CARDIOVASCULAR SUPPORT DURING COMBINED EXTRADURAL AND GENERAL ANAESTHESIA

P. M. C. WRIGHT AND J. P. H. FEE

SUMMARY

We have examined the effect of prophylactic treatment with i.v. fluid 1000 ml, ephedrine 24 mg or methoxamine 4 mg on cardiovascular responses to both extradural and combined extradural and general isoflurane anaesthesia in 45 adult patients undergoing knee arthroplasty. Heart rate (HR) and systemic arterial pressure (AP) were measured using automated oscillotonometry and cardiac output was measured using continuous wave suprasternal Doppler ultrasonography. After lumbar extradural anaesthesia (LEA) there were no significant differences in arterial pressure between treatments, although cardiac index was significantly greater after fluid preloading (mean 4.3 (95% confidence interval 3.7-4.9) litre min$^{-1}$ m$^{-2}$) than after ephedrine (3.1 (2.6-3.6) litre min$^{-1}$ m$^{-2}$) or methoxamine (2.6 (2.0-3.2) litre min$^{-1}$ m$^{-2}$). During combined LEA and general anaesthesia, systolic AP was significantly greater after ephedrine (114 (103-125) mm Hg) than after either preloading (98 (88-107) mm Hg) or methoxamine (97 (89-105) mm Hg). The reduction in AP after induction of general anaesthesia was associated with a decrease in cardiac index after fluid preloading and a decrease in vascular resistance after methoxamine.

KEY WORDS


Suggested advantages of lumbar extradural anaesthesia (LEA) in the perioperative period include a reduced incidence of postoperative thromboembolism, reduced blood loss and improved postoperative analgesia [1]. In addition, recent work has suggested that surgical outcome may be improved after extradural anaesthesia and analgesia [2].

Extradural anaesthesia has been shown to reduce thromboembolism after knee arthroplasty [3,4]. However, the presence of a thigh tourniquet obviates the other potential benefits of extradural anaesthesia: blood loss is already minimal and cannot be attenuated further, but hypertension associated with tourniquet inflation may be prevented [5]. In addition, adequate sensory block of the knee requires an extensive block, as the knee has a nerve supply from both lumbar and sacral nerve roots and good sacral block is difficult to achieve without large volumes of local anaesthetic. Combined LEA and general anaesthesia may be a potentially suitable technique for knee arthroplasty, but the combination results in a greater degree of hypotension than with each technique alone [6].

The purpose of this study was to compare the relative merits of i.v. fluid preloading, ephedrine and methoxamine as cardiovascular support during combined extradural and general anaesthesia in patients undergoing knee arthroplasty.

PATIENTS AND METHODS

This study was carried out with the approval of the University Medical Ethics Research Committee and informed written consent was given by each patient. We studied adult patients (age range 23-76 yr) in ASA categories I and II, undergoing knee arthroplasty. Patients were excluded if they had a haemoglobin concentration less than 10 g dl$^{-1}$, were receiving cardioactive medications, had a heart rhythm other than sinus or had evidence of aortic dilatation or unfolding on chest x-ray.

Patients were allocated randomly to one of three regimens intended to support the cardiovascular system during LEA and later combined LEA and general anaesthesia. One group received an i.v. fluid preload consisting of Dextran 70 in 0.9% sodium chloride 500 ml over 10 min preceding the main extradural injection and then 500 ml of Hartmann's solution during the 20 min after completion of the dose (group P). A second group received a bolus i.v. injection of ephedrine 12 mg 5 min after completion of the extradural injection and another 12 mg infused in 50 ml of 0.9% sodium chloride 500 ml over 10 min preceding the main extradural injection and then 500 ml of Hartmann's solution during the 20 min after completion of the dose (group E). A third group received a bolus i.v. injection of methoxamine 2 mg 5 min after completion of the extradural injection and another 2 mg infused in 50 ml of 0.9% sodium chloride over the subsequent 15 min (group M). The remaining patients received a bolus i.v. injection of methoxamine 2 mg 5 min after completion of the extradural injection and another 2 mg infused in 50 ml of 0.9% sodium chloride over the subsequent 15 min (group M). These doses of the vasopressors are known to be approximately equipotent at increasing the arterial pressure during regional anaesthesia [7].

