STUDIES OF ANAESTHESIA IN RELATION TO HYPERTENSION. VI: CARDIOVASCULAR RESPONSES TO EXTRADURAL BLOCKADE OF TREATED AND UNTREATED HYPERTENSIVE PATIENTS

J. Dagnino and C. Prys-Roberts

SUMMARY

The haemodynamic effects of extradural blockade were investigated in 25 hypertensive patients divided into three groups: 11 treated patients receiving lumbar extradural blockade (LT), nine treated hypertensive patients receiving thoracic extradural blockade (TT), and five untreated patients receiving lumbar extradural blockade (LU). Haemodynamic measurements were performed before and after the establishment of the extradural blockade, and repeated with the patients under light general anaesthesia. Mean upper level (T7) and range (T4-S1) of sensory blockade were similar in the two lumbar extradural groups, and mean segmental spread in the TT group was T4-L1. Changes from baseline to lowest arterial pressure showed a 22% (P<0.01) decrease in MAP in the LT group, 18% (P<0.05) in the TT group, and 42% (P<0.05) in the LU group. The decrease in arterial pressure was associated with a decrease in SVR in the LT group, and also with a decrease in cardiac output in the LU group. Three of the five untreated hypertensive patients demonstrated unacceptable decreases of arterial pressure, associated with abrupt and severe bradycardia, and required immediate treatment (head-down tilt, atropine and methoxamine). These complications were not observed in any of the treated hypertensive patients (P<0.018).

The cardiovascular effects of extradural blockade are the net result of a complex interaction of factors so that it is not possible to explain the observed results on the basis of any one alone (Stanton-Hicks, 1975; Bromage, 1978). Although the patient's pre-existing condition has been recognized as being probably the most important of these factors (Bonacci, 1978), most controlled studies on the haemodynamic response to extradural anaesthesia have been performed in volunteers, or in patients without cardiovascular disease (Kennedy et al., 1969; Bonica, Berges and Morikawa, 1970; Germain, Roberts and Prys-Roberts, 1979).

Hypertension and ischaemic heart disease are the most prevalent cardiovascular diseases in the adult population. The responses of patients with essential hypertension (Prys-Roberts, Meloche and Foëx, 1971; Prys-Roberts et al., 1971; Prys-Roberts et al., 1972; Prys-Roberts et al., 1973) and renal hypertension (Prys-Roberts, 1982) to general anaesthesia have been studied extensively, but the available information on the response of hypertensive patients to lower-body regional anaesthesia is limited. Spinal anaesthesia has been shown to induce more profound and unpredictable changes in arterial pressure in untreated hypertensive than in normotensive patients (Kety et al., 1950; Pugh and Wyndham, 1950; Kleinerman, Sancetta and Hackel, 1958). We are not aware of studies on the haemodynamic effects of extradural blockade in awake hypertensive patients. The present study was undertaken to evaluate such responses in treated and untreated hypertensive patients.

PATIENTS AND METHODS

Patients

Twenty-five hypertensive patients were studied immediately before elective surgery. Each patient was examined before operation by one of the investigators, and routine haematology, serum biochemistry, chest x-ray and an electrocardiogram obtained. Criteria for inclusion were a known history of high arterial pressure and current antihypertensive treatment or, in the case of untreated hypertensive patients, diastolic arterial pressures consistently greater than 100 mmHg on at least three resting supine measurements of arterial pressure on the day before surgery. Informed consent was obtained after careful explanation of the procedure.

According to treatment and type of surgery, patients were allocated to one of three groups: 11 treated hypertensive patients who received lumbar extradural blockade (LT), nine treated hypertensive patients receiving thoracic extradural blockade (TT) and five untreated hypertensive patients who...
TABLE I. Anthropometric and medical details of patients (see text). Age and weight are means and (SD). *P <0.05 compared with LT group. IHD = ischaemic heart disease: history of angina or previous myocardial infarction. LVH = left ventricular hypertrophy (SV1—RV5 > 35 mm) on electrocardiogram. BB = β-adrenoceptor antagonists. Others include hydralazine, methyldopa, nifedipine, reserpine and diuretics.

