Circulatory Responses to Epidural Blockade of Treated and Untreated Hypertensive Patients

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The circulatory effects of epidural blockade were studied in 82 hypertensive patients. To compare the circulatory responses to epidural blockade between treated and untreated hypertensive patients, patients were divided into 60 treated and 22 untreated hypertensive patients groupes, and to confirm the magnitude of hemodynamic changes according to the segment of epidural approach, each group was then subdivided into 3 groups as cervical, thoracic and lumbar.

The blood pressure and the pulse rate were measured before and after establishment of epidural blockade. The baseline blood pressure of untreated group was significantly higher than that of treated one ($P < 0.05$), but there was no difference between two groups in the initial pulse rate. At 20 min after meperidine administration to epidural space, blood pressure drop was more pronounced in untreated thoracic subgroup than in treated one ($P < 0.05$), but there was no significant difference in blood pressure drop between treated and untreated groups of cervical and lumbar blockade. Regarding the pulse rate change, there was no significant difference between treated and untreated cases of all the subgroups. The rate of fluid load required to maintain the adequate blood pressure in the first 20 min of epidural blockade was more in treated than in untreated cases of cervical subgroup. During surgery the administration of vasopressor agents was more frequently required to maintain blood pressure in treated and untreated thoracic subgroups, while the administration of vasodilator agents was more frequent in treated and untreated cases of cervical and thoracic subgroups. The incidence of rebound hypertension was relatively high in untreated cervical and thoracic subgroups as compared to treated ones. (Key words: cervical, thoracic and lumbar epidural blockade, treated and untreated hypertension, blood pressure, pulse rate)

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Hypertension and ischemic heart disease are the most prevalent cardiovascular diseases in the adult population.

Failure of circulatory regulation, which is often seen during general or regional anesthesia is frequently encountered in these hypertensive patients. The response of patients with essential hypertension[1] and renal hypertension[2] to general anesthesia has been studied extensively. Spinal anesthesia has been shown to induce more profound and unpredictable changes in arterial blood pressure in untreated hypertensive than in normotensive patients[3].

The circulatory effects of epidural blockade are the net results of interaction of

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Table 1. Demographic data of patients

	Treated			Untreated		
	Cervical	Thoracic	Lumbar	Cervical	Thoracic	Lumbar
n (Sex)	17 (8M/9F)	11 (7M/4F)	32 (18M/14F)	7 (2M/5F)	8 (7M/1F)	7 (4M/3F)
Age (yr)	60 ± 10.8	64 ± 6.0	66 ± 10.2	63 ± 14.6	67 ± 12.3	65 ± 10.0
Height (cm)	157 ± 7.1	159 ± 8.7	157 ± 9.0	155 ± 6.4	159 ± 14.5	161 ± 8.5
Weight (kg)	57 ± 8.6	56 ± 6.7	58 ± 10.4	57 ± 6.0	63 ± 10.1	60 ± 6.6
ASA						
Class II	14	7	25	4	3	6
Class III	3	4	7	3	5	1
NYHA						
Class I	10	5	19	4	2	4
Class II	7	6	13	3	6	3
CTR (%)	52 ± 6.8	49 ± 4.3	50 ± 6.4	50 ± 7.9	49 ± 4.9	55 ± 6.4

Mean ± SD

various factors so that it is not possible to explain the observed results on the basis of any one alone[4]. Unfortunately there are few available studies about the circulatory responses of hypertensive patient to epidural blockade. Therefore we did the present study to evaluate the circulatory responses of treated and untreated hypertensive patient to epidural blockade at cervical, thoracic and lumbar segments.

Patients and Methods

Eighty-two hypertensive patients were studied before the initiation of elective surgery. Preoperatively routine hematology, serum biochemistry, chest X-ray and electrocardiogram were examined for each patient. Criteria of hypertension consisted of known history of hypertension, current antihypertensive treatment or, in the case of untreated hypertensive patients, systolic blood pressure higher than 160 mmHg or diastolic blood pressure higher than 90 mmHg. The patients were divided into two groups; 60 treated and 22 untreated hypertensive patients, and each group was then subdivided into 3 subgroups as cervical, thoracic and lumbar according to the segment of epidural approach. Patient characteristics are summarized in table 1. There was no statistically significant difference between the groups as to the demographic data of the patients.

The treated hypertensive patients received various kinds of drugs such as diuretics, reserpine, beta-adrenergic blocker, calcium channel blocker and methyldopa. All the patients were premedicated with diazepam, atropine and meperidine. At least 500 ml of lactated Ringer's solution with 5% dextrose was loaded before epidural blockade was commenced. Initial blood pressure and pulse rate were taken by noninvasive method while the patients were lying calm and quiet on the operating table before starting anesthesia. Patients were placed in the lateral position. The 16 gauge Tuohy needle was inserted into the epidural space at C7-T1 for cervical, between T6 and T10 for thoracic and at L3-L4 for lumbar blockade, and a Portex epidural catheter was introduced approximately 3 cm to the cephalad direction.

