

# *The Role of the Venous System in Cardiocirculatory Dynamics during Spinal and Epidural Anesthesia in Man*

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Effects of spinal and epidural anesthesia uncomplicated by premedication or surgery were studied in 20 normal subjects. Venous blood flow, vascular resistance and venous distensibility of forearm and calf vessels were measured simultaneously before and after induction of spinal or epidural anesthesia. In anesthetized calf vessels, blood flow and vascular distensibility increased and vascular resistance decreased. Changes in cardiac output, whether decreased, increased or unchanged, did not affect the ratio of calf blood flow to cardiac output, which in all instances increased following spinal and epidural anesthesia. Changes in arterial blood pressure did not correlate with changes in cardiac output alone or changes in total peripheral resistance alone. However, changes in arterial blood pressure correlated with the interaction of changes in cardiac output and changes in total peripheral resistance.

Evidence presented indicates that arterial hypotension induced by either spinal or epidural anesthesia may be due primarily to increased vascular distensibility of capacitance vessels and secondarily to decreased resistivity in pre- and postcapillary resistance vessels in the anesthetized area.

AN INFLUENCE of the venous system on hemodynamic changes during spinal anesthesia has been suggested by several studies.<sup>1-4</sup> The systemic venous system may be analyzed as an

active vascular component<sup>5</sup> consisting of pre- and post-capillary resistance vessels and capacitance vessels.<sup>6</sup> The venous system reacts to both neurogenic and humoral stimuli. Changes in the venous system profoundly alter cardiac output and influence arterial blood pressure by controlling the rate at which blood is delivered to the systemic arterial bed.<sup>7</sup> Reduction of peripheral venous pressure occurs in man during high spinal anesthesia, irrespective of changes in arterial blood pressure.<sup>2</sup> Spinal anesthesia causes vasodilation in the anesthetized toe, with concomitant vasoconstriction in unanesthetized fingers.<sup>1, 3, 4</sup>

Most of the reported studies describing the effects of spinal anesthesia on the circulation in man have centered primarily on changes in arterial blood pressure, cardiac output and hemodynamics.<sup>8-11</sup> Several investigators believe that the decrease in cardiac output is the primary cause of hypotension, and that the change in total peripheral resistance plays an insignificant role.<sup>10, 11</sup> Other investigators report that subarachnoid block produces hypotension with reduction in cardiac output, stroke volume and total peripheral resistance,<sup>1, 12</sup> whereas epidural block with epinephrine in the anesthetic solution produces the same degree of hypotension, with increases in heart rate, stroke volume and cardiac output.<sup>12</sup>

The aim of the present study was to provide more definitive information as to whether spinal and epidural anesthesia induce changes in the venous circuit; if so, whether the effect is related to changes in arterial hemodynamics.

## Procedures

Experiments were performed prior to surgical operations on five patients between 22 and 61 years of age, and on 15 normal volunteers

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between 21 and 41 years of age.\* Each subject served as his own control. Fifteen subjects received spinal anesthesia and five, epidural anesthesia. Two subjects were studied twice, a week apart: in the first study, changes in arterial blood pressure were minimized during subarachnoid block by slow induction of anesthesia (20 and 30 min); in the second, blood pressure was lowered during the rapid onset (4 and 8 min) of an equivalent height of sensory anesthesia.

Neither preanesthetic drugs nor prophylactic vasopressors were used. The subjects were placed in the supine position in a quiet room at a temperature of 25 C. Catheter spinal and epidural anesthetic techniques have been described in detail.<sup>13</sup> The subjects were placed in the supine position with the wrist and elbow, ankle and lower portion of the thigh supported on foam rubber pads. Whitney strain gauges<sup>14</sup> were mounted on the middle portion of forearm and calf, using a maximum tension of less than 25 g.

After insertion of the spinal or epidural catheter, plethysmographs and ECG leads, at least 30 minutes were permitted to elapse to attain a resting state. Control observations ( $C_1$ ) were made when the end-expiratory carbon dioxide tension reached normal values (36–40 mm Hg). In ten studies, the second control observations ( $C_2$ ) were repeated following an average of 22 minutes (range 12 to 36 min) from the first control observations. In one subject observations were repeated over a three-hour period without administration of anesthesia, and in a second subject, measurements of arterial and venous dynamics were made following the injection of physiologic saline solution through the spinal catheter. The control studies were made to evaluate the status of venous and arterial circuits during

simulated study conditions, since it has been shown that the venous system responds readily to any reflex activation.<sup>7</sup>

A test dose of 6 mg of 0.3 per cent tetracaine was administered for spinal anesthesia; a dose of 30 mg of 1.5 per cent lidocaine for epidural anesthesia. Subsequent doses of tetracaine (average total dose per surface area: 0.9 mg/m<sup>2</sup>) or lidocaine (339 mg/m<sup>2</sup>) were administered until the sensory level was established between T4 and T7. Measurements of venous and arterial function were repeated 20 and/or 30 minutes after the onset of spinal and epidural anesthesia and were presented as average values.

