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Research Article

EFFECTS OF HIGH SPINAL ANESTHESIA ON CEREBRAL CIRCULATION AND METABOLISM IN MAN [1, 2]

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The effects of high spinal anesthesia on the general (1, 2), hepatic (3), and coronary (4) hemodynamics have been previously reported by our group. Because hypotension produced by high spinal anesthesia may be a potential hazard to the circulatory sufficiency of the brain, it was considered important to study the effects of high spinal anesthesia on the cerebral circulation and metabolism.

Studies by others have been concerned with the effects of differential spinal block on the cerebral circulation of hypertensive individuals (5) and the effect of hypotensive drugs on the cerebral circulation of aged persons (6) and hypertensives (7). The majority of patients in this study are normotensive, but the effects of high spinal anesthesia on a small group of hypertensives is included. The anesthetic level is higher and the amount of spinal anesthetic agent employed is larger than in studies reported by others, simulating the dosage used in producing surgical high spinal anesthesia, although surgical procedures were not undertaken in these subjects.

MATERIAL AND METHODS

Nineteen patients were selected from the medical wards. Of these, 15 were normotensive and free of cardiopulmonary disease, and 4 were hypertensive. Following an overnight fast and morning sedation (pentobarbital sodium, 0.1 Gm.) the patients were transported to the cardiovascular laboratory, where the ambient temperature was maintained between 23 and 24° C. In some instances a No. 6–8 F Goodale birds-eye catheter was introduced via a medial arm vein into the jugular bulb and the position of the tip checked repeatedly by fluoroscopic examination. In other instances direct puncture of the jugular bulb was employed. Blood samples were then drawn for basal determination of cerebral blood flow, arterial and jugular venous blood gases, pH, glucose, lactate, and pyruvate. Following the basal determinations, one group of 13 patients (age range, 22 to 60; average, 41.6 years) was given a spinal anesthetic of 150 to 200 mg. of procaine by barbotage. In all instances the arbitrary criteria previously described for high spinal anesthesia (1, 2) were met and confirmed by plethysmographic (8) demonstration of increased finger blood flow. Thirty minutes after the administration of the anesthetic all studies were repeated. A second group of six patients (age range, 44 to 66; average, 56 years) served as "double controls." Thirty minutes after the initial basal study a second set of determinations was performed without the administration of the anesthetic. In all instances patients were maintained in total head-down body tilt of 5 degrees. Vasopressor drugs were not given at any time.

Blood oxygen contents, carbon dioxide contents and pH determinations were performed in duplicate, and checks required to 0.2 volume per cent for oxygen and CO_2 and to 0.01 pH unit. Jugular venous and brachial arterial oxygen contents and arterial oxygen capacity were determined spectrophotometrically according to the method of Hickam and Frayser (9) and frequently spot-checked by simultaneous gasometric analysis. Carbon dioxide content of arterial blood was determined by the manometric method of Van Slyke and Neill (10) and arterial blood pH readings were obtained by use of the Cambridge glass electrode potentiometer at room temperature, and corrected to body temperature (11). Values for pCO_2 were obtained from the nomogram of Singer and Hastings (12). Glucose (13), lactate (14), and pyruvate (15) determinations were done in duplicate on arterial and venous blood.

Cerebral blood flow (CBF) was determined by the manometric technique of Kety and Schmidt (16) and expressed as ml. per 100 Gm. of brain tissue per minute. Cerebral oxygen consumption (CMR O_2) was calculated as the product of CBF and the arterial-jugular venous (A-VO_2) oxygen difference. This is expressed in terms of ml. per 100 Gm. of brain tissue per minute. Cerebral glucose consumption (CMR gl) was similarly calculated by using the arterial-jugular venous glucose (A-Vgl) difference and is expressed as mg. per 100 Gm. per minute.

The brachial arterial pressure was measured through a No. 19 indwelling arterial needle, transduced via Statham strain gauges, amplified by a Brush D.C. amplifier,

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and recorded on the Brush multi-channel oscillograph. Mean arterial blood pressures (MABP) were obtained by planimetric integration of the contours recorded during two successive respiratory cycles.

Cerebrovascular resistance (CVR) was calculated by the formula

$$CVR = \frac{\text{mean arterial blood pressure}}{\text{CBF per 100 Gm. per minute}},$$

and expressed as mm. Hg per ml. per 100 Gm. per minute. In calculating the mean arterial blood pressure, the readings taken prior to and immediately after the flows were averaged.