<table>
<thead>
<tr>
<th></th>
<th>LT</th>
<th>TT</th>
<th>LU</th>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>56 (7)</td>
<td>63 (4)</td>
<td>66 (6)*</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72 (9)</td>
<td>71 (12)</td>
<td>80 (11)</td>
</tr>
<tr>
<td>IHD</td>
<td>4</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>LVH</td>
<td>6</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BB only</td>
<td>7</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>BB + others</td>
<td>3</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>Others</td>
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<td>3</td>
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received lumbar extradural blockade (LU). Anthropometric and medical data are summarized in Table I.

None of the patients was premedicated. Treated hypertensive subjects received their antihypertensive drugs, usually the β-adrenoceptor antagonist atenolol, within 2-4 h of the start of anaesthesia. They were brought to the recovery room at least 2 h before surgery was scheduled to start. Patients were placed in a left lateral position, a 16-gauge Tuohy needle was inserted to the extradural space at L3—4 or T7—8, and a Portex catheter was introduced approximately 4—5 cm in a cephalad direction. The patients were then placed supine and an 18-gauge cannula (Abbocath) was introduced percutaneously to a brachial artery and a central venous catheter (Drum cartridge, Abbott Laboratories) was advanced from an antecubital vein until the pressure wave confirmed a central position. All these procedures were performed under local anaesthesia (1% lignocaine). Arterial and central venous catheters were connected to calibrated Gould–Statham P231D pressure transducers and to a Hewlett-Packard 78304A amplifier. ECG electrodes were positioned in a CM5 configuration. ECG, instantaneous heart rate (Devices 4522), and pressures were recorded continuously on an eight-channel recorder (Mingograph 81, Elema-Schönander). Cardiac output was measured by indicator dilution technique using 5 mg of indocyanine green and a Waters TD1 densitometer-amplifier. Arterial blood-gas tensions were measured using standard electrodes in a Radiometer ABL3 analyser. In 12 patients arterial blood samples were taken at 10-min intervals for determination of the plasma concentration of lignocaine by gas chromatography (Tucker, 1970).

Sequence
Once the extradural and intravascular catheters had been inserted the patients were left supine and undisturbed for at least 20 min. Baseline measurements of the haemodynamic indices and blood-gas tensions were obtained. Immediately afterwards 1.5% lignocaine 12—14 ml, calculated on the basis of age and height (Bromage, 1978), was injected through the extradural catheter, at a rate of 10 ml in 30 s in the LT and LU patients. The TT patients received 7 or 8 ml. Measurements of the haemodynamic variables, and blood-gas tensions, were repeated every 5 min until 30 min had elapsed. The levels of insensitivity to cold (ethyl chloride) and pinprick were assessed at 15 and 30 min.

Following the 30-min sequence of measurements, general anaesthesia was induced with fentanyl 1 ng kg⁻¹, and thiopentone 4 mg kg⁻¹. Intubation of the trachea with tracheal tubes coated with lignocaine ointment was facilitated with suxamethonium 1 mg kg⁻¹, and anaesthesia was maintained with 67% nitrous oxide in oxygen administered through a Bain system with a fresh gas flow of

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>10 min</th>
<th>20 min</th>
<th>30 min</th>
<th>During general anaesthesia*</th>
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<tbody>
<tr>
<td>LT</td>
<td>0.7</td>
<td>2.9</td>
<td>2.8</td>
<td>2.9</td>
<td>2.4</td>
</tr>
<tr>
<td>(n = 3)</td>
<td>(0.2)</td>
<td>(0.7)</td>
<td>(0.3)</td>
<td>(0.4)</td>
<td>(0.1)</td>
</tr>
<tr>
<td>TT</td>
<td>1.3</td>
<td>3.8</td>
<td>3.5</td>
<td>3.3</td>
<td>4.5</td>
</tr>
<tr>
<td>(n = 5)</td>
<td>(0.3)</td>
<td>(1.9)</td>
<td>(1.7)</td>
<td>(1.5)</td>
<td>(1.7)</td>
</tr>
<tr>
<td>LU</td>
<td>1.7</td>
<td>2.9</td>
<td>4.1</td>
<td>3.5</td>
<td>4.1</td>
</tr>
<tr>
<td>(n = 3)</td>
<td>(1.0)</td>
<td>(2.2)</td>
<td>(1.2)</td>
<td>(1.8)</td>
<td>(2.5)</td>
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150 ml kg$^{-1}$ min$^{-1}$. After 15–20 min of stable anaesthesia, and with the patients breathing spontaneously, a final set of haemodynamic measurements was obtained.