### Measuring Techniques

A venous occlusion plethysmograph with a Whitney strain gauge<sup>14</sup> was used for measurement of forearm and calf blood flow and estimation of vascular distensibility. Sphygmomanometric cuffs, 4 cm wide, were placed around the wrist and ankle and inflated to levels exceeding systolic arterial pressure before venous occlusion to arrest the circulation during measurements. Collecting cuffs, 11.5 cm wide, applied above the elbow and knee, were inflated to levels below diastolic systemic arterial pressure. The desired pressures in the collecting cuffs were reached in less than a second by opening the toggle valve connected to a reservoir of compressed air.

The Whitney strain gauge was connected to a Sanborn carrier preamplifier (Model 350-1100 B) through the impedance-matching circuit described by Elsner *et al.*,<sup>15</sup> with a temperature compensator as described by Honda.<sup>16</sup> Static calibration of the gauge was performed using a method described previously.<sup>17</sup> The correlation coefficient between changes in volume in one arm determined by this method and those in the other arm determined by the conventional water-filled plethysmograph<sup>18</sup> was +0.85 for 83 simultaneous determinations of the two values in six normal subjects. Peripheral blood flow was deduced from the rate of change in forearm or calf volume during rapid venous occlusion.<sup>14</sup> Ten sets of blood flow measurements were made at each point of determination and average values calculated.

\* The study was conducted in accordance with the fundamental ethical principles set forth in the 1964 Declaration of Helsinki and the Surgeon General's memorandum of July, 1966. Voluntary consent was obtained in writing from each subject after the disclosure of information concerning the nature and risk of the experimental procedure and administration of spinal or epidural anesthesia. Subjects were admitted to the clinical study unit at least one day prior to study. A complete physical examination and routine laboratory tests including chest x-ray were performed.

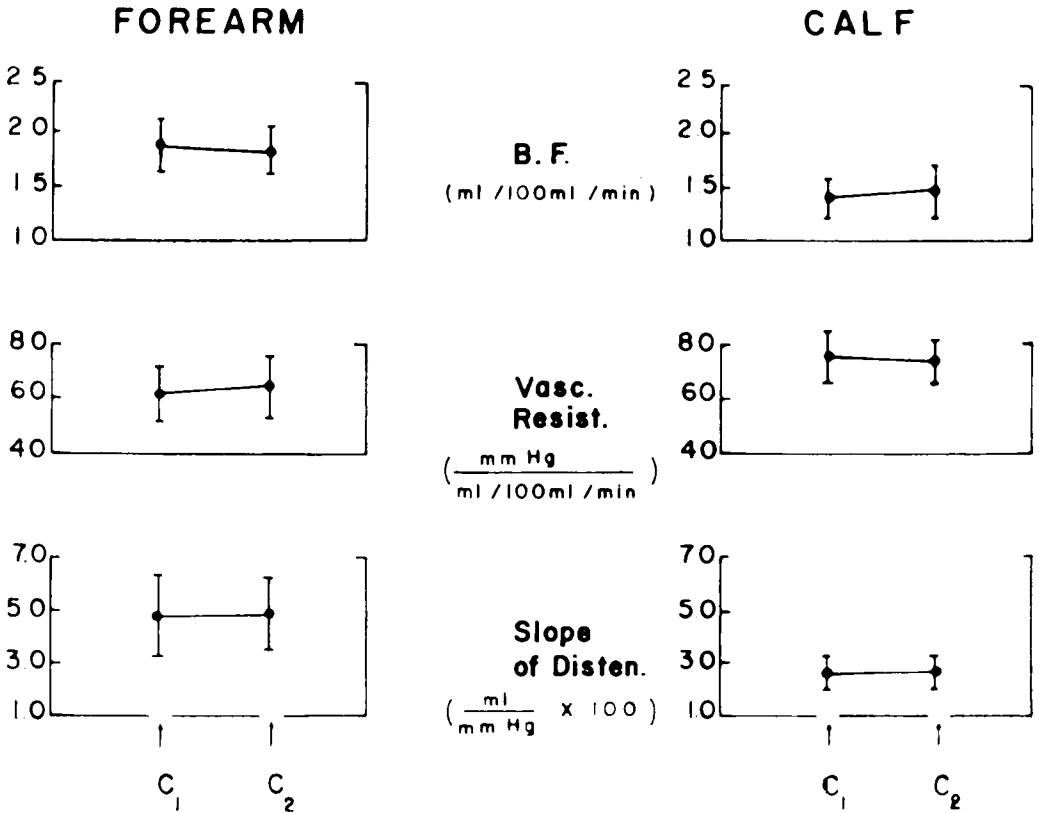


FIG. 1. Mean values of repeated determinations of blood flow, vascular resistance and slope of distensibility in forearm and calf vessels, obtained during the control period in ten subjects.  $C_1$  = the first control observations;  $C_2$  = the second control observations, made 22 minutes after  $C_1$ .

