.57	37.9 ± 2.15
Flow/100 g/min	15 ± 1.9	16 ± 2.1	16 ± 2.6	16 ± 2.5	18 ± 1.8
Miscellaneous¶					
Per cent CO	11.8 ± 0.95	11.4 ± 1.5	10.6 ± 1.2	11.8 ± 1.4	10.4 ± 1.0
Flow/100 g/min	38 ± 4.9	39 ± 8.2	34 ± 4.6	40 ± 7.2	35 ± 6.4

* $P < 0.05$.
† $P < 0.01$.
‡ Portal vein flow is the sum of blood flows to GI tract, mesentery, pancreas and spleen.
§ Carcass includes spinal cord and all skin, muscles and bones other than from right and left legs.
¶ Miscellaneous includes testes, penis, bladder, salivary glands, thyroid, adrenals, major vessels and nerves, trachea, esophagus, tongue, fat, lymph nodes, etc.

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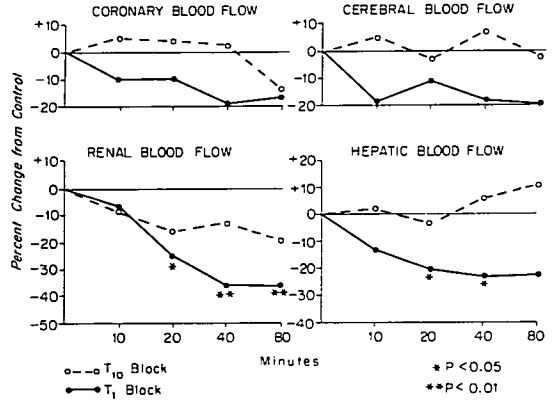

FIG. 2. The effects of T10 and T1 spinal anesthesia on coronary, cerebral, hepatic, and renal blood flows. The values at each time interval are the mean percentage changes from control of the mean from five monkeys. Blood flow is expressed as flow in ml/100 g of tissue/min. Statistical significance compared with control is indicated at the bottom.

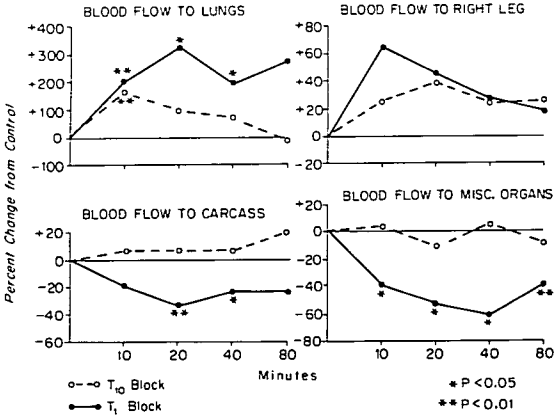

FIG. 3. The effects of T10 and T1 spinal anesthesia on blood flow to the lungs, right leg, carcass, and miscellaneous organs. The values at each time interval are mean percentage changes from control of the mean from five monkeys. Blood flow is expressed as flow in ml/100 g of tissue/min. Statistical significance compared with control is indicated at the bottom.

SYSTEMIC HEMODYNAMICS

The hemodynamic values presented in tables 1 and 2 are in concurrence with those found in previous studies in man.[5,12–16] As has been observed earlier by Sancetta *et al.*[15] and Stevens *et al.*,[16] we found greater changes in CO and MAP with higher levels of block. The only finding of ours that is not consonant with the majority of previous reports is the total peripheral resistance (TPR). While numerous workers[12,13,15,17,18] have found TPR to decrease significantly, a few studies[19–21] have reported only non-significant changes in TPR during spinal anesthesia. In our studies, we found only non-significant decreases in TPR during both levels of anesthesia.

