REGULATION OF BLOOD PRESSURE DURING SPINAL ANESTHESIA: OBSERVATIONS ON INTRAMUSCULAR PRESSURE AND SKIN TEMPERATURE *

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THE frequently occurring drop in arterial blood pressure during spinal anesthesia has been explained by the following theories:

1. *Paralysis of Vasomotor Centers*
   a. by diffusion of the anesthetic from the spinal fluid into the medullary centers (1),
   b. by absorption of the anesthetic into the blood stream (2, 3).
2. *Paralysis of Adrenal Nerves* with consequent oligo-adrenalinemia (4).
3. *Paralysis of Intercostal and Abdominal Muscles* leading to reduced pulmonary ventilation and decreased intrathoracic suction causing a diminished venous return (5, 6, 7).
4. *Stagnation in the Postarteriolar Bed* resulting from loss of skeletal muscle tone by paralysis of motor roots. A lowered intramuscular pressure (8) leads to diminished support of the capillary wall, venocapillary stasis and decreased venous return to the heart (8, 9, 10).
5. *Arteriolar Dilatation*
   a. in the splanchnic area owing to paralysis of splanchnic nerves (11, 12).
   b. in the entire vascular area—somatic and visceral—where anterior roots are paralyzed together with their sympathetic vasoconstrictor fibers (13, 14, 15, 16, 17, 18, 19).

The first three theories have merely historical value and can be dealth with briefly.

Paralysis of Vasomotor Centers.—It is improbable that the spinal anesthetic agent diffuses into the medullary region in sufficient concentration to affect the vasomotor centers, as the level of sensory paralysis rarely rises above the upper thoracic segments. The claim that the medullary centers are more sensitive to the anesthetic agent than are

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the spinal roots (20) cannot be maintained; on the contrary it has been proved experimentally that excessively high concentrations are required to bring about paralysis of the medullary centers (21, 22).

Absorption of the spinal anesthetic agent into the blood stream is even more unlikely to occur in effective concentrations. Intravenous injection of procaine in much larger doses than ordinarily used in spinal anesthesia does not produce hypotension (23, 24).

Paralysis of Adrenal Nerves.—An abrupt block of secretory impulses to the suprarenal medulla with resultant oligoadrenalinemia has been suggested as a possible cause of hypotension (4). Since neither ligation of the adrenal veins (25) nor bilateral adrenalectomy (26) is accompanied by a fall in blood pressure this assumption is untenable.

Paralysis of Intercostal and Abdominal Muscles.—Paralysis of these muscles not infrequently occurs in spinal anesthesia and a subsequent decrease in aspiration action of the thorax may influence blood pressure (5, 6, 7). Reduced pulmonary ventilation, however, need not follow intercostal paralysis since increased diaphragmatic action can adequately maintain oxygenation of the blood (14). Moreover it has been demonstrated that the tidal air in man and animals is not reduced under high spinal anesthesia (14). The hypotension accompanying spinal anesthesia, therefore, cannot be the result of anoxemia caused by intercostal paralysis.

The theories of postarteriolar stagnation and of arteriolar dilatation are the best established, and warrant detailed consideration. This investigation has been carried out to acquire further information on the mechanism of blood pressure regulation during spinal anesthesia in the light of these theories.

According to Henderson (8) the intramuscular pressure is a measure of skeletal muscle tone which supports the capillary wall and maintains the "venopressor mechanism" aiding the venous return to the heart. Henderson claims that a primary loss of muscle tone with subsequent stagnation in the postarteriolar bed of the lower part of the body and decreased venous return is the cause of the blood pressure drop in spinal anesthesia. If this assumption is correct, intramuscular pressure in the lower extremities should decrease in spinal anesthesia. A lowered intramuscular pressure in the legs might be secondary to hypotension, but in that case intramuscular pressure in the upper, nonparalyzed extremities ought to decrease as well and to the same degree.

In a group of 11 surgical patients, therefore, we performed simultaneous measurements of intramuscular pressure in the biceps brachii and gastrocnemius muscles together with blood pressure readings before, during and after spinal anesthesia.

In a second group of 21 cases (including several patients of the former series) skin temperatures in the lower and upper extremities were measured and blood pressure recordings made before, during

and after spinal anesthesia. This was done in order to gain additional evidence as to the role of arteriolar tone in the regulation of blood pressure during spinal anesthesia.