RESULTS

The data are presented in Tables I through III. The data have been treated statistically, although in the case of the hypertensives the small number of observations precluded such analysis.

Mean arterial blood pressure

The MABP in the normotensive group fell from 93 to 63 mm. Hg during high spinal anesthesia. The decrease in mean blood pressure for the four hypertensives was from 158 to 79 mm. Hg. In the double control group the change was from 100 to 106 mm. Hg. The marked decrease in the normotensive spinal anesthesia group is highly significant when compared to the variation in the double controls.

The cerebral blood flow

The mean CBF in the normotensive group did not change significantly during high spinal anesthesia. The prespinal value was 45 ml. per 100

TABLE I

Cerebral hemodynamics and blood gases in high spinal anesthesia *

	Normotensives													
Patient	F. D.		J. R.		B. M.		C. T.		M. L.		S. K.		M. B.	
Diagnosis	Chronic alcoholism		Pneumonia, recovered		Pneumonia, recovered		Chronic alcoholism		Pyelo-nephritis, recovered		General paresis, treated		Barbiturate intoxication, recovered	
Sex	M		M		M		M		F		M		F	
Age	22		38		26		60		26		42		51	
Spinal level	T_2–T_3		T_3		T_2(R)–T_3(L)		T_2–T_3		T_1		T_2		T_2	
	C	E	C	E	C	E	C	E	C	E	C	E	C	E
Art. O_2, vol. %	9.6	10.6	18.5	18.4	18.9	19.4	10.6	10.5	13.1	12.4	17.2	16.2	16.0	16.0
Jug. Ven. O_2, vol. %	3.1	4.0	12.7	12.6	13.7	12.5	4.9	3.8	6.8	6.9	8.4	8.3	10.2	10.3
A-V O_2, vol. %	6.5	6.6	5.8	5.8	5.2	6.9	5.7	6.7	6.3	5.5	8.8	7.9	5.8	5.7
O_2 Cap., vol. %	10.6	11.6	20.2	19.4	19.8	19.7	11.8	11.6	13.9	13.3	18.2	17.6	17.6	17.3
Art. Sat., %	91	91	92	95	96	99	90	90	94	93	95	93	91	91
Art. CO_2, vol. %	39.4	41.9	47.0	46.4	46.3	48.0	45.5	45.3	40.1	40.2	39.0	39.7	44.0	42.4
Art. pH	7.41	7.35	7.41	7.40	7.34	7.35	7.38	7.42	7.40	7.40	7.41	7.42	7.42	7.40
Art. pCO_2, mm. Hg	31	37	40	40	46	46	38	35	33	33	33	32	35	35
Pulse	75	60			83	86	91	94	81	73	70	68	79	68
MABP, mm. Hg	92	62	92	66	100	66	99	59	69	55	98	61	86	72
CBF, ml./100 Gm./min.	37	41	58	60	55	49	31	29	50	59	46	49	41	49
CMR O_2, ml./100 Gm./min.	2.4	2.7	3.2	3.3	2.9	3.4	1.8	1.9	3.1	3.2	4.1	3.9	2.4	2.8
CVR	2.4	1.7	1.7	1.0	1.8	1.3	2.9	2.0	1.4	0.9	2.1	1.4	2.1	1.5
CMR gl., mg./100 Gm./min.	6.3	4.1	3.3	3.4	1.6	2.9	2.5	2.0	3.0	5.9	4.1	2.9	5.6	0.6

* Abbreviations: MABP = mean arterial blood pressure; CBF = cerebral arterial blood pressure; CMR O_2 = cerebral oxygen consumption; CVR = cerebral vascular resistance expressed as mm. Hg/ml./100 Gm./min.; CMR gl = cerebral glucose consumption.
† Signifies a statistically significant difference when compared with the double control group ($p = <0.01$).