Vascular distensibility (pressure-volume curve of the extremity vessel) was determined according to the method of Wood and Eckstein.<sup>18</sup> Changes in distensibility were calculated as changes in slope of the pressure-volume curves.<sup>19</sup> Vascular resistance was calculated as the ratio of mean arterial pressure to extremity blood flow, expressed in mm Hg per ml/100 g/min.

Arterial blood pressure was measured with a Statham strain gauge (P23Db) through an indwelling teflon arterial needle placed into the brachial artery of the forearm opposite that in which venous blood flow was determined. In 14 experiments, cardiac output was determined by the indicator-dilution method using indocyanine dye.<sup>19</sup> The area under a dye-dilution curve was integrated at the same time as the output determination by means of an

online analogue computer (Sanborn 130).<sup>†</sup> Methods of calculating stroke volume index, total peripheral resistance and left ventricular work per minute have been described.<sup>13, 19</sup> Arterial blood was sampled periodically for  $P_{aCO_2}$ ,  $P_{aO_2}$ , and  $pH$  by methods previously described.<sup>21</sup> In eight subjects, plasma catecholamine concentrations were measured by a previously-described method<sup>22</sup> before and during spinal or epidural anesthesia. The data were analyzed by means of Fisher's  $t$  test and average values expressed as the mean value  $\pm$  SEM. The difference between the groups was considered statistically significant when  $P < 0.05$ .

<sup>†</sup> Evaluation of the computer revealed that the correlation coefficient relating the cardiac output value determined by the computer to the value obtained by manual computation for each curve was 0.999 for 119 cardiac outputs measured in four subjects.<sup>20</sup>

## Results

### CONTROL STUDIES

*Reproducibility.* Measurements of venous and arterial functions were repeated in each of ten subjects, with an average resting period of 22 minutes intervening.

Figure 1 summarizes average changes in blood flow, vascular resistance and distensibility of the forearm and calf vessels during the first control ( $C_1$ ) and second control ( $C_2$ ) periods. Mean values of the two control measurements for cardiac index, heart rate, stroke volume index, left ventricular minute work, mean arterial pressure, and total peripheral resistance are shown in figure 2. The average values of each variable of  $C_1$  did not differ from those of  $C_2$  ( $P > 0.4$  for each variable).

*Evaluation of the Effects of Study Time and Environment without Anesthesia.* Figure 3 illustrates a three-hour period of the stable course of the venous circuits and hemodynamics together with skin temperature in one sub-

ject following injection of 10 ml of physiologic saline solution into the spinal catheter. Changes in arterial and venous dynamics observed in the second control subject were the same as those described.

### SPINAL AND EPIDURAL ANESTHESIA

The direction and magnitude of changes in venous and arterial systems observed in subjects with spinal anesthesia were similar to those with epidural block. Hence, data were pooled and statistical analyses made. Table 1 summarizes mean values of blood flow, vascular resistance, and distensibility and the ratio of blood flow to cardiac output in anesthetized calf vessels and unanesthetized forearm vessels before and after (20 to 30 min) administration of spinal and epidural anesthesia in 20 subjects.

Changes in mean arterial pressure following anesthesia averaged  $-16$  per cent ( $P < 0.01$ ). In the anesthetized calf vessels, increases in blood flow, vascular distensibility and ratio of