Peter M. C. Wright*, M.B., F.F.A.R.C.S.I.; J. P. H. Fee, M.D., Ph.D., F.F.A.R.C.S.I., Department of Anaesthetics, The Queen's University of Belfast, Whitsa Medical Building, Belfast BT9 7BL, and Musgrave Park Hospital, Belfast. Accepted for Publication: January 3, 1992.

*Present address: Department of Clinical Anaesthesia, Royal Victoria Hospital, Grosvenor Road, Belfast. Correspondence to J. P. H. F.
Anaesthetic technique

Premedication comprised temazepam 20 mg by mouth. On arrival of the patient in the anaesthetic room, a 14-gauge cannula was inserted in a forearm vein. An 18-gauge extradural cannula was placed through a 16-gauge Tuohy needle so that its tip was at the body of the third lumbar vertebra. The patient rested for 10 min and, after baseline variables were recorded, a test dose consisting of 2% lignocaine 3 ml was given through the extradural cannula. After 5 min, during which time the patients in group P received the i.v. fluid preload, a further 2% plain lignocaine 17 ml was injected over 2 min. Twenty minutes after completion of this injection, the extent of anaesthesia to pinprick was determined. General anaesthesia was induced with thiopentone sufficient to obtund the eyelash reflex and maintained subsequently with 66% nitrous oxide and isoflurane (controlled to an end-tidal concentration of 0.5% (Ohmeda 5330 agent monitor)) in oxygen, with the patient breathing spontaneously via a face mask and circle system. End-tidal carbon dioxide pressure was monitored and controlled within 10% of the initial value, although controlled ventilation was never required during this study.

Measurements

Heart rate (HR) and arterial pressure (AP) were measured using an automated oscillogonometer (Datascope Accutorr). Measurements were made immediately before the extradural injection, after the fluid preload and subsequently at 1-min intervals during extradural anaesthesia and at 2.5-min intervals after the induction of general anaesthesia. Cardiac output was measured non-invasively using continuous wave suprasternal Doppler ultrasonography with a dedicated commercially available signal analyser (Datascope Accucom); the physical characteristics required for the numerical representation of cardiac index were determined empirically. Measurements were made immediately before the extradural injection, after the i.v. fluid preload and at 2-min intervals during extradural anaesthesia and 5-min intervals during general anaesthesia and surgery. The patients were blinded to the treatment administered, but it was not possible to double-blind the treatment allocation as the i.v. fluid preload could not be concealed and the acute slowing in heart rate which follows a bolus of methoxamine is obvious to the investigator using the suprasternal Doppler probe. However, data were logged by computer unseen by the investigator, the computer program demanding two estimations of cardiac output at each time point, and not allowing further measurements.

Intraoperative ECG, AP, \( P_{\text{CO}_2} \) and \( S_{\text{PO}_2} \) were monitored continuously, as was airway isoflurane concentration. Subjective symptoms after vasconstrictor administration were recorded as they were volunteered. Adverse cardiovascular events (hypotension (systolic AP (SAP) < 100 mm Hg) or hypertension (SAP > 180 mm Hg), arrhythmias) were recorded during and for 4 h after the operation. During the subsequent hospital stay, clinically apparent surgical complications (wound infection, thromboembolic events and adverse cardiovascular events) were recorded.

Analysis of data

Parametric data are reported as mean (95% confidence intervals) and frequency data are represented as frequencies and percentage. The 95% confidence interval of the difference between the means is given where this is appropriate. Within-group changes were analysed using repeated measures analysis of variance and between-group comparisons made using one-way analysis of variance followed by Student-Newman-Keuls multiple range test. Statistical significance was assumed when \( P < 0.05 \).

RESULTS

We studied 45 patients—15 in each of the three groups. The groups were similar in age, weight, height and gender distribution (table I) and cardiovascular variables on admission (table II). The combination of the extradural block and general anaesthesia was adequate for surgery in all the patients.

Acute bolus drug effects

The administration of dextran 70 in saline 500 ml before extradural anaesthesia resulted in little change in HR or AP, but there was a significant increase in cardiac index (CI), from mean 3.03 (95% confidence interval 2.53–3.52) litre min\(^{-1}\) m\(^{-2}\) to 3.54 (2.96–3.57) litre min\(^{-1}\) m\(^{-2}\). The mean maximum changes in measured variables after a bolus of either eph-