Since TPR reflects the sum total of the resistance in entire systemic circulation, it is

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TABLE 4. Regional Blood Flow during T1 Spinal Anesthesia in Five Monkeys (Mean ± SE)

	Control	10 Minutes	20 Minutes	40 Minutes	80 Minutes
Heart					
Per cent CO	5.1 ± 0.63	4.9 ± 0.67	5.8 ± 0.65	5.2 ± 0.51	5.3 ± 0.72
Flow/100 g/min	384 ± 48.7	346 ± 49.5	348 ± 56.4	310 ± 51.2	318 ± 38.9
Brain					
Per cent CO	3.7 ± 0.57	3.2 ± 0.39	4.1 ± 0.57	3.9 ± 0.65	3.8 ± 0.73
Flow/100 g/min	71 ± 11.9	58 ± 5.4	63 ± 6.1	58 ± 7.2	57 ± 8.0
Kidneys					
Per cent CO	15.5 ± 1.36	16.0 ± 2.46	14.6 ± 1.12	13.0 ± 1.38	12.3 ± 1.03*
Flow/100 g/min	960 ± 73.3	896 ± 64.7	717 ± 53*	614 ± 19.9†	611 ± 21.2†
Liver (hepatic artery and portal vein‡)					
Per cent CO	19.7 ± 1.41	18.5 ± 1.38	20 ± 1.39	19.9 ± 1.89	19.5 ± 1.94
Flow/100 g/min	207 ± 20.6	180 ± 16.9	166 ± 18.7*	160 ± 16.3*	162 ± 12.7
Lungs					
Per cent CO	0.5 ± 0.15	1.7 ± 0.33†	2.9 ± 0.96*	1.9 ± 0.54*	2.3 ± 1.22
Flow/100 g/min	21 ± 58	64 ± 9.6†	89 ± 23*	62 ± 13.3*	78 ± 39.2
Right leg (nonischemic)					
Per cent CO	5.31 ± 0.8	8.4 ± 0.99*	8.8 ± 0.8*	8.1 ± 0.66*	7.43 ± 0.91
Flow/100 g/min	11 ± 1.6	18 ± 3.9	16 ± 2.6	14 ± 2.1	13 ± 1.8
Carcass§					
Per cent CO	37.1 ± 1.3	34.1 ± 2.8	31.7 ± 0.85*	36 ± 2.7	35.6 ± 3.4
Flow/100 g/min	21 ± 2.2	17 ± 2.4	14 ± 1.7†	16 ± 2.1*	16 ± 1.6
Miscellaneous¶					
Per cent CO	10.7 ± 1.2	6.3 ± 0.6*	5.8 ± 0.8*	5.3 ± 0.6*	7.6 ± 0.4*
Flow/100 g/min	57 ± 12.1	35 ± 12.8*	27 ± 7.7*	22 ± 4.2*	35 ± 8.4†

* $P < 0.05$.
† $P < 0.01$.
‡ Portal vein flow is the sum of blood flows to GI tract, mesentery, pancreas and spleen.
§ Carcass includes spinal cord and all skin, muscles and bones other than from right and left legs.
¶ Miscellaneous includes testes, penis, bladder, salivary glands, thyroid, adrenals, major vessels and nerves, trachea, esophagus, tongue, fat, lymph nodes, etc.

conceivable that during T10 spinal anesthesia vasoconstriction above the level of the block could have compensated for the vasodilation in the sympathetically denervated portions of the body, resulting in little or no change in TPR. However, very little compensatory vasoconstriction could have occurred during T1 spinal anesthesia by which we had achieved total sympathetic blockade. It can be postulated that the marked 22 per cent decrease in MAP could have activated the renin–angiotensin mechanism in the kidneys. Although renin release from the kidneys is known to be modulated by sympathetic activity, renin release can occur in response to lowered MAP even in nonsecreting, denervated kidneys in adrenalectomized dogs.[22] Angiotensin has a direct vasoconstrictive effect on the denervated arteriole.[23] This could account for the nonsignificant changes in TPR during T1 spinal anesthesia. Plasma renin–angiotensin activity during hypotension produced by spinal anesthesia has not been measured before. Work in this area is indicated.

REGIONAL BLOOD FLOW: T_{10} SPINAL ANESTHESIA

With the exception of the work of Mueller,[5] not many studies have been done on the

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TABLE 5. Regional Vascular Resistance during T10 and T1 Spinal Anesthesia