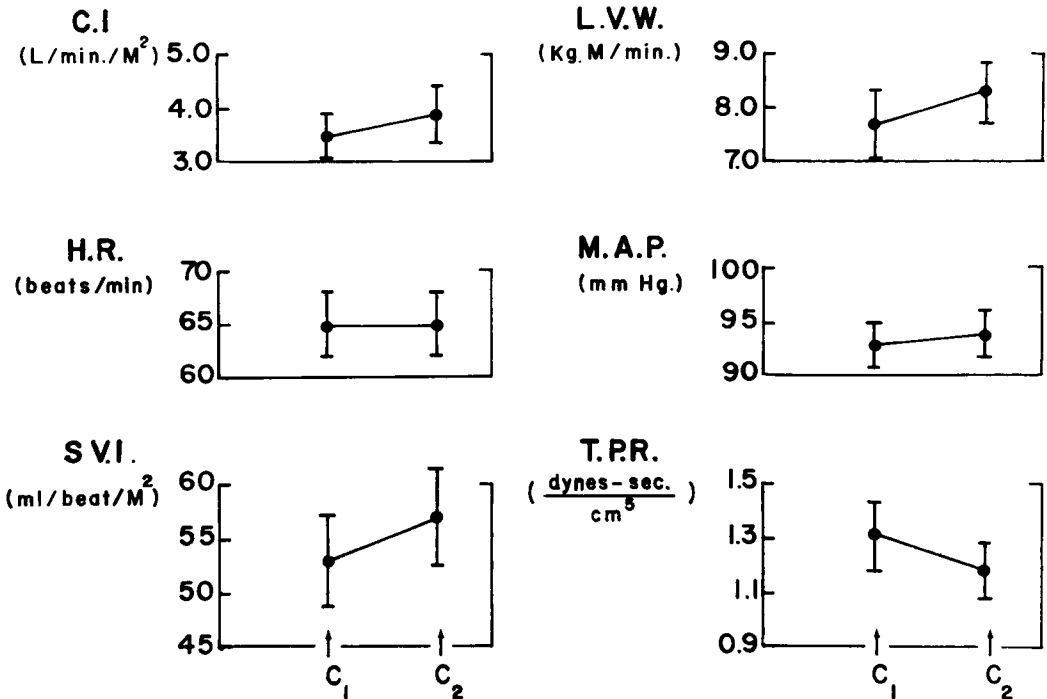


FIG. 2. Mean values of repeated determinations of hemodynamic parameters obtained during the control period in ten subjects. C.I. = cardiac index; H.R. = heart rate; S.V.I. = stroke volume index; M.A.P. = mean arterial blood pressure; L.V.W. = left ventricular minute work; T.P.R. = total peripheral resistance.

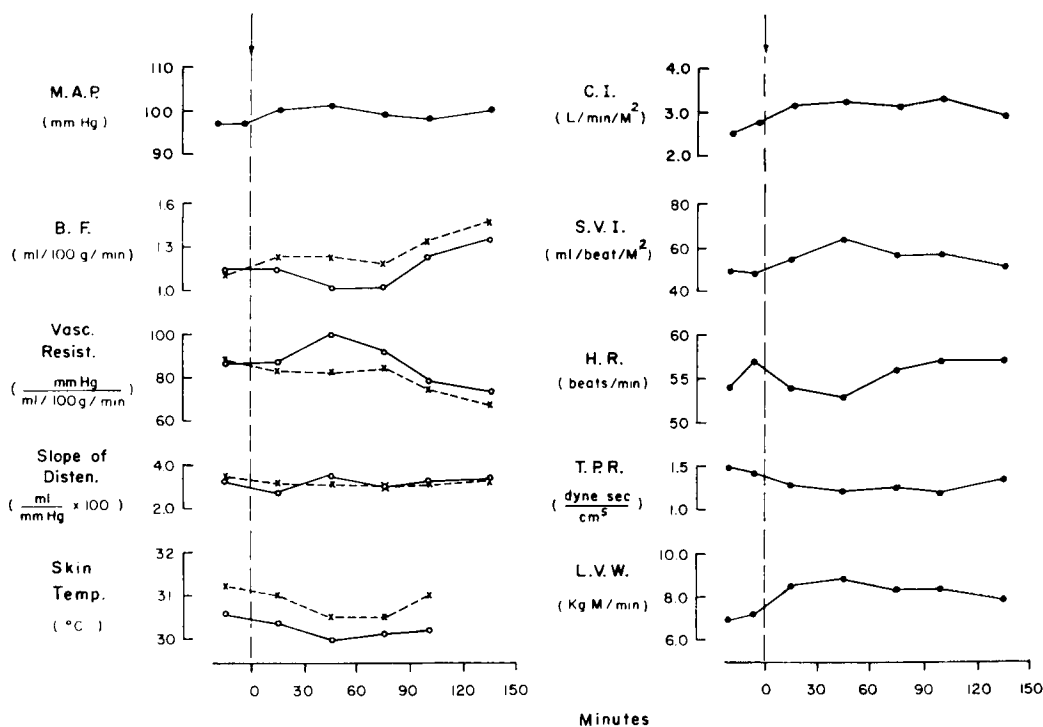


FIG. 3. *Left:* Time course of changes in mean arterial blood pressure (M.A.P.), blood flow (B.F.), vascular resistance (vasc. resist.) and slope of distensibility (disten.) and skin temperature (skin temp.), following placement of a spinal catheter in one subject. The arrow indicates the injection of 10 ml of physiologic saline solution to the catheter. O = values for forearm vessels; X = values for calf vessels. *Right:* Hemodynamics before and following the saline injection into the catheter. C.I. = cardiac index; S.V.I. = stroke volume index; H.R. = heart rate; T.P.R. = total peripheral resistance; L.V.W. = left ventricular work.

blood flow to cardiac output averaged +64 per cent ( $P < 0.01$ ), +17 per cent ( $P < 0.02$ ), and +77 per cent ( $P < 0.01$ ), respectively. Calf vascular resistance decreased, on the average, 46 per cent ( $P < 0.01$ ). In contrast, changes in these parameters in the unanesthetized forearm vessels were not significant (table 1).