**Statistics**

Non-parametric statistical tests (Siegel, 1956) were used because of inequalities of variances. Randomization and Wilcoxon tests were used to assess changes within groups and the Mann–Whitney $U$ test to assess changes between groups. Fisher's exact probability test was used for nominal scale data. As multiple comparisons were made, the Bonferroni method was used to control the error rate (Wallenstein, Zucker and Fleiss, 1980) with $P<0.05$ taken as significant.

**RESULTS**

The mean doses of extradural lignocaine were 206 mg in the LT group, 117 mg in the TT group and 210 mg in the LU group. The mean upper level of sensory blockade (T7) and range (T4–S1) were similar in both lumbar extradural groups, while in the thoracic group mean somatic segmental spread was from T4 to L1. Plasma concentrations of lignocaine reached maximal values between 10 and 20 min in each group (table II), but no significant differences were observed between the groups despite the larger doses in the lumbar extradural groups.

Baseline haemodynamic data are summarized in table III. The systolic and diastolic arterial pressures in the untreated hypertensive patients were not significantly different from those of the treated hypertensives. The low diastolic pressures were derived from intra-arterial measurements which we have found to be consistently lower than those recorded by sphygmomanometry in the same patient. In the LU group HR and CO were higher and SVR lower than in the two groups of treated patients, although only the differences in heart rate were significant ($P<0.05$). Changes from baseline at 5-min intervals and until 30 min after the extradural injection of lignocaine, and values during steady-state general anaesthesia are depicted in figure 1. Measurements at 25 min were excluded because of missing values in both lumbar extradural groups.

The lowest arterial pressures in individual patients were observed at different times after the administration of lignocaine. Measurements at the time of these lowest pressures are shown in table IV.

**Table III. Baseline haemodynamic data expressed as means and (SD). SV and CO are standardized to 70 kg. *P < 0.05 compared with the LT group**

<table>
<thead>
<tr>
<th></th>
<th>LT</th>
<th>TT</th>
<th>LU</th>
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<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>190 (16)</td>
<td>176 (29)</td>
<td>200 (18)</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>87 (12)</td>
<td>82 (15)</td>
<td>88 (10)</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>2.8 (1.7)</td>
<td>5.0 (3.6)</td>
<td>5.4 (3.5)</td>
</tr>
<tr>
<td>Heart rate (beat min$^{-1}$)</td>
<td>57 (9)</td>
<td>64 (15)</td>
<td>80 (15)*</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>77 (19)</td>
<td>84 (20)</td>
<td>77 (21)</td>
</tr>
<tr>
<td>Cardiac output (litre min$^{-1}$)</td>
<td>4.3 (0.9)</td>
<td>5.2 (0.8)</td>
<td>6.3 (2.4)</td>
</tr>
<tr>
<td>SVR (dyn cm$^{-5}$)</td>
<td>2241 (520)</td>
<td>1714 (506)</td>
<td>1488 (474)</td>
</tr>
<tr>
<td>Arterial dP/dr (mmHg s$^{-1}$)</td>
<td>684 (130)</td>
<td>632 (165)</td>
<td>732 (323)</td>
</tr>
</tbody>
</table>

**Table IV. Haemodynamic data at time of lowest arterial pressures expressed as means and (SD). SV and CO are standardized to 70 kg. Data for LU group are based on measurements made after the treatment of hypotension in three of the five patients. *P < 0.05 compared with baseline values. †P < 0.05 compared with LT group**