MATERIAL AND METHODS

The subjects studied were male and female patients aged from 19 to 78 years, in whom surgical interventions, for example hernioplasty, cystotomy, appendectomy and endoscopic urologic procedures were carried out under spinal anesthesia. In some instances test spinal anesthesia was induced without any surgical interference. Preoperative medication consisted of morhpine 0.01 Gm. and scopolamine 0.00025 Gm. subcutaneously half an hour before the anesthesia.

Spinal puncture was made in the second, third or fourth lumbar interspace in the lateral position, procaine crystals (125 to 200 mg.) dissolved in spinal liquid being injected intrathecally. The patient was placed horizontally on his back immediately after the injection.

Measurements of pulse rate, arterial blood pressure, intramuscular pressure in the biceps brachii and gastrocnemius, and skin temperature of hands and feet were performed and in a few cases venous pressure readings were taken. All the measurements were carried out in the operating theater where the patients were allowed to acclimatize at least half an hour previously with uncovered extremities. Basic values were obtained by repeated measurements before spinal puncture was done. After injection of the spinal anesthetic agent, readings were taken at frequent intervals (three to five minutes) and continued until the effect of the anesthetic disappeared. An interval of fifteen to twenty minutes was allowed to elapse between the induction of spinal anesthesia and the beginning of operation. No additional medication was given during the period of observation except for oxygen inhalation in some cases. The upper limit of skin anesthesia, as determined by pin prick, was assumed to give the highest level reached by the spinal anesthesia. The segmental distribution was recorded according to the following scheme: umbilicus, eleventh thoracic; costal arch (midclavicular line), ninth thoracic; xiphoid process, sixth thoracic; nipples, fourth thoracic, and clavicle, second thoracic.

Intramuscular pressure was measured with the apparatus of Gunther and Henstell (27). Normal values of intramuscular pressure were obtained by us in a series of healthy individuals and found to range from 60 to 100 mm. of water in the biceps brachii and 70 to 110 mm. of water in the gastrocnemius (28). The lower of two readings differing not more than 5 mm. was recorded as the intramuscular pressure (I.M.P.)

Skin temperature was measured with thermocouples at fingers, wrist, toes and ankle. The findings for fingers and toes were recorded as the mean temperatures obtained at the nailfolds and tips.

TABLE 1

No.	Pat.	Sex	Age	Surgery performed	Interspace of spinal puncture	Dosage of procaine, mg.	Highest level of anesth.	Spinal anesth.	Pulse rate	Arterial blood pressure, mm. Hg	Intramuscular pressure, mm. of water		Period of observation after spinal puncture in minutes	Remarks
											Biceps brachii	Gastrocnemius		
1.	A. B.	F.	39	Ventral herniaplasty	L II	150	Th. IV	Before During	88 90	120/70 80/45	70–80 80–90	90 85–95	75	
2.	W. M.	M.	41	Vaso-epidyd. anastomosis	L III	180	Th. IV	Before During	70 88	130/80 75/45	100–105 75–90	110 95–105	90	Vomiting, pallor, oxygen inhlalation.
3.	J. W.	M.	57	Transurethral resection of prostate	L III	130	Th. XI	Before During	86 74	140/85 120/70	65 55–70	75–80 75–80	90	Duration of operation 35 minutes, slight blood loss.
4.	M. B. R.	F.	20	Appendectomy (for chronic appendicitis)	L II	150	Th. IV	Before During	60 60	120/80 65/35	85 100–120	75–80 75–100	180	Oxygen inhalation.
5.	M. H.	M.	70	Transurethral resection of prostate	L III	120	Th. VI	Before During	100 120	140/80 85–55	60–65 60–65	75–85 75–85	60	Duration of operation 20 minutes, slight blood loss.
6.	Z. H.	M.	74	Transurethral resection of prostate	L III	120	Th. XI	Before During	88 88	150/75 125/60	50–60 40–50	70–75 70–75	70	Duration of operation 30 minutes, slight blood loss.

TABLE 1—*Continued*

No.	Pat.	Sex	Age	Surgery performed	Interspace of spinal puncture	Dosage of procaine, mg.	Highest level of anesth.	Spinal anesth.	Pulse rate	Arterial blood pressure, mm. Hg	Intramuscular pressure, mm. of water		Period of observation after spinal puncture in minutes	Remarks
											Biceps brachii	Gastrocnemius		
7.	K. M.	M.	29	Inguinal hernioplasty	L II	140	Th. IV	Before During	70 60	130/80 85/60	65–70 60–75	75–00 80–90	120	Paresthesia of upper extremities.
8.	S. C.	M.	73	Transurethral resection of prostate	L III	120	Th. XI	Before During	96 88	150/85 120/80	55–60 60–65	55–60 75–80	65	Duration of operation 20 minutes, slight blood loss.
9.	S. B.	M.	23	Inguinal hernioplasty	L III	150	Th. V	Before During	60 60	140/80 90/55	95–100 80–95	100 85–95	80	
10.	M. H.	M.	61	Cystoscopy	L IV	125	Th. VI	Before During	96 60	145/85 95/65	75–80 55–75	80–85 80–00	105	
11.	S. I.	M.	72	Transurethral resection of prostate 2nd operation	L III	125	Th. IX	Before During	86 64	170/85 135/65	65 60	85–90 100	45	Duration of operation 25 minutes