During spinal and epidural anesthesia, percentage changes in arterial blood pressure did not correlate with those changes in cardiac output (correlation coefficient: +0.58). The mean values for cardiac index, stroke volume index, heart rate, and total peripheral resistance in 17 subjects during the control state did not differ significantly from those obtained during spinal and epidural anesthesia (table 2). Figure 4 shows that changes in venous and arterial dynamics during spinal anesthesia without hypotension (A) were essentially the

same as those during hypotensive spinal anesthesia (B) in the same subject studied twice on different days.

#### pH, PaCO<sub>2</sub>, PaO<sub>2</sub> AND END-EXPIRATORY P<sub>CO<sub>2</sub></sub>

Arterial pH was determined before and during spinal anesthesia in 15 subjects. During the control period arterial blood pH averaged  $7.42 \pm 0.01$ ; during spinal or epidural anesthesia it averaged  $7.42 \pm 0.01$  ( $P > 0.5$ ). PaCO<sub>2</sub> and PaO<sub>2</sub> averaged  $39.1 \pm 0.5$  mm Hg and  $102.5 \pm 1.7$  mm Hg before spinal anesthesia, and  $38.8 \pm 0.8$  mm Hg and  $107.8 \pm 4.2$  mm Hg after spinal anesthesia. There were no significant changes in pH, PaCO<sub>2</sub> and PaO<sub>2</sub> during epidural anesthesia. The average values for end-expiratory CO<sub>2</sub> tension measured by means of an infrared analyzer were  $37.7 \pm 1.0$  mm Hg during the control

TABLE 1. Changes in Arterial Pressure and Venous Functions of Calf and Forearm Vessels before and after Spinal and Epidural Anesthesia\*

Subject	Age, Sex	Calf Vessel										Forearm Vessel									
		MAP		B.F.		V.R.		V.D.		B.F./C.O.		B.F.		V.R.		V.D.		B.F./C.O.			
		C	A	C	A	C	A	C	A	C	A	C	A	C	A	C	A	C	A		
F. G.	40 F	75	69	1.31	1.63	57	42	2.1	3.0	—	—	1.78	1.47	42	47	4.4	4.0	—	—		
R. V.	45 M	83	77	1.34	3.41	62	23	3.2	5.4	—	—	3.96	3.23	21	24	8.5	7.0	—	—		
G. R.	61 M	96	83	1.56	1.99	62	49	2.7	2.0	0.26	0.42	3.11	1.59	31	55	6.5	3.8	0.53	0.30		
J. G.	33 M	93	82	0.89	2.04	106	40	2.5	3.2	0.21	0.43	1.86	2.09	51	40	9.8	8.9	0.44	0.45		
R. G.**	23 M	96	92	1.58	3.66	60	25	4.8	5.4	0.28	0.47	1.09	0.89	86	103	3.7	2.5	0.19	0.11		
R. H.**	27 M	90	80	1.00	2.20	90	36	1.9	2.8	0.17	0.49	0.84	0.73	107	110	3.7	2.6	0.14	0.16		
J. G.	27 M	98	98	1.40	1.92	70	51	3.0	—	0.30	0.41	7.97	4.47	12	22	6.5	9.1	2.87	0.96		
C. C.	41 M	67	64	2.30	2.67	29	25	3.4	4.4	0.39	0.46	5.00	3.42	13	20	7.0	6.6	0.85	0.43		
N. H.	27 M	92	82	1.93	2.32	48	36	3.1	2.9	0.24	0.42	2.47	3.03	37	27	3.7	4.2	0.31	0.55		
A. S.	24 M	89	73	1.30	1.69	69	44	3.1	3.0	0.18	0.29	2.67	2.20	33	34	5.1	6.2	0.36	0.38		
R. M.	23 M	103	87	1.73	4.10	59	23	3.9	4.0	0.25	0.56	2.40	3.44	42	29	4.7	4.3	0.34	0.66		
J. D.	25 M	92	54	1.31	1.37	70	38	3.0	3.0	0.28	0.43	1.82	2.13	51	24	3.4	2.8	0.38	0.67		
R. G.**	23 M	108	67	1.06	1.11	105	67	1.0	1.5	0.17	0.23	2.04	2.39	54	31	4.2	5.8	0.33	0.49		
R. H.**	27 M	91	62	0.94	2.28	99	27	2.9	3.9	0.16	0.41	0.68	1.13	137	56	2.5	5.0	0.12	0.21		
R. D.	24 M	90	67	2.11	3.23	42	22	4.2	5.5	0.31	0.89	3.70	4.30	24	17	7.6	6.2	0.54	1.19		
R. F.	25 M	90	74	1.00	1.34	90	55	2.9	2.7	—	—	1.97	1.07	46	70	6.8	3.5	—	—		
J. G.	23 M	92	104	0.92	2.66	100	40	3.3	3.6	0.16	0.40	1.49	1.23	62	87	8.2	5.1	0.27	0.18		
J. R.	23 M	96	75	2.57	4.12	40	20	4.8	7.5	0.47	0.74	1.68	1.03	61	74	6.2	3.8	0.31	0.13		
J. S.	37 M	103	79	1.45	1.19	71	71	2.0	1.5	0.29	0.44	1.27	1.52	81	58	2.9	3.7	0.25	0.56		
J. R.	21 M	101	70	1.10	2.33	92	30	2.2	1.9	0.15	0.26	3.66	3.54	28	20	7.2	7.4	0.50	0.39		
Mean		92	77	1.44	2.36	71	38	3.0	3.5	0.26	0.46	2.57	2.25	51	47	5.6	5.1	0.51	0.46		
SEM		2	3	0.10	0.20	5	3	0.2	0.3	0.02	0.04	0.37	0.26	7	6	0.5	0.4	0.15	0.07		
P		< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.02	< 0.02	< 0.01	< 0.01	> 0.1	> 0.1	> 0.5	> 0.5	> 0.1	> 0.1	> 0.15	> 0.5		