<table>
<thead>
<tr>
<th></th>
<th>LT</th>
<th>TT</th>
<th>LU</th>
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<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>144 (30)*</td>
<td>142 (26)*</td>
<td>112 (39)*</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>68 (16)*</td>
<td>68 (15)*</td>
<td>54 (13)*</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>3.0 (1.8)</td>
<td>4.7 (3.8)</td>
<td>3.5 (3.2)</td>
</tr>
<tr>
<td>Heart rate (beat min$^{-1}$)</td>
<td>61 (12)</td>
<td>62 (13)</td>
<td>70 (15)</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>67 (15)*</td>
<td>77 (16)</td>
<td>74 (24)</td>
</tr>
<tr>
<td>Cardiac output (litre min$^{-1}$)</td>
<td>4.0 (1.1)</td>
<td>4.6 (0.65)*</td>
<td>5.3 (2.1)</td>
</tr>
<tr>
<td>SVR (dyn cm$^{-5}$)</td>
<td>1829 (474)*</td>
<td>1647 (386)</td>
<td>1059 (176)*</td>
</tr>
<tr>
<td>Arterial dP/dr (mm Hg s$^{-1}$)</td>
<td>562 (178)*</td>
<td>506 (117)*</td>
<td>384 (166)</td>
</tr>
</tbody>
</table>
fig. 1. Haemodynamic changes from baseline (means and SEM), after the end of the extradural injection of lignocaine, and during light general anaesthesia (GA). Significant differences within and between the hypertensive patients groups are shown for the lumbar treated group (LT) ●●, thoracic treated group (TT) ○○, and lumbar untreated group (LU) △△.
EXTRADURAL BLOCKADE IN HYPERTENSIVE PATIENTS

**Lumbar extradural: treated**

In the LT group haemodynamic changes were apparent after 10 min of extradural blockade. SAP decreased by 46 mm Hg (24%) from baseline to lowest \( (P < 0.01) \), with a 19 mm Hg (22%) decrease in DAP \( (P < 0.01) \). This was associated with an 18% decrease in SVR \( (P < 0.05) \), a 13% decrease in SV \( (P < 0.05) \) and an 18% decrease in arterial \( dP/dt \) \( (P < 0.05) \). Changes in HR (7%) and CO (−6%) were not significant. General anaesthesia produced a further but not significant decrease in arterial pressure associated with a 21% decrease from baseline of SV.

**Thoracic extradural: treated**

In the TT group the decrease in arterial pressure up to 15 min of blockade was associated with a decrease in SVR but no change in CO. From 15 to 30 min arterial pressure and SVR increased slightly with only minor decreases of HR, SV and CO. Only the changes in arterial pressure were significant \( (P < 0.05) \). The SAP and DAP decreases from baseline to lowest were 34 mm Hg (19%) and 14 mm Hg (17%), respectively, associated with a 12% decrease in CO \( (P < 0.05) \), a 20% decrease in arterial \( dP/dt \) \( (P < 0.05) \), and decreases in SV (8%) and SVR (4%) (n.s.). General anaesthesia was associated with further decreases in arterial pressure to values similar to those in the LT group but associated with significant decreases in SV (21%) and CO (23%).

**Lumbar extradural: untreated**

In three of the five untreated hypertensive patients the experimental design had to be altered when unacceptable arterial hypotension (<50% of baseline values) and abrupt bradycardia presented, in association with symptoms of confusion, blurred vision and agitation. Two of the patients were treated initially with atropine i.v. and 15° head-down tilt between 10 and 15 min after the extradural injection of lignocaine. All three received methoxamine (Vasoxine) i.v. in 2-mg increments between 20 and 30 min. Further doses of methoxamine were administered to one of the patients after general anaesthesia had been induced. At 15 min, despite atropine and head-down tilt in two patients, arterial pressures had decreased by 40%, associated with decreases in SVR (21%) and CO (23%). Increases in arterial pressure, SV, CO, arterial \( dP/dt \) and SVR at 30 min reflect the effect of treatment given to the three patients. The decreases in SAP and DAP, from baseline to lowest, including values of the three patients after initial therapy, averaged 88 and 34 mm Hg (44 and 39%) respectively \( (P < 0.05) \) and were linked with decreases in arterial \( dP/dt \) (45%), SVR (27%) and CO (16%), although these were not statistically significant. No i.v. fluids were given at this time as, in previous studies (Prys-Roberts and Meloche, 1980), the rapid administration of fluid (1 litre) i.v. increased cardiac output but failed to increase arterial pressure. General anaesthesia induced a further decrease in arterial pressure associated mainly with a decrease of SVR.

Changes in central venous pressure were not consistent, except for the modest increase in all three groups with the induction of general anaesthesia.