TABLE I—*Continued*

Cerebral hemodynamics and blood gases in high spinal anesthesia

Normotensives—*continued*						Hypertensives									
M. S.		A. G.		Mean		M. B.		E. T.		J. H.		F. E.		Mean	
Pneumonia, recovered		Chronic pancreatitis				Essential hypertension		Essential hypertension		Essential hypertension		Essential hypertension			
M		F		6 male 3 female		F		F		M		F		1 male 3 female	
35		54		39.3		39		36		52		60		46.8	
T_3		T_3				T_1		T_1		T_3		T_2-T_4			
C	E	C	E	C	E	C	E	C	E	C	E	C	E	C	E
20.1	19.2	19.2	18.4	15.9	15.7	16.4	14.6	15.4	13.6	20.2	19.0	18.1	17.1	17.5	16.1
12.5	10.0	12.3	10.3	9.4	8.7	9.1	6.0	9.5	6.8	11.0	5.1	11.7	9.8	10.3	6.9
7.6	9.2	6.9	8.1	6.5	6.9	7.4	8.6	5.9	6.8	9.2	13.9	6.4	7.3	7.2	9.2
21.2	20.4	20.5	19.7	17.1	16.8	17.6	16.0	16.4	14.6			19.2	18.5	17.7	16.4
95	94	94	93	93	93	94	91	94	93			94	93	94	92
48.4	42.7	45.2	44.1	43.9	43.4	38.3	36.3			43.0	34.9	49.4	48.0	43.6	39.7
7.41	7.43			7.40	7.40	7.40	7.39	7.43	7.41	7.41	7.54	7.42	7.41	7.41	7.44
41	35			37	37	33	31			37	23	41	40	37	31
79	79	68	62	78	74	89	79	81	58	84	68	98	102	88	77
91	69	106	61	92.5	63.4†	124	59	157	110	151	48	199	98	158	79
37	38	46	41	44.6	46.1	42	34	46	44	60	32	38	40	46.5	37.5
2.8	3.5	3.2	3.3	2.9	3.1	3.1	2.9	2.7	3.0	5.5	4.3	2.4	2.9	3.4	3.3
2.5	1.8	2.3	1.5	2.1	1.5†	3.0	1.7	3.4	2.5	2.5	1.5	5.2	2.5	3.5	2.1
5.7	5.5	6.0	3.3	4.2	3.2	2.1	3.4			2.4	4.5				

Gm. per minute as compared to 46 ml. during high spinal study. Three of the four hypertensive patients showed a decrease in CBF during spinal anesthesia. The double control studies showed a variation in mean values from 45 ml. to 43 ml. per 100 Gm. per minute. When the variation in the mean CBF in the normotensive high spinal group is compared to the double control variations, the change is not statistically significant.

The cerebral oxygen consumption

There was no significant change in the mean CMR O_2 in the normotensive patients during high spinal anesthesia as compared to the prespinal value. The small group of hypertensives similarly showed no change in mean CMR O_2 during high spinal anesthesia. The mean CMR O_2 was also unchanged during the double control studies. When the variation in mean CMR O_2 values from prespinal to high spinal was compared to those noted in the double control groups, there was no significant difference.

The cerebral vascular resistance

There was a marked and statistically significant decrease in the mean CVR in the normotensive patients during high spinal anesthesia. The hypertensive patients showed a similar change. In double control studies there was a slight increase from 2.1 to 2.4 units; this change was not significant. The marked decrease in mean CVR in the normotensive patients during high spinal anesthesia was significant when compared to the variation in the double control group.

TABLE II

Cerebral hemodynamics and blood gases in double controls

Patient	W. O.		D. M.		J. C.		A. B.		V. P.		H. R.		Mean	
Diagnosis	Chronic alcoholism		Pneumonia, recovered		Cerebral arterio-sclerosis		Essential hypertension, uncomplicated		Chronic rheumatoid arthritis		Acute tonsillitis, recovered			
Sex	Male		Male		Male		Female		Male		Male		5 male 1 female	
Age	57		55		58		44		55		66		56	
	C	E	C	E	C	E	C	E	C	E	C	E	C	E
Art. O_2, vol. %	14.5	14.4	10.9	10.9	16.7	16.8	16.6	16.8	19.0	19.1	14.0	13.9	15.3	15.3
J. V. O_2, vol. %	9.6	7.8	5.9	5.8	9.5	9.5	10.6	10.7	12.1	12.4	7.1	7.0	9.1	8.9
A-V O_2, vol. %	4.9	6.6	5.0	5.1	7.2	7.3	6.0	6.1	6.9	6.7	6.9	6.9	6.2	6.5
O_2 Cap., vol. %	15.9	15.9	12.0	12.0	18.3	18.3	17.8	17.8	20.1	20.0	14.9	14.9	16.5	16.5
Art., % sat.	91	91	91	91	91	92	93	95	91	91	94	94	92	92
Art. CO_2, vol. %	48.7	45.2	40.1	39.4	44.4	44.0	47.1	42.7	43.1	42.5	45.3	44.5	44.8	43.1
Art. pH	7.43	7.43	7.43	7.40	7.40	7.40	7.40	7.41	7.41	7.45	7.39	7.39	7.41	7.42
Art. pCO_2, mm. Hg	41	37	32	30	38	37	41	36	38	34	39	38	38.2	35.3
Pulse	76	71	94	100	79	80	68	60	75	75	82	79	79	78
MABP, mm. Hg	102	114	87	93	105	106	113	134	94	96	100	92	100	106
CBF, ml./100 Gm./min.	49	41	46	38	56	51	45	50	44	45	31	31	45	43
CMR O_2, ml./100 Gm./min.	2.4	2.7	2.3	1.9	4.0	3.7	2.7	3.1	3.0	3.0	2.1	2.2	2.8	2.8
CVR	2.1	2.8	1.9	2.45	1.9	2.1	2.5	2.7	2.1	2.1	3.1	3.0	2.1	2.4
CMR gl., mg./100 Gm./min.	3.3	1.02	3.7	2.4	6.7	8.2	5.0	3.4	5.7	5.0	1.8	3.4	4.4	3.9