Venous pressure was recorded continuously by the direct method of Moritz and Tabora (29).

RESULTS

The results of our observations on intramuscular pressure during spinal anesthesia in 11 cases are given in table 1.

Comment on table 1.—The pulse rate and blood pressure recordings during spinal anesthesia relate to the lowest values observed. The intramuscular pressure is given as the range between lowest and highest values found.

In Cases 1, 2, 4, 7, 9 and 10, blood loss during operation was negligible. In the prostatic resections (Cases 3, 5, 6, 8 and 11) operation in no case exceeded thirty-five minutes. The blood loss was slight except in Case 11 in which after twenty-five minutes of operation a major hemorrhage occurred; the values obtained thereafter are not included in this table.

In 7 cases (1, 2, 4, 5, 7, 9 and 10) systolic as well as diastolic blood pressure levels dropped markedly during the course of the anesthesia. In these cases the anesthetic reached levels of the sixth to the fourth thoracic segments. In only two of these cases did the pulse rate drop significantly.

In four cases (3, 6, 8, 11) blood pressure fall was not prominent; the anesthesia attained levels of the eleventh to ninth thoracic segments. The pulse rate dropped only in one of these cases (11).

The intramuscular pressure in the gastrocnemius, that is in the area of motor paralysis, did not drop significantly in any case. In Cases 5 and 9 the drop recorded amounted only to 10 to 15 mm. of water and even the lowest values observed during anesthesia were well within the normal range. In Case 8 there was even an increase of 20 mm.

The intramuscular pressure in the biceps brachii showed no significant drop in 7 cases (1, 3, 5, 6, 7, 8, 11) whereas in 3 cases (2, 9, 10) a decline of 15 to 25 mm. of water was observed. In Case 4 a rise of 35 mm. of water was noted.

Interpretation.—No correlation could be demonstrated between blood pressure fall and intramuscular pressure in the gastrocnemius during spinal anesthesia.

The fact that the intramuscular pressure in the paralyzed lower extremities did not decrease may be explained in two ways. First, if according to Henderson (8) intramuscular pressure is a measure of skeletal muscle tone, then the latter seems not to be decreased in spinal anesthesia. In that case the hypotension of spinal anesthesia cannot, of course, be owing to a lowered skeletal muscle tone. Second, according to Wells et al. (30) the skeletal muscle together with its fascia has the hemodynamic properties of a closed, relatively inelastic space.

Thus, muscle tone may be actually decreased but normal values for intramuscular pressure might nevertheless be obtained owing to vasodilatation of muscular blood vessels caused by subarachnoid sympathetic block. If this is the case, a lowered muscle tone can play only a secondary role in bringing about hypotension during spinal anesthesia.

In table 2 our observations on skin temperature in the lower and upper extremities together with blood pressure readings, and so forth are recorded in 21 cases of spinal anesthesia.

Comment on table 2.—Blood pressure, pulse rate and skin temperature readings given in this table refer to the time at which the recorded level was reached by the anesthetic agent. Blood loss was absent or negligible in Cases 1, 3, 5, 6, 8, 9, 10, 11, 13, 14, 15, 16, 17, 18, 20, 21. In the prostatic resections (Cases 2, 4, 7, 19) blood loss was slight. Only in Case 12 (identical with Case 11 of table 1) did a major hemorrhage occur after twenty-five minutes of operation, but the values obtained thereafter are not included. In Cases 5, 10 and 14 test spinal anesthesia was given without further surgical intervention.

The initial blood pressure values in all cases but one were within the normal range considering the age of the patients. Only Case 12 showed a moderate systolic hypertension.

According to the behavior of their blood pressure during spinal anesthesia the cases recorded in table 2 may be subdivided into cases with *moderate* and cases with *marked* drop in blood pressure readings.