After injection of test dose of 1% mepivacaine via catheter patients were left supine. The main dose of plain mepivacaine, calculated as 1 ml for one cervical, 1.4 ml for one thoracic and 2 ml for a lumbar segment, was injected through the catheter. The concentration of mepivacaine was 1% for cervical and thoracic, and 2% for lumbar blockade. Ten minutes later the level of anesthesia was checked by pinprick. The blood pressure and pulse rate were measured 20 min after injection of main dose and circulatory responses to epidural blockade were evaluated. The crystalloid solution was loaded at the rate of 15–25 ml/kg/h. to avoid the sudden

Table 2. Main dose of local anesthetics, anesthetic level and fluid loading

	Treated			Untreated		
	Cervical	Thoracic	Lumbar	Cervical	Thoracic	Lumbar
Main dose of mepivacaine (ml)	11 ± 2.0	14 ± 1.2	13 ± 2.2	11 ± 1.1	15 ± 0.9	12 ± 2.5
Level of anesthesia	C3–T6	T3–L1	T7–S3	C3–T5	T3–L1	T9–C2
Fluid loading (ml/kg/hr)	19.5 ± 4.3	19.9 ± 3.9	17.6 ± 5.0	15.0 ± 6.6	25.3 ± 7.8	16.3 ± 2.8

Mean ± SD

Table 3. Baseline hemodynamic data

	sBP (mmHg)	dBP (mmHg)	PR (bpm)
Treated			
Total (n = 60)	149 ± 16.4	86 ± 13.5	78 ± 13.9
Cervical	149 ± 15.0	91 ± 10.8	80 ± 12.9
Thoracic	151 ± 9.1	85 ± 12.0	80 ± 14.2
Lumbar	148 ± 19.0	82 ± 15.0	76 ± 14.0
Untreated			
Total (n = 22)	168 ± 16.4*	96 ± 11.6*	84 ± 18.2
Cervical	169 ± 10.9	101 ± 10.6	82 ± 17.8
Thoracic	166 ± 23.8	96 ± 11.7	93 ± 17.0
Lumbar	168 ± 12.1	91 ± 11.8	77 ± 18.5

Mean ± SD

*$P < 0.05$ cmpared with Total Treated group

drop of blood pressure in the initial 20 min of the study. In cervical and thoracic groups light general anesthesia was supplemented and respiration was controlled manually via an endotracheal tube.

Differences between each paired data were analyzed by the Student's t-test, and $P < 0.05$ indicated a statistical significance.

Results

The doses of mepivacaine, anesthetic level and the amount of fluid given in each group are shown in table 2.

The amount of fluid loaded was less in untreated than in treated cases of cervical subgroup, on the other hand, it was more in untreated than in treated cases of thoracic subgroup, but there was no significant difference between each paired subgroups.

Baseline hemodynamic data are shown in table 3. Mean value of initial systolic pressure, diastolic pressure and pulse rate were 149 ± 16.4 mmHg, 86 ± 13.5 mmHg, 78 ± 13.9 bpm respectively in treated group, and 168 ± 16.4 mmHg, 96 ± 11.6 mmHg, 84 ± 18.2 bpm respectively in untreated group. Compared with treated group blood pressure was significantly higher in untreated group ($P < 0.05$), but no significant difference in pulse rate between two groups.

The hemodynamic changes 20 min after the administration of mepivacaine in epidural space are shown in table 4. Blood pressure and pulse rate were decreased in all the groups due to epidural blockade. The decrease in blood pressure was more profound in untreated than in treated cases of thoracic subroup ($P < 0.05$), but there was no statistically significant difference in blood pressure drop between treated and untreated cases of cervical or lumbar subgroup. Concerning the decrease in pulse rate no statistically significant difference was found between treated and untreated cases of all the subgroups.

During surgery there were some patients with fluctuation of blood pressure, to whom

Table 4. Hemodynamic changes twenty minutes after the administration of local anesthetic in epidural space

	sBP (mmHg)	dBP (mmHg)	PR (bpm)
Cervical			
Treated	−32 ± 17.5	−19 ± 17.0	− 9 ± 13.1
Untreated	−37 ± 13.5	−16 ± 7.3	−14 ± 14.1
Thoracic			
Treated	−35 ± 17.8	−15 ± 9.5	−10 ± 11.0
Untreated	−50 ± 15.4*	−29 ± 14.3*	−11 ± 16.6
Lumbar			
Treated	−26 ± 14.2	−15 ± 9.3	− 4 ± 5.3
Untreated	−30 ± 12.0	−10 ± 10.4	− 1 ± 6.9

Mean ± SD

*$P < 0.05$ cmpared with Thoracic Treated group

Table 5. The administration of vasopressor and vasodilator agents and incidence of rebound hypertension

	Vasopressor		Vasodilator		Rebound hypertension	
	Case	%	Case	%	Case	%
Treated						
Cervical	2	11.8	7	41.2	4	23.5
Thoracic	6	54.5	7	63.6	4	36.4
Lumbar	8	25.0	4	12.5	1	3.1
Untreated						
Cervical	1	14.3	4	57.1	6	85.7
Thoracic	6	75.0	4	50.0	7	87.5
Lumbar	0	0.0	1	14.3	0	0.0

vasopressor agents (e.g. dopamine, dobutamine) or vasodilator agents (e.g. nitroglycerin, phentolamine) were administered for the control of hypotension or hypertension. The administration of vasopressor agents was relatively more frequent in treated and untreated thoracic subgroups while the administration of vasodilator agents was more frequent in treated and untreated cases of cervical and thoracic subgroups. The incidence of rebound hypertension was relatively high in untreated cervical and thoracic subgroups compared with treated ones (table 5).