TABLE III

Glucose, lactate and pyruvate values—high spinal anesthesia

Patient	Art. gluc. mg. % C	Art. gluc. mg. % E	Ven. gluc. mg. % C	Ven. gluc. mg. % E	A-V gluc. mg. % C	A-V gluc. mg. % E	Patient	Art. lact. mg. % C	Art. lact. mg. % E	A-V-lact. mg. % C	A-V-lact. mg. % E	Art. pyruv. mg. % C	Art. pyruv. mg. % E	A-V pyruv. mg. % C	A-V pyruv. mg. % E
F. D.	86	89	67	79	17	10	S. K.	3.6	4.7	−0.7	−0.4	1.03	1.25	−0.12	−0.18
J. R.	91	96	85	90	6.0	6.0	A. B.	4.6	9.4	+1.6	+1.8	1.20	1.54	−0.23	−0.03
B. M.	85	93	82	87	3.0	6.0	G. S.	4.0	8.0	−3.5	−1.6	1.55	1.80	+0.24	−0.11
C. T.	85	84	77	77	8	7	M. B.	5.2	6.4	−2.3	−0.5	1.10	1.30	−0.13	
M. L.	66	71	60	61	6	10	D. S.	4.3	4.7	−0.9	−0.4	1.86	2.78	−0.16	−0.15
S. K.	68	68	59	62	9	6	M. S.					1.08	1.30	+0.12	−0.04
M. B.	99	97	87	96	13	1.0	M. C.*	12.2	13.7			2.02	2.29		
A. G.	96	108	83	100	13	8	M. K.*	14.3	21.6						
M. S.	82	82	66	67	16	15	A. G.*	6.8	8.3			1.36	1.71		
Mean	84.2	87.8	74	79.9	10.1	7.7	Mean	6.9	9.6†	−1.8	−0.26	1.40	1.75†	−0.13	−0.05
							Double Controls								
W. O.	76	75	70	73	6	2	W. O.	5.3	5.0	−0.44	+0.30	1.00	1.10	−0.23	−0.17
D. M.	76	80	67	74	8	6	J. C.	7.3	5.7	−0.06	−0.06	1.60	1.40	−0.23	−0.06
J. C.	114	118	102	102	12	16	B. H.*	4.8	4.8			2.42	2.07		
A. B.	69	69	59	62	10	7	E. T.*	4.7	4.7			1.25	1.26		
V. P.	68	66	55	55	13	11	J. W.*	4.8	5.7			1.19	1.20		
H. R.	79	82	73	71	6	11	R. C.*	8.0	7.6			1.68	1.62		
							P. D.*	7.7	7.4			2.03	1.87		
Mean	80	82	71	73	9.3	8.9	Mean	6.1	5.8			1.60	1.50		

* Taken from studies of Hackel, Sancetta, and Kleinerman (4).

† Signifies a statistically significant difference when compared with the variation in double control series ($p = <0.01$).

The cerebral glucose consumption

The mean CMR gl showed no significant change in the normotensive high spinal group, varying from 4.2 mg. per 100 Gm. per minute in the prespinal study to 3.2 mg. per 100 Gm. per minute in the spinal period. In the double control group the mean values decreased from 4.4 mg. per 100 Gm. per minute in the first study to 3.9 mg. per 100 Gm. per minute in the second, again a change which was not significant. The values in the hypertensive patients during high spinal anesthesia showed no consistent change. The mean change in CMR gl in the normotensives during high spinal anesthesia was not significantly different from the mean change observed in double control studies.