\* C = values obtained during control state before spinal and/or epidural anesthesia; A = average values obtained following anesthesia (20-30 min); MAP = mean arterial blood pressure; B.F. = blood flow; V.R. = vascular resistance; V.D. = vascular distensibility; B.F./C.O. = ratio of blood flow to cardiac output.

\*\* Two subjects studied twice on different days.

period and  $36.6 \pm 1.3$  mm Hg during spinal and epidural anesthesia.

PLASMA CATECHOLAMINE CONCENTRATION AND SKIN TEMPERATURE

Plasma catecholamine concentrations were determined before and during spinal or epidural anesthesia in eight subjects. During the control period, plasma epinephrine and norepinephrine concentrations averaged  $0.19 \pm 0.14$   $\mu\text{g/l}$  (range 0.05 to  $-0.66$   $\mu\text{g/l}$ ) and  $1.14 \pm 0.19$   $\mu\text{g/l}$  (range 0.39 to  $1.84$   $\mu\text{g/l}$ ). During spinal or epidural anesthesia, plasma epinephrine and norepinephrine concentrations averaged  $0.23 \pm 0.15$   $\mu\text{g/l}$  (range  $-0.60$  to  $0.70$   $\mu\text{g/l}$ ) and  $0.71 \pm 0.19$   $\mu\text{g/l}$  (range 0.05 to  $1.39$   $\mu\text{g/l}$ ), respectively. The changes in epinephrine and norepinephrine during spinal or epidural anesthesia were not significant ( $P > 0.5$ ).

Mean values of calf skin temperature measured by means of a Yellow Springs telethermometer (model 43-TA) with skin probes during the control period 15 minutes and 30 minutes after the onset of the spinal and epidural anesthesia were  $31.0 \pm 0.7$  C, and  $32.0 \pm 0.6$  C. Forearm skin temperature averaged  $31.7 \pm 0.6$  C during the control period,  $31.6 \pm 0.7$  C at 15 minutes, and  $31.9 \pm 0.7$  C 30 minutes after the onset of spinal or epidural anesthesia. Changes in skin temperature were not significant in either the calf or the forearm.

Discussion

Results of this study suggest that decreases in cardiac output during spinal and epidural anesthesia at a level of T4 or below are not primarily responsible for the arterial hypotension, but rather the consequences of changes in activity of the systemic venous bed. In the calf vessels, both spinal and epidural anesthesia resulted in significant increases in blood flow and vascular distensibility, accompanied by decreased vascular resistance, indicating that the anesthesia caused dilatation of both resistance and capacitance vessels in the anesthetized area.