Discussion

Goldman et al.[5] reported that elective surgery in the absence of ideal antihypertensive control is not necessarily an added clinical risk provided a) diastolic blood pressure is stable and not higher than 110 mmHg, b) intraoperative and postoperative blood pressure values are closely monitored and controlled to prevent hypertensive or hypotensive episodes. They also suggested that effective intraoperative management might be more important than preoperative hypertensive control in terms of decreasing clinically significant blood pressure instability and cardiovascular complications in patients who had mild to moderate hypertension.

On the contrary Prys-Roberts et al.[1] demonstrated that untreated or inadequately treated hypertensive patients had larger absolute decreases in blood pressure during anesthesia and operation than did adequately treated hypertensive patients. Thus,

ideally all the untreated hypertensive patients should be identified and adequately treated prior to being subjected to anesthesia and operation, and they need more careful anesthetic management to prevent the myocardial ischemia that can result from severe intraoperative hypotension[5]. In case of hypertensive patients general anesthesia easily decreased blood pressure during induction period, abruptly increased blood pressure by tracheal intubation, and hyperdynamic circulatory state is followed by recovery period of anesthesia. For minimizing these sympathoadrenal responses epidural blockade is far more advantageous to the hypertensive patients than general anesthesia, if indicated.

The hemodynamic effects of epidural blockade mainly result from dilatation of both resistance and capacitance blood vessels by interruption of preganglionic sympathetic nerve fibers which normally convey vasoconstrictor impulses to the blood vessels[6].

Although in hypertensive patients the arteioles maintain the ability to constrict or dilate normally in response to the hemodynamic changes, the changes in resistance to blood flow due to a given change of vascular smooth muscle activity are greater in the hypertensive patients than in the normotensive patients because of the presence of medial hyperplasia and hypertrophy of blood vessels[7].

These structural changes of blood vessels and their functional consequences play a primary role in the hemodynamic responses of hypertensive patients to general or regional anesthesia, and explain why a similar degree of sympathetic block due to epidural blockade will induce greater decrease in blood pressure in the hypertensive than in the normotensive patients.

Prolonged antihypertensive treatment may induce regression of the structural changes in arterioles and, consequently, about normal functional response to vasodilatation or vasoconstriction than untreated state may attain.

According to the data of Dagnino et al.[8], in the patients with well treated hypertension there were 24% decrease of blood pressure in lumbar epidural blockade and 18% decrease in thoracic blockade. On the other hand in the patients with untreated hypertension the degree of decrease in blood pressure was much less predictable, and there was an average decrease of 44% of blood pressure due to epidural blockade. In our study baseline blood pressure of the untreated hypertensive patients was higher than that of the treated patients. Due to epidural blockade the decrease of blood pressure was more profound in untreated thoracic subgroup than in treated one. Surgical intervention was more stressful in the cervical and thoracic subgroups than in the lumbar subgroup because of the difference in the concentration of mepivacaine administered, so the administration of vasodilator agents for the control of the sudden increase in blood pressure was more frequent in these subgroups. Especially rebound hypertension after discontinuation of vasodilator was seen more frequently in untreated cervical and thoracic subgroups than in treated ones.

Due to epidural blockade the decrease of venous return to the heart may produce bradycardia by the activation of a vagal reflex designed to prevent damage caused by the forceful contraction of the empty ventricle[9]. The total blood volume in the hypertensive patients is reported to be normal or slightly decreased, but there is central redistribution of this volume, probably as a result of increased tone in capacitance so this distribution is essencial for the maintenance of cardiac output[10].

The hypertensive patients is more vulnerable to the dilatation of capacitance vessels with the concurrent decrease in venous return and, possibly, the loss of the central redistribution of the blood volume. Antihypertensive treatment can also lead to regression of left ventricular hypertrophy and this can explain partially the absence of abrupt bradycardia in the treated hypertensive patients[7,11]. In our study the slight decrease of pulse rate both in treated and untreated groups 20 min after the administration of local anesthetic in epidural space was not statistically significant.

As the decrease of venous return appears to be a major factor responsible for the hemodynamic response to epidural blockade and in the genesis of bradycardia[12], preanesthetic fluid loading can be useful in preventing the episodes of vagal overactivity in the untreated hypertensive patients if the central redistribution of blood volume can be maintained. However it should be stressed that the myocardium of the hypertensive patient with left ventricular hypertrophy is less tolerable to fluid overload. In our study the fluid loading was more in untreated thoracic subgroup than in treated one.

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