There were insignificant changes in blood-gas variables during the extradural blockade but, after general anaesthesia had been induced, \( P_{aCO_2} \) increased significantly \( (P < 0.05) \) in all groups: from 4.9 kPa (0.53 SD) at 30 min to 6.0 kPa (0.8 SD) in the LT group, from 4.9 kPa (0.67 SD) to 6.1 kPa (1.1 SD) in the TT group, and from 5.3 kPa (0.8 SD) to 6.4 kPa (1.1 SD) in the LU group.

**DISCUSSION**

The baseline cardiovascular status was similar in the two treated hypertensive groups (LT, TT) showing a high arterial pressure, a high systemic vascular resistance and a normal cardiac output, with values comparable to those found by Frohlich, Tarazi and Dustan (1969). The higher SVR observed in the LT group, although not significant, was probably the result of the higher proportion of patients being treated with \( \beta \)-adrenoceptor antagonists as opposed to vasodilator drugs such as hydralazine and methyldopa.

Baseline values in the LU group showed quite a different pattern, characteristic of labile hypertension, in that the high arterial pressure was associated with a high cardiac output but a SVR within normal limits (Frohlich, Tarazi and Dustan, 1969). The higher SVR observed in the LT group, although not significant, was probably the result of the higher proportion of patients being treated with \( \beta \)-adrenoceptor antagonists as opposed to vasodilator drugs such as hydralazine and methyldopa.

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ma concentrations of lignocaine, secondary to local infiltration, could be playing a role through its inotropic and chronotropic actions (Foldes et al., 1960; Harrison, Sprouse and Morrow, 1963; Covino, 1980). As this effect of lignocaine is the result of sympathetic activity secondary to a central nervous system action (Kao and Jalar, 1959; Blair, 1975) it could, presumably, be blocked by the β-adrenoceptor antagonist therapy in the two treated groups, but not in the untreated patients.

The haemodynamic values in the LU group were influenced by the treatment administered to three of the five patients after the onset of blockade. Because of the high incidence of severe haemodynamic disturbances we decided not to pursue the study in the untreated hypertensive patients despite the fact that the small numbers made statistical evaluation within and between groups more difficult.

The haemodynamic changes in the LT group were very different in magnitude from the minimal changes observed in young volunteers (Bonica, Berges and Morikawa, 1970) and slightly greater than those observed in normotensive premedicated surgical patients using a similar experimental design (Germann, Roberts and Prys-Roberts, 1979). As premedication can induce significant depressant effects on the cardiovascular response to extradural blockade (Bonica, 1978), it is reasonable to suggest that the changes in arterial pressure in our unpremedicated treated hypertensive patients could have been even more significant had they been premedicated.

The haemodynamic effects of extradural anaesthesia with a plain solution of local anaesthetic result mainly from vasodilatation of resistance and capacitance vessels as a result of blockade of sympathetic nerve fibres (Bromage, 1978). Although the arterioles of hypertensive patients are capable of a normal range of vasoconstriction—vasodilatation, the changes in resistance to flow in response to a given change in vascular smooth muscle activity are greater than in the normotensive patient because of the presence of medial hyperplasia and hypertrophy (Folkow, 1978). These structural changes and their functional consequences play a primary role in the haemodynamic response of hypertensive patients to general anaesthesia and surgery (Prys-Roberts, 1980), and explain why a similar degree of sympathetic blockade after extradural anaesthesia will induce greater decreases in SVR and arterial pressure in the hypertensive than in the normotensive patient. Prolonged antihypertensive treatment may induce regression of the structural changes in arterioles (Folkow, 1978) and, consequently, a more normal functional response to vasodilatation or constriction. Thus, the observations of the present study, and those made after the induction of spinal anaesthesia in untreated hypertensive subjects (Kety et al., 1950; Kleinerman, Sancetta and Hackel, 1958), are compatible with these pathophysiological concepts.