The first group comprises 11 cases (2, 4, 5, 7, 10, 12, 15, 16, 18, 19, 21). In all of them the blood pressure showed an insignificant or moderate decline, hypotensive levels not being reached. All these cases presented a rise of skin temperature in the lower extremities except Case 19 in which motor paralysis did not occur. This patient had a severe deformity of the lumbar spine and we presume that, owing to this and the low site of injection, the anterior roots were not affected by the anesthetic agent. Among the 10 cases with moderate drop in the blood pressure level and rise of skin temperature in the legs, a decline in skin temperature of the hands was observed in 8 cases (2, 4, 5, 7, 10, 12, 15, 21). The highest levels reached by the anesthesia in these cases were from the first lumbar to the ninth thoracic segments. In Case 17 the anesthesia reached the eleventh thoracic segment and the skin temperature of the fingers remained unchanged. In Case 18 the skin temperature of the hands increased, the highest level attained by the anesthesia being the fourth thoracic segment. In this case marked tachycardia occurred during spinal anesthesia in contradistinction to all other cases of this group in which the pulse rate either did not change or decreased. The tachycardia may have been compensatory and probably prevented the appearance of hypotension in this case.

TABLE 2

No.	Name	Sex	Age	Surgery performed	Interspace of spinal puncture	Dosage of procaine, mg.	Spinal anesth.	Pulse rate	Arterial blood pressure mm. Hg	Skin temperature °C.				Remarks
										Toes	Ankle	Fingers	Wrist	
1.	A. B.	F.	39	Ventral hernioplasty	L II	150	Before	88	120/70	27.5	29.5	28.5	30.5	
							Level of anesth. Th. XI	92	110/65	31.5	30.5	26	29	
							Level of anesth. Th. IV	86	80/45	32	30.5	30	31	
2.	J. W.	M.	57	Transurethral resection of prostate	L III	130	Before	88	140/85	25	27	20	31	
							Level of anesth. Th. XI	80	120/70	20.5	30	24	28	
3.	M. B. R.	F.	20	Appendectomy	L II	150	Before	60	120/80	26	20	25	30	
							Level of anesth. Th. IV	60	65/35	36	38	36	38	
4.	Z. H.	M.	74	Transurethral resection of prostate	L III	120	Before	88	150/75	23	27	24	29	
							Level of anesth. Th. XI	88	125/60	25	28	22	26	
5.	H. B.	F.	37	None (status post left thoracic (II–III) sympathectomy)	L II	150	Before	96	140/95	20	22	22	27	Finger and wrist temperature refer to the right upper extremity; see Chart 3.
							Level of anesth. Th. IX	96	110/80	36	36	18.5	24.5	
6.	K. M.	M.	29	Inguinal hernioplasty	L II	140	Before	78	130/80	26	28	32	32	
							Level of anesth. Th. IV	60	85/60	35	36	34	34	
							Th XI	60	110/75	33	34	23.5	30	
7.	S. C.	M.	73	Transurethral resection of prostate	L III	120	Before	96	150/85	28	33	30	35	
							Level of anesth. Th. XI	88	120/80	37	36	27	30	

TABLE 2—*Continued*

No.	Name	Sex	Age	Surgery performed	Interspace of spinal puncture	Dosage of procaine, mg.	Spinal anesth.	Pulse rate	Arterial blood pressure mm. Hg	Skin temperature °C.				Remarks
										Toes	Ankle	Fingers	Wrist	
8.	S. B.	M.	23	Inguinal hernioplasty	L III	150	Before Level of Th. IV anesth.	90 60	140/80 90/55	25 37	29 36	26 31	30 34	
9.	M. H.	M.	61	Cystoscopy	L IV	125	Before Level of Th. IX anesth. Th. XI	90 60 60	145/90 95/65 105/75	28 34 33.5	31 32.5 32	34 34 27	35 35 31	
10.	P. W.	M.	45	None (extradural spinal tumor complete block at L. 1)	L III	100	Before Level of L. I anesth.	84 60	130/80 120/80	30 30.5	33 30	30.5 32	35 33.5	
11.	S. F.	F.	23	Inguinal hernioplasty	L III	150	Before Level of Th. II anesth.	88 72	115/05 65/25	25 34	29 31	30 38	31 37	See Chart 1
12.	S. L.	M.	72	Transurethral resection of prostate	L III	125	Before Level of Th. IX anesth.	80 80	170/85 135/05	26 34	29 33	20.5 25	33.5 30	
13.	A. B.	M.	57	Right lumbar sympathectomy (II–IV)	L II	175	Before Level of Th. IV anesth.	90 72	130/90 75/40	31.5 34	31 35	35 37	35.5 37	Measurements performed before section of sympathetic trunk.
14.	D. F.	M.	31	None (status post left lumbar (II–IV) sympathectomy and left stellectomy)	L III	125	Before Level of Th. XI anesth. Th. II	70 68	110/05 105/65 80/45	32.5 30 30	34 35 35	34.5 29 37.5	34 30 36.5	Skin temperatures refer to the right extremities; see Chart 4.