Blood gases

In normotensive patients the mean changes in arterial oxygen content, jugular vein oxygen content, arterial-jugular venous oxygen difference, oxygen capacity and arterial oxygen saturation during high spinal anesthesia were not of statistical significance when compared to the prespinal values. This was also true for the mean changes in arterial CO_2 content, arterial pH and arterial pCO_2 in the normotensives. The small changes in oxygen and carbon dioxide concentration and in arterial pH likewise were not significant when compared to the variations observed in the double control group. In the hypertensive group there were more distinct and consistent decreases in the mean arterial oxygen content, jugular vein oxygen content, and oxygen capacity, and a notable increase in the arterial-jugular vein oxygen difference. It is of interest that the decrease in mean oxygen capacity in the hypertensives during high spinal anesthesia paralleled that of the mean arterial oxygen content so that no change in mean arterial oxygen saturation occurred. The mean arterial CO_2 content and arterial pCO_2 showed small but consistent decreases during high spinal anesthesia as compared to prespinal values. Although the small number of hypertensives studied precluded a statistical analysis, there was a suggestive difference between the normotensive and hypertensive groups in the response of oxygen values and carbon dioxide contents and pressures to spinal anesthesia.

Carbohydrate metabolites

The mean arterial glucose and venous glucose levels showed slight increases during high spinal anesthesia and similar but smaller increases during the double control studies. The mean arterial-jugular vein glucose difference showed a slight decrease in the normotensive spinal group and a similar but smaller decrease in the double control group. The hypertensives behaved similarly in this regard. These changes in the spinal group when analyzed against the double control changes were not statistically significant.

In a small series of five determinations there appeared to be a slight increase in the mean arterial lactate level during high spinal anesthesia. This was not seen in two double control studies. A slight increase was also consistently present in the mean arterial pyruvate level in six high spinal studies, but was seen in only one of two arterial pyruvate studies done in the control series. There were no consistent changes in the mean arterial-jugular vein lactate and pyruvate differences of the high spinal and the double control groups. However, the mean arterial-jugular venous lactate difference, although negative, was less negative in four of five observations during high spinal anesthesia. These results are summarized in Table III.

DISCUSSION

These studies indicate the cerebral vessels in normotensive persons can dilate sufficiently to maintain an entirely adequate cerebral circulation despite the pronounced decrease in mean arterial blood pressure caused by high spinal anesthesia. On the basis of our limited studies in hypertensives, these persons do not appear to have the same capacity for cerebral vascular compensation as do the normotensives. This is in agreement with the results obtained by Kety, King, Horvath, Jeffers, and Hafkenschiel (5) in hypertensives.

The MABP in the normotensive group fell approximately 32 per cent from the control value while the hypertensives decreased to 50 per cent of the control value during the spinal anesthetic studies. During differential spinal block the mean blood pressure drop was 26 per cent in Kety's study. This difference undoubtedly is a reflection of the higher anesthetic level and greater anesthetic

dosage in the present study (above T_4 with evidence of vascular dilatation in the upper extremities). The greatest absolute drop in MABP was obtained in the four hypertensive patients, but the absolute level of the MABP during spinal anesthesia in these hypertensives was no different than the arterial pressure level in the normotensives during spinal anesthesia. The greater decrease in arterial pressure in the four hypertensives as compared to the normotensives is consonant with the known greater reactivity of these patients to spinal anesthesia. Kety and his co-workers pointed to the correlation between the change in mean arterial blood pressure and the change in internal jugular oxygen content as evidence of inadequate cerebral circulatory homeostasis at the greater blood pressure drops. Our limited data in hypertensives corroborate this observation and suggest an inability of the hypertensive patients to obtain complete cerebral circulatory compensation during the severe hypotensive episodes. The arterial-jugular venous oxygen content in the hypertensives showed a consistent increase during the high spinal study, a finding which would be in keeping with a decreased cerebral blood flow if cerebral oxygen consumption is to be maintained.

It is of interest that the arterial lactate and arterial pyruvate levels showed a consistent increase during spinal anesthesia as compared to the prespinal values. Because our observations were numerically inadequate to evaluate these findings, we have combined the present data with those previously reported by our group in another facet of this study (4). This compilation of material is reasonable since all determinations were performed by the same laboratory, using the same technique and methods. Table III indicates that there was a constant and statistically significant increase in the arterial lactate and pyruvate levels during high spinal anesthesia, when compared to the changes observed in the double control group. The arterial glucose and cerebral arteriovenous differences of glucose, lactate, and pyruvate showed no such consistent change.