Lumbar extradural anaesthesia produces little or no change in cardiac output in volunteers (Bonica, Berges and Morikawa, 1970) or in patients without cardiovascular disease (Germann, Roberts and Prys-Roberts, 1979), unless the upper thoracic cord segments are involved (Otton and Wilson, 1966; Stanton-Hicks, 1975), or if blockade is induced in hypovolaemic subjects (Bonica et al., 1972). The response of our untreated hypertensive patients was similar to that observed in hypovolaemic volunteers in whom the extradural blockade induced episodes of severe hypotension and abrupt bradycardia. The decrease in venous return to the heart may produce bradycardia and syncope by the activation of a vagal reflex designed to prevent damage caused by the forceful contraction of the empty ventricle (Öberg and Thorén, 1972). Total blood volume in hypertensive patients is normal or decreased only slightly, but there is a central redistribution of this volume, probably as a result of an increased tone in capacitance vessels (Lund-Johansen, 1980). This redistribution is essential for the maintenance of cardiac output as the hypertrophied left ventricle of the hypertensive patient is more dependent on the Frank—Starling mechanism to maintain its performance (Folkow, 1978; Tarazi, 1982; Tarazi and Levy, 1982). Thus, the hypertensive patient is more vulnerable to the dilatation of capacitance vessels with a concurrent decrease in venous return and, possibly, the loss of the central redistribution of blood volume. Antihypertensive treatment can also lead to regression of left ventricular hypertrophy (Folkow, 1978; Tarazi, 1982) and this could explain partially the absence of abrupt bradycardia in the treated hypertensive patients. Alternatively, it could result from a protective effect of β-adrenoceptor antagonism as pronounced bradycardia was, as a rule, seen only when there was evidence of strong sympathetic stimulation on the heart (Öberg and Thorén, 1972). This also agrees with the high incidence of bradycardia seen in hypovolaemic volunteers (Bonica et al., 1972), and with the fact that it is not commonly found in subjects with
EXTRADURAL BLOCKADE IN HYPERTENSIVE PATIENTS

1071

extradural or spinal anaesthesia combined with general anaesthesia (Scott, 1982).

As the decrease in venous return appears to be a major factor responsible for the haemodynamic consequences of extradural blockade (Shimosato and Etsten, 1964) and in the genesis of bradycardia and syncope, prior fluid loading could be useful in preventing the episodes of vagal overactivity in the untreated hypertensive patients if the central redistribution of blood volume can be maintained. It should be stressed, however, that the heart of the hypertensive patient with left ventricular hypertrophy is less tolerant to fluid overload. Clearly this aspect needs further study.

The other major factor involved in the haemodynamic response to extradural anaesthesia is the decrease in SVR. Methoxamine appears to be a sensible choice for the treatment of hypotension after extradural blockade in the treated hypertensive patient as it is important to avoid tachycardia and the consequent increase in myocardial oxygen consumption. However, when the hypotension is associated with bradycardia and a decrease in cardiac output, as in the untreated hypertensive patients, the use of a mixed-action drug such as ephedrine would be preferable (Ward et al., 1966).

Thoracic segmental blockade from T4 to L1 induced only moderate changes in arterial pressure in our treated hypertensive patients. The changes were mainly the result of a decrease in SVR, there being little or no change in cardiac output—effects probably attributable to the fact that the upper thoracic sympathetic fibres were spared. The absence of untoward effects agrees with observations made in a mixed group of patients, treated with β-adrenoceptor antagonists for hypertension or ischaemic heart disease, in whom thoracic extradural blockade was induced while the patients were under general anaesthesia (Reiz et al., 1982).

General anaesthesia induced a further decrease in arterial pressure in all three groups, but especially in the untreated hypertensive patients, although data for this group included those of one patient who received 4 mg of methoxamine after the induction of general anaesthesia. Decreases in arterial pressure in the two treated groups were less, and those in the untreated group greater, than those described for treated and untreated hypertensive patients without an extradural blockade but with 1% halothane (Prys-Roberts, Meloche and Foëx, 1971).

Anatomical differences between the thoracic and lumbar extradural space (Bromage, 1978) could explain why the plasma concentrations of lignocaine were similar in the three groups despite the much larger doses in the lumbar groups. In a younger group of patients Mayumi, Dohi and Takahashi (1983) found no differences in plasma concentrations after the injection of equal doses of lignocaine to the thoracic or lumbar extradural spaces. It must be emphasized that local infiltration for the cannulation procedures produced significant plasma concentrations of lignocaine.