TABLE 2—*Continued*

No.	Name	Sex	Age	Surgery performed	Interspace of spinal puncture	Dosage of procaine, mg.	Spinal anesth.	Pulse rate	Arterial blood pressure mm. Hg	Skin temperature °C.				Remarks
										Toes	Ankle	Fingers	Wrist	
15.	J. B.	M.	19	Inguinal hernioplasty	L III	150	Before	86	125/85	26	30	29	31.5	
							Level of anesth. Th. IX	72	110/60	36	33	23.5	26	
16.	A. M.	M.	60	Inguinal hernioplasty	L III	150	Before	72	150/85	26	30	30	33	See Chart 2.
							Level of anesth. Th. XI	78	170/80	35	32	26	30	
							Th. III		75/50	36	33	35.5	33	
17.	A. G.	M.	57	Lithotripsy	L IV	150	Before	80	140/85	27	31	35	37	
							Level of anesth. Th. XI	80	110/60	34.5	33	35	36	
18.	J. G.	M.	39	Biopsy of retroperitoneal sarcoma	L III	125	Before	104	140/85	26.5	30	30	34	The only case with tachycardia. In spite of the anesth. reaching Th. IV, no significant blood pressure drop occurred.
							Level of anesth. Th. IV	140	120/85	35	34	33	35	
19.	M. H. G.	M.	66	Transurethral resection of prostate	L IV	125	Before	88	135/80	26	29	35	35.5	Sensory anesthesia up to Th. XI without motor paralysis of lower extremities and abdominal muscles.
							Level of anesth. Th. XI	70	120/75	27	29.5	34	34	
20.	M. H. G.	M.	66	Inguinal hernioplasty	L II	150	Before	66	120/70	24	27	32	33	Same patient as Case 19 three weeks later. This time injection of greater dosage of procaine at higher interspace.
							Level of anesth. Th. IV	60	55/45	32	32.5	31	32	
							Th. XI	60	90/55	32	32	27	29	
21.	S. G.	M.	44	Wiring of patellar fracture	L III	120	Before	90	140/85	29	31	32	35	Skin of trunk below umbilicus warm, pink and dry; above umbilicus cool, pale and moist.
							Level of anesth. Th. IX	100	120/70	35	34	23	29	

The second group consisted of 10 cases (1, 3, 6, 8, 9, 11, 13, 14, 17, 20). In these the drop of systolic blood pressure ranged from 30 to 75 mm., and that of diastolic pressure from 20 to 50 mm. of mercury. Hypotensive levels were reached in all these cases, the lowest being 65 mm. systolic and 25 mm. diastolic. The typical rise of skin temperature in the legs was observed in all 10 cases. In 8, a rise in skin temperature of the hands occurred. Only in Cases 9 and 20 did the skin temperature of the fingers not increase. The levels of anesthesia reached in 9 cases of this group were the second to the fourth thoracic segments. Only in Case 9 was the ninth thoracic the upper limit of the anesthesia. In this case no rise in skin temperature of the hands was recorded and the lowest blood pressure observed was 95 mm. systolic and 65 mm. diastolic.

The pulse rate did not change significantly in 4 and dropped moderately in 6 cases.

Interpretation.—The above findings show a definite correlation between the degree of blood pressure drop and the upper level reached by the spinal anesthetic agent.

In none of the cases in which only a moderate decline in blood pressure occurred was the level attained by the anesthesia higher than the ninth thoracic segment.

On the other hand, in 9 of the 10 cases with marked drop in blood pressure, the levels reached by the anesthetic agent were between the second to the fourth thoracic segments.

It being understood that a rise in skin temperature indicates vasodilatation and a drop, vasoconstriction, our thermometric recordings (see table 2) reveal vasodilatation in the upper extremities in the majority of cases with marked blood pressure drop, whereas most of the cases with moderate decline in blood pressure show vasoconstriction in the upper extremities.

Vasoconstriction in the hands occurred only in those patients in whom the upper limit of anesthesia did not exceed the ninth thoracic, while vasodilatation was observed in all cases in which the anesthesia levels reached the fourth to the second thoracic segments. The vasoconstriction in the upper extremities indicates vasoconstriction in that part of the body not affected by the anesthetic. This appears to compensate for the vasodilatation in the anesthetized area.