| Table II. Cardiovascular characteristics on admission to hospital and induction dose of sodium thiopentone (mean (SD or range)) |
|-----------------|-----------------|-----------------|
|                 | Group P         | Group E         | Group M         |
| HR (beats min\(^{-1}\)) | 74 (7)          | 71 (13)         | 69 (8)          |
| Systolic AP (mm Hg) | 139 (24)        | 132 (18)        | 135 (17)        |
| Diastolic AP (mm Hg) | 78 (9)          | 75 (7)          | 76 (10)         |
| Thiopentone (mg kg\(^{-1}\)) | 4.3 (1.1) | 4.2 (1.0)       | 4.2 (0.8)       |
| Upper block height | T5 (T3–T7)     | T5 (T2–T9)      | T6 (T3–T9)      |
| Lower block height  | S3 (S2–S4)     | S3 (S1–S4)      | S3 (S1–S5)      |
Table III. Mean (95% confidence intervals) maximum changes in cardiovascular variables in the 5 min after a bolus dose of vasopressor.

<table>
<thead>
<tr>
<th></th>
<th>Ephedrine</th>
<th>Methoxamine</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beat min⁻¹)</td>
<td>9.5 (3.3-15.6)</td>
<td>-19 (-15 to -23)</td>
</tr>
<tr>
<td>Systolic AP (mm Hg)</td>
<td>21 (12-31)</td>
<td>23 (19-28)</td>
</tr>
<tr>
<td>Diastolic AP (mm Hg)</td>
<td>11.3 (6.8-15.8)</td>
<td>11.9 (7.8-16.1)</td>
</tr>
<tr>
<td>CI (litre min⁻¹ m⁻²)</td>
<td>-0.15 (-0.42 to 0.12)</td>
<td>-0.83 (-0.44 to -1.20)</td>
</tr>
</tbody>
</table>

Changes during extradural anaesthesia

Values for HR, AP and CI were determined for each patient after the cardiovascular changes had reached a steady state at least 12 min after completion of the extradural injection. Systemic vascular resistance index (SVRI) and left ventricular minute work index (LVMWI) were determined using standard equations (central venous pressure was assumed to be constant). The changes in measured and derived variables associated with extradural anaesthesia in each of the three groups are shown in figures 1 and 2. HR decreased after methoxamine and was significantly reduced compared with either group P (95% confidence intervals for difference between means 5.1-20.2 beat min⁻¹) or group E (4.7-21.3 beat min⁻¹). Mean SAP in group P was significantly reduced compared with baseline and was markedly reduced compared with either group E (-5.6 to 26.6 mm Hg) or group M (-4.0 to 33.8 mm Hg). CI increased significantly in group P and was significantly greater than either group E (0.94-3.66 litre min⁻¹ m⁻²) or group M (1.59-4.55 litre min⁻¹ m⁻²). Mean SVRI remained unchanged in group M, but decreased significantly in group E and significantly more so in group P. LVMWI was significantly increased from baseline in group P and was significantly greater than the value in both vasoconstrictor groups.

Changes during combined extradural and general anaesthesia

Values of HR, AP and CI were determined and SVRI and LVMWI calculated for each patient at
least 10 min after induction of general anaesthesia, but before tourniquet application or surgical stimulation; these values are also shown in figures 1 and 2. Heart rate reduced significantly in all groups after induction of general anaesthesia and that in group M continued to be reduced significantly compared with either group P (95% confidence interval for difference between means 6.7-20.3 beat min\(^{-1}\)) or group E (5.0-19.8 beat min\(^{-1}\)). SAP reduced significantly in all groups, but more so in groups P and M, and was significantly less in these two groups than in group E (2.0-29.0 mm Hg and 4.3-29.5 mm Hg compared with groups P and M, respectively). The induction of general anaesthesia resulted in a significant reduction in CI in group P, with relatively little alteration in the vasoconstrictor groups, although CI remained significantly greater in group P compared with group M (0.55-1.87 litre min\(^{-1}\) m\(^{-2}\)). Systemic vascular resistance reduced in groups M and E and after induction of general anaesthesia there were no significant differences between the groups.

Side effects

There were no subjective symptoms reported by the patients during extradural anaesthesia alone. Postoperative hypotension occurred in all groups and there was a single case of postoperative hypotension in each of the vasoconstrictor groups (no significant differences between groups) (table IV).

**DISCUSSION**

We have found that, during extradural anaesthesia, both ephedrine and methoxamine, in appropriate dosage, were more effective at maintaining AP during extradural anaesthesia than fluid preloading; both achieved this by modulating decreases in SVRI that would normally occur as a result of extradural block. In addition, the combination of extradural anaesthesia and general anaesthesia, as expected, produced large reductions in AP [6, 8-10]. The reductions were greatest in the fluid preloading and methoxamine groups after large decreases in cardiac output and systemic vascular resistance, respectively.