Treated hypertensive patients respond to lumbar or thoracic extradural anaesthesia with a moderate but significant decrease in arterial pressure associated mainly with a decrease in systemic vascular resistance and with no untoward effects. In contrast, a similar segmental spread of lumbar extradural blockade in the untreated hypertensive patients caused a much greater decrease in arterial pressure and the frequent occurrence of sudden and life-threatening bradycardia. Atropine and head-down tilt only partially restored arterial pressure, although the heart rate increased. We recommend that hypertensive patients be monitored closely before and after the induction of an extradural blockade, including an electrocardiogram and direct measurement of arterial pressure. Atropine and a vasopressor should be prepared before beginning the procedure. Until the effectiveness of measures to prevent or treat the severe cardiovascular depression seen in the untreated hypertensive patients is investigated, the use of regional anaesthesia in these patients should be approached with caution.

REFERENCES


ANESTHESIE CHEZ L'HYPERTENDU. VI: REPONSES CARDIOVASCULAIRES AU BLOC PERIDURAL CHEZ LES HYPERTENDUS TRAITES OU NON TRAITES

RESUME

Nous avons étudié les effets hémodynamiques d’un bloc péridural chez 25 sujets hypertendus divisés en trois groupes: 11 patients traités recevant une péridurale lombaire (LT), neuf patients non traités recevant une péridurale thoracique (TT) et cinq patients traités recevant une péridurale lombaire et thoracique. Les mesures hémodynamiques étaient faites avant et après l’installation du bloc péridural et répétées une fois les patients sous anesthésie générale. Le niveau supérieur moyen (D4 - S1) du bloc sensitif était le même dans les deux groupes (D4 – S1) du bloc sensitif étaient les mêmes dans les deux groupes. Les mesures hémodynamiques étaient faites avant et après l’installation du bloc péridural et répétées une fois les patients sous anesthésie générale.

Le niveau supérieur moyen (D7) et l’étendue segmentaire moyenne du bloc peridural et répétées une fois les patients sous anesthésie générale. Le niveau supérieur moyen (D4 – S1) du bloc sensitif étaient les mêmes dans les deux groupes.
EXTRADURAL BLOCKADE IN HYPERTENSIVE PATIENTS

brutale et sévère nécessitant un traitement immédiat (mise en décline, atropine et methoxamine). Aucun patient hypertendu traité n'a présenté de telles complications ($P=0.018$).

ESTUDIOS SOBRE ANESTESIA CON RELACIÓN A LA HIPERTENSION. VI: RESPUESTAS CARDIOVASCULARES AL BLOQUEO EXTRADURAL DE PACIENTES HIPERTENSIVOS TRATADOS Y NO-TRATADOS

SUMARIO

Se investigaron los efectos hemodinámicos del bloqueo extradural en 25 pacientes hipertensivos divididos en tres grupos: 11 pacientes tratados recibieron un bloqueo extradural lumbar (LT), nueve pacientes hipertensivos tratados recibieron un bloqueo extradural torácico (TT) y cinco pacientes no-tratados recibieron un bloqueo extradural lumbar (LU). Se llevaron a cabo las mediciones hemodinámicas antes y después del establecimiento del bloqueo extradural y se repitieron en los pacientes bajo anestesia general ligera. El nivel superior medio (TT) y la gama (T4-S1) del bloqueo sensorial eran similares en los dos grupos extradurales lumbar y en el grupo TT, la disperación segmental media era de T4-L1. Los cambios de línea de base hasta la presión arterial más baja demostraron una disminución del 22% ($P<0.01$) del MAP en el grupo LT, del 18% ($P<0.05$) en el grupo TT y del 42% ($P<0.05$) en el grupo LU. El descenso de presión arterial se encontró asociado con una disminución del SVR en el grupo LT así como de un descenso del volumen minuto en el grupo LU. Tres de los cinco pacientes no-tratados demostraron disminuciones inaceptables de la presión arterial, asociadas con una bradicardia abrupta y severa y exigieron un tratamiento inmediato (inclinación de cabeza por abajo, atropina y methoxamina). Dicha complicaciones no ocurrieron en ninguno de los pacientes hipertensivos tratados ($P=0.018$).