In every instance, the ratio of calf blood flow to cardiac output increased, regardless of the direction and extent of changes in cardiac output (table 1). Changes in arterial blood

TABLE 2. Average Values of Hemodynamics in 17 Subjects before and after Spinal and Epidural Anesthesia (Level T4 or Below)

	Control	Anesthesia
Cardiac index (liter/min/M <sup>2</sup> )	$3.21 \pm 0.21$	$3.11 \pm 0.23$ ( $P > 0.5$ )
Stroke volume index (ml/beats/M <sup>2</sup> )	$47 \pm 3$	$47 \pm 3$ ( $P > 0.5$ )
Heart rate (beats/min)	$69 \pm 2$	$69 \pm 3$ ( $P > 0.5$ )
Total peripheral resistance (dynes/sec/cm <sup>5</sup> )	$1.35 \pm 0.21$	$1.19 \pm 0.09$ ( $P > 0.2$ )

pressure did not correlate with those of cardiac output (correlation coefficient,  $\gamma = +0.58$ ). We also found that mean values for cardiac index, stroke volume index, heart rate and total peripheral resistance obtained during the control state did not differ significantly from those obtained following anesthesia. It has been shown that the paralysis of the sympathetic vasoconstrictor fibers to the arterioles, capillaries, and veins results in a reduction in total peripheral resistance and an increase in the capacity of the peripheral vascular bed in the anesthetized areas.<sup>23, 24</sup> It has also been suggested that tonic constrictor impulses to the veins are blocked by spinal anesthesia, causing dilatation of veins.<sup>2</sup> In the present study spinal and epidural anesthesia caused significant decreases in vascular resistance and increases in vascular distensibility of the anesthetized calf vessels. We observed, too, that anesthesia caused a significant increase in calf blood flow ( $P < 0.01$ ) for the group as a whole, with unchanged cardiac output ( $P > 0.5$ ), resulting in a significant increase in the ratio of the calf blood flow to cardiac output ( $P < 0.01$ ). These findings may be related to a redistribution of the cardiac output following preganglionic sympathetic block, causing pooling of blood in the anesthetized areas. Thus, the decreased venous return to the heart and consequent decreases in cardiac output may be secondary to the changes in the veins following anesthesia. Since the increases in calf vascular distensibility ( $P < 0.02$ ) and decreases in calf vascular resistance ( $P < 0.01$ ) produced by spinal and epidural anes-