	Control	10 Minutes	20 Minutes	40 Minutes	80 Minutes
Heart					
T10	0.4 ± 0.08	0.36 ± 0.08	0.31 ± 0.02	0.37 ± 0.04	0.47 ± 0.12
T1	0.31 ± 0.07	0.26 ± 0.03	0.24 ± 0.03	0.28 ± 0.03	0.49 ± 0.18
Brain					
T10	2.0 ± 0.46	1.88 ± 0.51	1.96 ± 0.4	1.87 ± 0.41	1.86 ± 0.31
T1	1.66 ± 0.23	1.49 ± 0.15	1.36 ± 0.21	1.48 ± 0.18	1.76 ± 0.25
Kidneys					
T10	0.16 ± 0.03	0.15 ± 0.03	0.16 ± 0.01	0.17 ± 0.02	0.18 ± 0.01
T1	0.11 ± 0.01	0.09 ± 0.01*	0.11 ± 0.01	0.13 ± 0.01	0.15 ± 0.01
Liver					
T10	0.59 ± 0.11	0.63 ± 0.19	0.61 ± 0.12	0.61 ± 0.14	0.56 ± 0.11
T1	0.54 ± 0.07	0.47 ± 0.05	0.5 ± 0.05	0.51 ± 0.04	0.59 ± 0.06
Right leg					
T10	13.6 ± 0.42	9.13 ± 1.15*	9.05 ± 0.89*	10.65 ± 1.72	11.07 ± 0.9
T1	10.58 ± 1.64	5.25 ± 0.56*	5.59 ± 0.59*	6.26 ± 0.52	8.16 ± 1.77

Data represents means ± SE from five monkeys for each level of anesthesia. Peripheral vascular resistance is expressed as mm Hg/ml/100 g of tissue/min.
* $P < 0.05$.

effect of low spinal anesthesia on organ blood flow. Using BSP clearance, Mueller found an 18 per cent decrease in total hepatic blood flow in man during low spinal anesthesia. We did not observe such changes. While splanchnic sympathetic stimulation in dogs decreases hepatic-artery flow[24] and splanchnic denervation increases hepatic-artery flow,[25] the total hepatic blood flow (sum of hepatic-artery and portal-vein flows) is dependent on MAP.[25,26] This may in part explain the differences between Mueller's and our findings. Mueller's group of five patients hospitalized for various illnesses (fatty liver, chronic alcoholism, cerebral thrombosis, etc.) showed a greater decrease of 22 per cent in MAP,[5] as opposed to the 11 per cent decrease in our experiments. It should be mentioned that portal-vein flow in our studies was computed as the sum of arterial flows to the GI tract, mesentery, pancreas and spleen, since the microsphere technique measures only arterial flow directly. The microspheres do not enter the portal circulation, as a result of entrapment in the arteriolar and capillary bed.

In view of the dearth of earlier studies of organ blood flow during low spinal anesthesia, we conclude that a slight 11 per cent reduction in MAP without significant alteration in CO was insufficient to cause changes in blood flow to any of the major organs. The observed changes in blood flows to the lungs (bronchial artery) and right leg are discussed below.

REGIONAL BLOOD FLOW: T, SPINAL ANESTHESIA

In dogs Eckenhoff *et al.*[2] and in man Hackel *et al.*[27] found coronary blood flow to be consistently decreased coincident with the decrease in blood pressure following high spinal anesthesia. Our findings were not that consistent; two of the monkeys showed increases in coronary blood flow, while three showed decreases, and the mean values were not statistically significant. However, myocardial minute work, calculated as the product of MAP and CO, was significantly decreased in all our animals. Even in the three animals that showed decreases in coronary blood flow, myocardial minute work was decreased more than the decrease in coronary blood flow (table 6). This agrees with the finding of Eckenhoff *et al.*[2] and Hackel *et al.*[27] and indicates adequate perfusion relative to work load on the myocardium.

Kety and co-workers[3,28] and Kleinerman *et*

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TABLE 6. Coronary Blood Flow vs. Cardiac Work during T1 Spinal Anesthesia in Three Monkeys That Had Decreases in Coronary Blood Flow

	10 Minutes	20 Minutes	40 Minutes	80 Minutes
Mean change in flow/100 g (per cent)	−25	−29	−35	−32
Mean change in cardiac work/min (per cent)	−29	−39	−43	−25

al.[4] found 12 and 17 per cent decreases in cerebral blood flow, respectively, following high spinal anesthesia in hypertensive human subjects. Kleinerman[4] also reported that normotensive subjects did not show a decrease in cerebral blood flow following spinal hypotension since they were able to compensate by a proportionate decrease in cerebrovascular resistance. This autoregulatory phenomenon was seen in four of our five monkeys in which cerebral blood flow was maintained near control levels with significant reductions in cerebrovascular resistance. The one animal that showed a decrease in cerebral blood flow and a marked increase in resistance also had abnormally low Pa_{CO_2} and *p*H values, ranging from 21 to 30 mm Hg and 7.58 to 7.68, respectively. Moreover, this hypocarbic monkey had marked changes in cerebral blood flow and resistance (40–60 per cent) with small fluctuations in Pa_{CO_2} (3–5 mm Hg). Galindo,[29] while studying the effect of epidural sympathetic block and hypercarbia on internal carotid flow, suggested that total sympathetic block could decrease the response of the cerebral vessels to hypercarbia. It might be worthwhile to investigate whether total sympathetic block can increase the sensitivity of the cerebral vessels to hypocarbia.