The mechanism of this increase in arterial lactate and pyruvate is not clear. It seems possible that anoxia of one or several organ systems may be responsible for the lactate and pyruvate changes. Of the systems studied, the heart (4), the splanchnic bed (3), and the kidneys (17) suffer a decreased blood flow incident to the hypotension of high spinal anesthesia. Evidence has been presented that the hepatosplanchnic oxygen consumption remains unaltered despite a decreased blood flow. The splanchnic bed appears to compensate for the decreased flow by an increased arteriovenous oxygen extraction. The heart, in spite of a decreased oxygen consumption, has a markedly decreased work load and the myocardial oxygen extraction coefficient is not increased. This does not preclude the possible development of an unrecognized oxygen debt. The effects of spinal anesthesia on the renal circulation are more difficult to assess. Recent studies (17) of the effects of high spinal anesthesia in pregnant women point to a decrease in renal plasma flow and an associated increase in renal vascular resistance. No studies of renal oxygen consumption were made. However, earlier studies of spinal anesthesia (18, 19), in which the anesthetic level was lower, showed no consistent change in renal blood flow. The critical difference between these results appear to be the effect of the spinal anesthetic on the blood pressure. In the former study, hypotension was produced, whereas the blood pressure level in the latter studies was not affected. It is reasonable to assume that when spinal anesthesia is associated with hypotension, as was the case in our studies, a decrease in renal blood flow can occur, and renal anoxemia may occur. This organ may conceivably be the source of the increased lactate and pyruvate levels during the high spinal studies. It is, of course, possible that temporary ischemia of the liver and splanchnic bed, not discernible by short term physiologic studies, may also contribute.

The mechanism of the decreased cerebrovascular resistance during high spinal anesthesia remains obscure. It is unlikely that there was a direct effect on the cerebral vessels, since the anesthetic level was never above T_1 and previous studies (20) have shown that bilateral block of the stellate ganglion does not produce a decreased cerebrovascular resistance in normotensive or hypertensive patients. Another mechanism which might decrease the cerebrovascular resistance during high spinal anesthesia is a decrease in the pCO_2 or pH of the cerebral tissues. The jugular-venous blood reflects these changes in the cerebral tissues. We have not gathered sufficient data on

jugular-venous pH and pCO_2 to implicate this mechanism, but it seems unlikely that metabolic local changes in the cerebral tissues of sufficient degree to alter pH or pCO_2 could occur in the absence of any change in cerebral oxygen uptake, cerebral blood flow, or jugular venous oxygen content. It is possible that local baroreceptors (21) in the carotid arteries and sinus may produce a reflex vasodilation of the cerebral vessels in response to hypotension.

While these results indicate that there is complete circulatory compensation in the brain during the hypotension of high spinal anesthesia in normotensive persons, it must be emphasized that this response is dependent upon local ability to vasodilate and decrease vascular resistance. Foci with vascular sclerosis or other disease may not be able to compensate completely, and may therefore be liable to ischemia. Since the technique employed gives values only for total cerebral circulation and metabolism, small foci of brain receiving a diminished blood flow may not be detected.

CONCLUSIONS

1. The effects of the hypotension induced by high spinal anesthesia on the cerebral circulation and metabolism of 13 subjects have been studied. Nine of these were normotensive and four were hypertensive. A series of six double control studies served for comparison. In the normotensive group given high spinal anesthesia the MABP fell from a prespinal level of 93 to 63 mm. Hg, but the CBF did not decrease significantly, and the CMR O_2 was unchanged. The CVR decreased significantly and was responsible for the maintenance of the cerebral blood flow in the face of the 32 per cent decrease in MABP.

In a small group of hypertensive patients, the MABP fell to 50 per cent of the prespinal value during high spinal anesthesia. Suggestive decreases were found in CBF and CVR but the CMR O_2 did not appear to change in this group.

2. There were no significant changes in the blood gases and pH in the normotensive group during high spinal anesthesia. The arterial oxygen content, jugular venous oxygen content and oxygen capacity appeared to decrease and the arterial jugular vein oxygen difference to increase in hypertensives during high spinal anesthesia. The arterial pCO_2 and arterial CO_2 contents showed consistent decreases during spinal anesthesia in the hypertensive patients.

3. The arterial blood lactate and pyruvate were significantly elevated during high spinal anesthesia associated with hypotension. It is suggested that renal or splanchnic ischemia may be responsible for these effects.

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