The method used here to measure cardiac output (suprasternal continuous wave Doppler) is subject to errors and uncertainties; this subject has been discussed extensively elsewhere [11, 12]. Probably the major source of error with this technique is the determination of aortic cross-sectional area. In an investigation such as this, which is concerned with relative changes with time, such error as does arise from this source should remain the same in each patient and should be unimportant, especially as suprasternal Doppler actually measures the aortic minute distance which is proportional to CI [13] (hence the presentation of cardiac output as indexed to body surface area throughout this study). The accuracy of the device used in this study (Datascope Accucom) in tracking changes in a particular patient has been reported previously [14].

The circulatory effects of extradural anaesthesia have been investigated extensively. If the upper level of block is maintained below T2 in normal healthy supine volunteers, little decrease in AP is to be expected [15], although any degree of hypovolaemia may result in severe circulatory depression [16]. When hypotension does develop, it is a result of failure of venous return [17], as cardiac output must be maintained at much greater values than normal to compensate for reductions in systemic vascular resistance. The patients in this study who received the fluid preload did experience a reduction in AP, the most likely explanation for which is their greater age compared with subjects in previous studies [18].

The efficacy of i.v. fluid preloading to support AP during regional anaesthesia in an ageing population has been questioned [19] and this study was designed to evaluate vasoconstrictors as an alternative. Both ephedrine and methoxamine were effective in maintaining AP during LEA alone. Previous work carried out during treatment, rather than prophylaxis of hypotension, has indicated that bolus doses of methoxamine briefly decrease cardiac output [7] and bolus doses of ephedrine increase it in both obstetric [20] and non-obstetric [7] patients. During this study, the vasoconstrictors were given prophylactically and, although the brief reduction in cardiac output after methoxamine was confirmed, ephedrine had little effect on cardiac output.

The cardiovascular effects of combined extradural and general anaesthesia have received relatively little attention in the literature to date. A high incidence of hypotension was recorded during earlier studies [8, 10] and further reductions in AP when adrenaline was used with the extradural solution also have been documented [9]. The failure to sustain cardiac output after induction of isoflurane anaesthesia observed in the fluid preload group was similar to that observed with halothane anaesthesia [6]. The possibility that this resulted from interaction with circulating lignocaine has been discounted elsewhere [21]. Isoflurane has cardiac effects which differ from those of halothane and the reason why cardiac output should decrease with isoflurane during extradural anaesthesia remains obscure. Cardiac output is normally maintained during isoflurane administration as a result of sympathetic stimulation, and we can only speculate that, in the presence of extradural anaesthesia, increases in sympathetic outflow are unavailable to compensate for its direct cardiac effects [22].
The different responses in the two vasoconstrictor groups after the induction of general anaesthesia represent some of the most interesting findings of this study. After ephedrine, both cardiac output and systemic vascular resistance altered little after introduction of isoflurane, so that AP altered little. In contrast, after methoxamine, systemic vascular resistance decreased immediately after introduction of isoflurane. This failure of methoxamine to maintain SVR during general anaesthesia was unexpected, as the action of another alpha, adrenergic agonist drug, phenylephrine, is unaltered in the presence of halothane [23].

It has been argued that the use of vasoconstrictor drugs as prophylaxis against hypotension during regional block is illogical, as hypotension is a result of a failure of venous return [17]. However, by maintaining SVR at pre-block values they reduce the need for an increase in cardiac output and make the failure of venous return less likely in patients under extradural block. Methoxamine failed to maintain SVR after nitrous oxide-isoflurane, while preloading failed to maintain cardiac output after nitrous oxide-isoflurane; with both regimens there was a large reduction in AP with a combined anaesthetic. It has been argued that the use of vasoconstrictor drugs as prophylaxis against hypotension during regional block is illogical, as hypotension is a result of a failure of venous return [17]. However, by maintaining SVR at pre-block values they reduce the need for an increase in cardiac output and make the failure of venous return less likely in patients under extradural block. Methoxamine failed to maintain SVR after nitrous oxide-isoflurane, while preloading failed to maintain cardiac output after nitrous oxide-isoflurane; with both regimens there was a large reduction in AP with a combined anaesthetic.

REFERENCES