The preganglionic sympathetic vasoconstrictor fibers for the upper extremities leave the spinal cord by way of the anterior roots of the fourth to eighth thoracic (according to Krieg (31) or by way of the third to seventh thoracic (according to Best and Taylor (32)). It is fair to assume that the highest anterior roots affected by the spinal anesthetic agent correspond to the highest posterior roots blocked as determined by the upper limit of cutaneous pin prick anesthesia.

We infer that the vasodilatation in the upper extremities observed in the hypotensive group is the result of a blocking by the spinal anes-

thetic of the sympathetic vasoconstrictor fibers in the anterior roots of the fourth to eighth thoracic. It appears, therefore, that the degree of blood pressure drop depends upon the upper level attained by the spinal anesthesia. The upper level of anesthesia divides an area of vasodilatation below from an area of vasoconstriction above. The relation between these two areas decisively influences regulation of blood pressure during spinal anesthesia. If the anesthetic reaches higher thoracic levels, the ratio is in favor of the area of vasodilatation, and hypotension ensues. If the anesthetic remains at lower levels, a sufficient number of unblocked roots capable of transmitting compensatory vasoconstrictor impulses are left to prevent a major drop in arterial blood pressure.

The following charts demonstrate this principle.

Chart 1 (representing Case 11 of table 2) is characteristic of the hypotensive group. The anesthetic reached the level of the second thoracic segment, and marked blood pressure fall coincided with vasodilatation in the lower and upper extremities.

Chart 2 (representing Case 15 of table 2) is typical of the group with moderate decline in blood pressure. The highest level of anesthesia was the ninth thoracic segment, and no major drop of blood pressure occurred since vasoconstriction in the upper extremities compensated for the vasodilatation in the lower extremities.

The sympathetic control of vasodilator and constrictor impulses in spinal anesthesia was clearly demonstrated by 2 cases in which test spinal anesthesia was induced several months after sympathetic denervation of one upper extremity.

Chart 3 (representing Case 5 of table 2) shows a low spinal anesthesia in such a case. The highest level of anesthesia being the ninth thoracic, the blood pressure fall was moderate and coincided with marked vasoconstriction in the right upper extremity. In the left (sympathectomized) upper extremity, unchanged skin temperatures were recorded during the entire period of observation.

Chart 4 (representing Case 14 of table 2) shows a high spinal anesthesia in a patient with left cervicothoracic sympathectomy. As long as the anesthesia remained at levels between the eleventh and ninth thoracic segments, a moderate decline of blood pressure occurred, together with vasoconstriction in the right upper extremity. With the anesthesia rising to the second thoracic, marked hypotension coincided with extreme vasodilatation in the right upper extremity. The left (sympathectomized) upper extremity did not show significant changes in skin temperature throughout the course of anesthesia.

DISCUSSION

Among the theories advanced to explain the hypotension of spinal anesthesia, only two are supported by present-day investigators, i.e. the

theory of stagnation in the postarteriolar bed (8, 9, 10) and the theory of arteriolar dilatation (13, 14, 15, 16, 17, 18, 19).

In favor of postarteriolar stagnation are certain hemodynamic findings obtained during spinal anesthesia. The hemodynamic features of postarteriolar stagnation are a decrease in venous return and venous pressure, a decrease in pulse pressure, an increase in arteriovenous oxygen difference and a fall in cardiac output preceding the drop in blood pressure.

A diminished cardiac output during spinal anesthesia was observed in man as well as in rabbits (14), in dogs (7, 17) and in surgical patients (33). The cardiac ouput decreased, however, only in those patients in whom the diastolic pressure dropped markedly (14). It is emphasized that if there is a decrease in cardiac output this is always preceded by a drop in arterial blood pressure (14, 17, 34, 35). To our knowledge no case of spinal anesthesia is on record in which a diminished cardiac output coincided with an unchanged diastolic blood pressure.

A considerable fall of systolic blood pressure with only insignificant changes in diastolic pressure during spinal anesthesia is reported by Neumann et al. (19). This is, however, at variance with the findings of Koster et al. (21), Goldfarb et al. (33), Seevers and Waters (7) and Schneider (14), which show a significant drop in diastolic as well as in systolic pressure. Our own observations are in accordance with those of these authors.