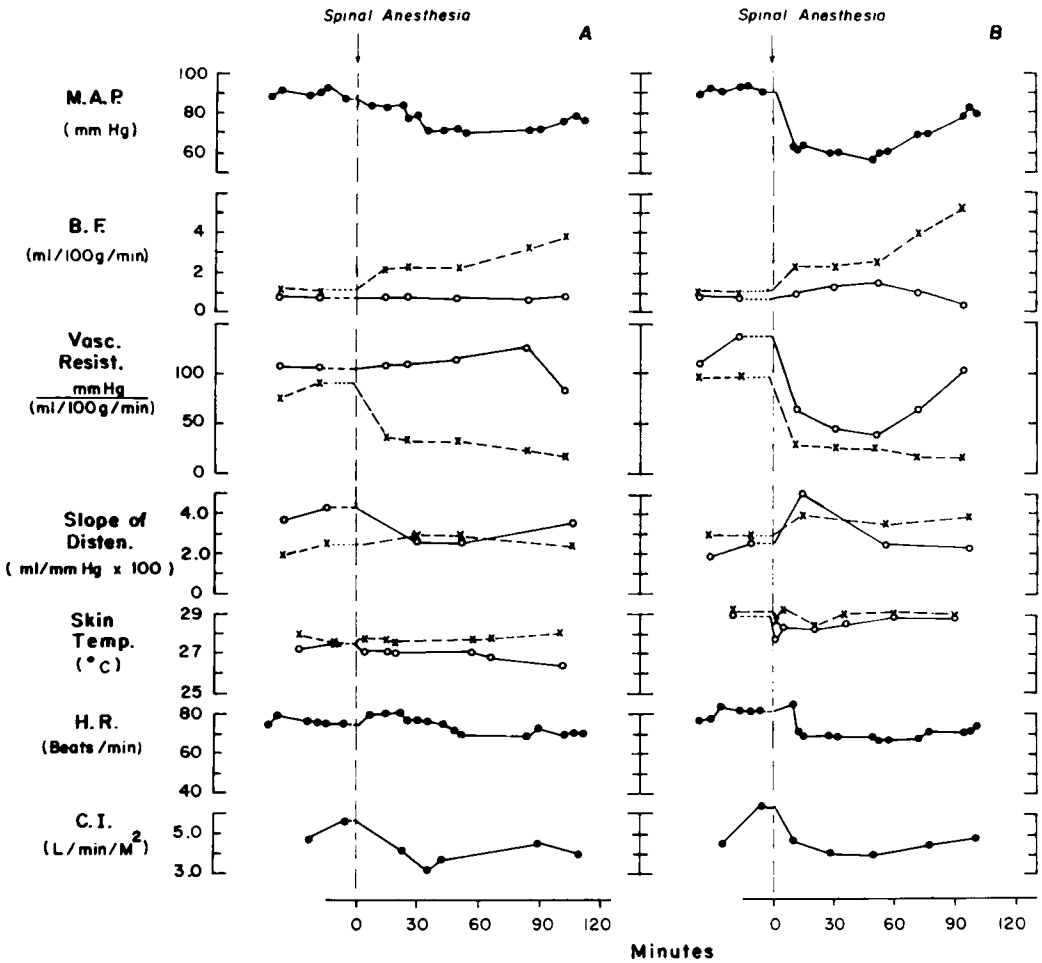


FIG. 4. Time course of changes in venous and arterial dynamics obtained in one subject before and after spinal anesthesia. A, spinal anesthesia (T4) without hypotension; B, spinal anesthesia (T4) with hypotension in the same subject.

thetia occurred in the presence of relatively unaltered cardiac output ( $P > 0.5$ ), hypotension following the anesthesia may be directly related to changes in the smooth muscle of vascular walls of the veins.

These findings are not consonant with those of previous studies in which a correlation between fall in arterial blood pressure and decrease in cardiac output was found.<sup>1, 8-11</sup> Recent studies have revealed that high subarachnoid block produces hypotension, with reductions in stroke volume, cardiac output and peripheral resistance, whereas high epidural block with epinephrine in the anesthetic solution produces the same degree of hypotension

but increases heart rate, stroke volume and cardiac output.<sup>12</sup> Furthermore, the hemodynamic effect of subarachnoid block is intensified in the presence of acute blood loss.<sup>25</sup> Under these conditions, the reduction in cardiac output may be caused by decrease in either stroke volume or heart rate. In the present studies spinal and epidural anesthesia were induced by a single injection of local anesthetic and onset of anesthesia was rapid. In the present study, the average times required to establish the desired levels of spinal and epidural anesthesia were  $12 \pm 3$  and  $25 \pm 3$  minutes ( $\pm$ SE), respectively. Therefore, the difference between hemodynamic responses to



spinal or epidural anesthesia observed in the present study and responses observed in previous studies may be related to different rates of onset of anesthesia. Changes in stroke volume and heart rate for the group as a whole following anesthesia were not significant ( $P > 0.5$ ). Therefore, it is conceivable that a more physiologic circulatory adjustment may take place when anesthesia is induced at a slower rate. Plasma catecholamine concentrations did not change significantly following spinal or epidural anesthesia. Apparently, when spinal anesthesia is induced slowly, the circulatory homeostatic reflexes may remain relatively intact.

It should be pointed out that the calculated total peripheral resistance refers to the overall resistance of the whole systemic circulation.<sup>26</sup> However, total peripheral resistance is related to the sum of the resistances in the various shunt paths offered by flows to the head, upper extremity, kidney, liver, lower extremity, etc.<sup>26</sup> It is quite possible that an increase in resistance in one shunt path may occur while in other shunt paths decreases occur. Thus, the overall peripheral resistance (*i.e.*, TPR) may be increased, decreased or unchanged, depending upon the degree and direction of alteration in each shunt-path resistance. In contrast, the calculation of vascular resistance of the limb vessels is directly related to changes in resistance in the area under consideration and may provide more direct information as to the status of the resistance vessels. Therefore, the decreased vascular resistance in the anesthetized calf vessels observed in the present study was not influenced by the changes in resistances of the other areas of body, and may indicate that hypotension caused by spinal and epidural anesthesia is related to changes in pre- and post-capillary resistance vessels.

Recent studies suggest that upper thoracic epidural analgesia depresses cardiac performance by slowing the heart rate and by reducing the myocardial responses to filling pressure.<sup>27</sup> However, it should be pointed out that cardiac output may be altered by three important mechanisms: 1) the Frank-Starling mechanism, 2) the inotropic state of the myocardium, and 3) mean aortic pressure or afterload.<sup>28</sup> The reduction of cardiac output may

occur at a given filling pressure while the inotropic state is unaltered. In contrast, increases in cardiac output may occur if the resistance to ejection (afterload) is decreased while the intrinsic contractile state of the heart (myocardial contractility) is unchanged. Recently, it has been demonstrated that left ventricular performance measured in terms of the initial ventricular impulse (time integral of applied force) is not altered by subarachnoid block in man.<sup>25</sup> It is apparent, therefore, that mere decreases in cardiac output at any given filling pressure do not necessarily indicate the change in the myocardial inotropic state.

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### Erratum

In the Abstract, "Thiobarbiturate-Succinylcholine-Oxygen for Uncomplicated Cesarean Section" (*ANESTHESIOLOGY* **30**: 344, 1969), co-author T. Takahashi's name was misspelled.