Smith *et al.*[1] and Kennedy *et al.*[17] observed decreases in renal plasma flow of 4 and 7 per cent, respectively, during high spinal anesthesia, with concomitant decreases in MAP of 14 and 18 per cent, respectively. The decreases in renal plasma flow in both of the above-mentioned studies were not significant. Kennedy[17] also reported a significant decrease of 10 per cent in renal blood flow during the experiment, as a result of decreasing hematocrit due to mannitol infusion and significant decreases in renal vascular resistance. We observed much greater decreases of as much as 36 per cent in renal blood flow following induction of high spinal anesthesia. Renal vascular resistance showed a significant decrease at 10 minutes, when there was little change in renal blood flow, and thereafter showed only non-significant increases. The difference between our findings and those of Smith *et al.*[1] and Kennedy *et al.*[17] is perhaps due to the higher level of block (T1 as opposed to T5) and a greater decrease in MAP (22 per cent as opposed to 14 and 18 per cent) in our monkeys. Moreover, in both of the above-mentioned studies,[1,17] osmotic diuretics were administered to the subjects to ensure adequate urine flow. Osmotic diuretics increase extracellular fluid volume and have been shown to increase renal blood flow,[30] so that any decrease in renal blood flow that might have occurred as a consequence of spinal anesthesia could have been effectively abolished. Assali *et al.*,[31] however, observed decreases of as much as 60 per cent in MAP and renal plasma flow, but the study was done in "hydropenic" pregnant women in the third trimester of pregnancy who had been fluid-restricted for 16–20 hours before the experiment. Renal blood flow is well autoregulated over a range of perfusion pressures from 80 to 180 mm Hg,[32] but there appears to be a threshold MAP below which renal blood flow is decreased.[33] Our study shows that decreases in mean arterial pressure to an extent that can affect renal autoregulation can occur during high spinal anesthesia under normovolemic conditions.

As mentioned earlier, hepatic blood flow is dependent on MAP.[25,26] With the 22 per cent decrease in MAP we observed a parallel decrease of 23 per cent in total hepatic blood flow. Splanchnic vascular resistance was not altered significantly. All these findings have been reported earlier.[5,18,24]

One of the most consistent findings in the study was the increased radioactivity found in the lung tissue following induction of both low and high spinal anesthesia. Since this technique measures only arterial and not

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venous flows, we could not account for significant increases in bronchial-artery blood flow. Forsyth *et al.*,[7] in their validation studies, found that a small fraction (0.3 per cent) of microspheres did indeed pass through the systemic arteriovenous network when they were injected beyond an occlusive clamp placed distal to the origin of the bronchial arteries. Greene,[35] based on the findings of increased oxygen saturation of femoral venous blood and decreased arterial–femoral vein oxygen difference during spinal anesthesia,[36,37] postulates that as a result of arteriolar dilatation, many true anatomic arteriovenous shunts may open up. The microspheres could presumably slip through these arteriovenous shunts and return to the lungs, lodging in the pulmonary capillary bed. This could account for the increased radioactivity we found in the lungs. Similar results were observed by Amory *et al.*[38] in monkeys during halothane anesthesia, which also produces arteriolar dilatation.

The capacity of peripheral vascular bed and blood flow in the peripheral vascular bed are known to be increased following spinal anesthesia,[20,35] but in our studies we found only non-significant increases in the blood flow to the right leg. Again, a large number of open arteriovenous shunts in the periphery, through which microspheres could pass into the venous circulation, would explain the lower than expected counts of radioactivity in the right leg and hence, only non-significant increases in the blood flow. Vascular resistance was significantly decreased in the right leg, indicating arteriolar dilatation.

The significant decreases in the percentages of CO and absolute blood flows to carcass and miscellaneous organs and tissues during T1 spinal anesthesia indicate diversion of the CO in an attempt to maintain blood flow to the vital organs.

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