A decrease in venous pressure has been observed in surgical patients under spinal anesthesia (33), coinciding with arterial blood pressure drop (36). The reports on the behavior of venous pressure during spinal anesthesia, however, are by no means uniform, some authors recording inconsistent changes (14), others normal levels (37). Even an increase in venous pressure has been found to accompany the hypotension of spinal anesthesia (7). According to our observations on 4 patients on whom minor surgical procedures were performed under spinal anesthesia, only a slight decrease in venous pressure occurred. In all of them a considerable drop in arterial blood pressure preceded the venous pressure changes.

The arteriovenous oxygen difference was found by some authors (14, 38, 40) to be increased during spinal anesthesia, and by others (41) to be decreased.

On the strength of some of these data it must be admitted that postarteriolar stagnation may be present in spinal anesthesia. Two mechanisms may be imagined to bring about stasis in the postarteriolar bed during spinal anesthesia: firstly, paralysis of postarteriolar constrictor fibers in the white rami; secondly, a loss of skeletal muscle tone owing to paralysis of motor fibers in the anterior roots. It would be illogical to assume that a selective block of venocapillary constrictors would

occur as a result of paralysis of white rami without at the same time affecting the arteriolar vasomotor nerves.

A loss of skeletal muscle tone has been postulated to be the cause of venocapillary stasis (8), and the consequently decreased venous return is assumed to be the main factor in producing hypotension during spinal anesthesia (8, 9). Henderson (8) bases this claim on experimental findings of Schuberth (14) and Schneider (15), who reported diminished cardiac output and venous pressure during spinal anesthesia. It is, however, overlooked by Henderson that cardiac output decreased only in cases with marked drop of diastolic blood pressure (14) and that hypotension always occurred prior to the drop in cardiac output (14, 17, 34, 35). Thus, the latter can hardly be the cause of the former. Furthermore, postmortem studies on animals that died in shock from spinal anesthesia failed to show capillary congestion in the skeletal muscles (14). Schuberth (14) is, therefore, not of the opinion that venocapillary stasis is the principal cause of blood pressure fall in spinal anesthesia. A similar view is taken by Schneider (15) who considers reduced muscle tone only an additional factor in bringing about hypotension during spinal anesthesia.

Sarnoff and Arrowood (42) recently succeeded in producing "differential spinal block" by intrathecal injection of low concentrations of procaine, capable of blocking only autonomous nerves but leaving the motor roots unaffected. Since arterial blood pressure fell to the same extent in cases of differential spinal block and regular spinal anesthesia, the authors convincingly conclude that muscular flaccidity cannot be held responsible for the blood pressure drop during spinal anesthesia.

Our own observations on 11 patients under spinal anesthesia (see table 1), the majority of whom developed marked hypotension, failed to reveal any significant decrease of intramuscular pressure in the paralyzed lower extremities. It can thus be concluded that a decreased skeletal muscle tone cannot be the main cause of blood pressure fall in spinal anesthesia.

The majority of investigators (13, 14, 15, 16, 17, 18, 19) consider the chief cause of hypotension in spinal anesthesia to be arteriolar dialatation owing to vasoconstrictor paralysis. The principal hemodynamic feature of arteriolar dilatation is a decrease in peripheral resistance. This is characterized by a greater fall of diastolic than of systolic blood pressure, while cardiac output and venous pressure need not decline at all. Harrison (35) stresses that in primary arteriolar dilatation the blood pressure drop always precedes any decrease in cardiac output. The arteriovenous oxygen difference may remain normal.

Burch and Harrison (17), working on dogs under spinal anesthesia, found that when the blood pressure drop was slight, cardiac output remained normal. A greater decline in arterial blood pressure was usually followed by a moderate decrease in cardiac output. Only in cases with extreme drop in blood pressure was a considerable fall of

cardiac output recorded. They further investigated arteriolar tone in dogs under spinal anesthesia with the reversed perfusion method (18) and observed an increased perfusion rate of 20 to 200 per cent indicating arteriolar dilatation. They point out that in spinal anesthesia the initial change is arteriolar dilatation and that venous return and cardiac output are only secondarily affected. The important observation that, in spinal anesthesia, the blood pressure fall always precedes any decrease in cardiac output has been corroborated by several authors (14, 17, 34, 35).

Bradshaw (43) induced spinal anesthesia in normal and sympathectomized cats. In the normal cats the blood pressure level fell considerably while in the sympathectomized animals the initial level remained unchanged. The conclusion is drawn therefrom that vasomotor paralysis is responsible for the blood pressure drop in spinal anesthesia.

Neumann and co-workers (19), working with the Turner plethysmograph, reported an increased blood flow in the toes of patients under spinal anesthesia, indicating arteriolar dilatation. They hold the arteriolar dilatation observed in the toes to be representative of arteriolar dilatation in the entire anesthetized area.

A constant finding in spinal anesthesia is a definite rise of skin temperature in the lower extremities, indicating release of vasoconstrictor tone owing to sympathetic block (33, 44).

Some workers ascribed the main role in producing hypotension during spinal anesthesia to vasodilatation in the splanchnic area (11, 12). They noted that a significant drop in blood pressure occurred only when the anesthetic reached the thoracic portion of the spinal canal (11), and assumed that splanchnic paralysis was the cause. A five-fold increase in the size of the spleen was found in experimental spinal anesthesia (39).

Ferguson and North (13), however, observed that section of both splanchnic nerves in dogs resulted in only minor changes in blood pressure. Induction of spinal anesthesia into these splanchnotomized dogs produced the same profound decline in blood pressure as in healthy animals. According to these authors, the splanchnic nerves exercise only a minor influence on the maintenance of the general blood pressure level.

Sarnoff and Arrowood (42) reported that in "differential spinal block," affecting only sympathetic fibers, arterial blood pressure drops to the same extent as in regular spinal anesthesia. The degree of blood pressure fall was dependent upon the extent of the area of skin temperature rise on the body surface. They concluded from these observations that the hypotension of spinal anesthesia is the result of sympathetic paralysis and the resulting vascular dilatation.

Sufficient evidence has been assembled above to warrant the conclusion that arteriolar dilatation is the principal cause of blood pressure fall in spinal anesthesia.

Why does the blood pressure drop considerably in some cases while in others no significant change occurs? According to Ferguson and North (13) the degree of blood pressure decline is dependent upon the level attained by the anesthesia, a significant drop occurring especially if middle and higher thoracic segments are reached. They stated that the degree of blood pressure drop is in direct ratio to the number of white rami anesthetized, determining the area of lowered peripheral resistance.

In 1932 (7) and 1936 (14) it was suggested that compensatory vasoconstriction in parts of the body unaffected by the anesthetic might play a part in the maintenance of normal blood pressure levels in spinal anesthesia. Actual proof that compensatory vasoconstriction may occur in the upper part of the body was given in 1945 by Neumann et al. (19). Working with the Turner plethysmograph in men under spinal anesthesia, they observed that in those cases in which no fall in blood pressure occurred, the vasodilatation in the toes was accompanied by persistent vasoconstriction in the fingers. This they interpret as a compensatory measure in support of the circulation. When this compensatory vasoconstriction did not occur, hypotension ensued. The authors stated that no explanation can be given at present why this physiologic circulatory adjustment takes place in a certain number of cases and does not in others.

Our observations on skin temperature changes in 21 cases of spinal anesthesia (see table 2) are in accord with the findings of Neumann and co-workers (19). We could further substantiate the claim that the level attained by the anesthesia determines the degree of blood pressure drop (13).

The question why compensatory vasoconstriction occurs in some cases and not in others (19) can now be answered. Compensatory vasoconstriction in the upper extremities can take place only if their sympathetic vasoconstrictor fibers, deriving from the fourth to eighth thoracic segments, are intact, that is if the spinal anesthetic agent remains below these levels. In high spinal anesthesia affecting the upper thoracic segments the sympathetic pathways to the upper extremities are blocked. This precludes the possibility of compensatory vasoconstriction and necessarily leads to vasodilatation in the upper extremities, which was actually found to be present in all our patients in whom marked hypotension occurred.

SUMMARY AND CONCLUSIONS

Intramuscular pressure in the lower and upper extremities of 11 patients under spinal anesthesia was measured to investigate the possible correlation between skeletal muscle tone and blood pressure fall during spinal anesthesia.

Skin temperature measurements were performed in the lower and upper extremities of 21 patients under spinal anesthesia to ascertain

the role of vasodilatation in producing hypotension and of compensatory vasoconstriction in preventing it during spinal anesthesia.

The following conclusions are drawn:

Arteriolar dilatation resulting from vasoconstrictor paralysis in the anesthetized part of the body is the principal cause of blood pressure fall during spinal anesthesia.

Compensatory vasoconstriction occurs in the area not under the effect of the anesthetic agent.

The ratio between the area of vasodilatation and that of compensatory vasoconstriction determines the degree of blood pressure drop in spinal anesthesia. In high spinal anesthesia the majority of vasoconstrictor fibers, including those supplying the upper extremities (fourth to eighth thoracic), are blocked, and hypotension ensues. In low spinal anesthesia a sufficient number of vasoconstrictor fibers are left intact to prevent a major drop in blood pressure.

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