

A comparison of the haemodynamic effects of intrathecal meperidine, meperidine-bupivacaine mixture and hyperbaric bupivacaine

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Purpose: To study the haemodynamic effects of intrathecal meperidine, administered either alone or mixed with bupivacaine.

Methods: We studied 42 Chinese patients, aged 59–87 yr, scheduled for transurethral bladder or prostate surgery, randomized into three equal groups, that received either meperidine 0.8 mg·kg⁻¹, meperidine 0.4 mg·kg⁻¹ plus 1.5 ml of 0.5% heavy bupivacaine or 3 ml of heavy bupivacaine 0.5%. Non-invasive systolic (SAP) and mean (MAP) arterial pressures, central venous pressure and cardiac index, stroke index and heart rate (HR) measured by the BoMed NCCOM3-R7S bioimpedance device, were recorded over the first 25 min. Systemic vascular resistance index (SVRI) was derived. Onset of sensory and motor block was also measured. Decreases in MAP of 25% were treated with colloid and metaraminol.

Results: The onset of block was slower in the meperidine group ($P < 0.05$). Decreases in SAP, MAP and SVRI (all; $P < 0.001$) occurred within five minutes in all three groups. The HR was increased in the bupivacaine group ($P = 0.03$), but bradycardias treated with atropine occurred in six patients receiving meperidine and four patients receiving the mixture. Six patients receiving meperidine and two patients receiving the mixture required general anaesthesia for inadequate

block. The incidence of nausea and vomiting was higher in the patients receiving meperidine ($P < 0.05$). No other complications were encountered.

Conclusions: Intrathecal meperidine used alone or mixed with bupivacaine has no intra-operative advantage over heavy bupivacaine 0.5%.

Objectif: Etudier les effets de la mépéridine sous-arachnoïdienne administrée seule ou associée à la bupivacaine.

Méthode: Étaient inclus dans l'étude 42 patients de race chinoise, âgés de 59–87 ans; programmés pour une chirurgie prostatique ou vésicale, répartis aléatoirement en trois groupes égaux: mépéridine 0,8 mg·kg⁻¹, mépéridine 0,4 mg·kg⁻¹ avec bupivacaine hyperbare 0,5% 1,5 ml ou bupivacaine hyperbare 3 ml. Les pressions artérielle systolique (PAS) et moyenne (PAM) non effractives, la pression veineuse centrale et l'index cardiaque, l'index systolique et la fréquence cardiaque (Fc) ont été mesurés à l'aide de l'appareil à impédance BoMed NCCOM3-R7S et enregistrés pendant les 25 premières minutes. L'index de résistance vasculaire systémique (IRVS) a été déduit. Le début du bloc sensitif et moteur a aussi été mesuré. Les baisses de PAM de 25% ont été traitées avec un colloïde et du métaraminol.

Résultats: Le bloc a débuté plus lentement dans le groupe mépéridine ($P < 0,05$). Des baisses de PAS, PAM et de IRVS ($P < 0,001$ pour les trois) sont survenues en moins de cinq minutes dans les trois groupes. La Fc a augmenté dans le groupe bupivacaine ($P = 0,03$), mais des bradycardies traitées avec de l'atropine sont survenues chez six patients qui recevaient de la mépéridine et quatre qui recevaient le mélange. Six patients recevant la mépéridine et deux patients le mélange ont eu besoin d'une anesthésie générale parce que le bloc était insuffisant. L'incidence des nausées et des vomissements a été plus élevée chez les patients qui recevaient de la mépéridine ($P < 0,05$). Il n'y a pas eu d'autres complications.

Conclusion: La mépéridine sous-arachnoïdienne seule ou mélangée à la bupivacaine n'offre pas d'avantages sur la bupivacaine 0,5 hyperbare.

Key words

ANAESTHETIC TECHNIQUE: spinal;
ANAESTHETICS, LOCAL: bupivacaine;
ANALGESICS: meperidine;
MEASUREMENT TECHNIQUES: blood pressure, cardiac output.

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Intrathecal opioids are well established in the management of postoperative pain.¹ However, in recent years, interest has been directed towards using the opioid meperidine as an intrathecal anaesthetic agent. Meperidine differs from the other opioids in that it also possesses considerable local anaesthetic properties.^{2,3} In several recent studies meperidine compared favourably with both bupivacaine and lidocaine as the sole anaesthetic agent,⁴⁻⁶ providing excellent conditions for lower abdominal and pelvic surgery for over one hour.⁴⁻¹¹ Intrathecal meperidine has been shown to have fewer side effects and prolonged postoperative analgesia.^{7,12,13} However, there are relatively few studies examining the haemodynamic effects of intrathecal meperidine and their results are limited to a few intermittent readings from eight patients.⁸ By mixing smaller doses of intrathecal meperidine with bupivacaine, Nguyen Thi *et al.*¹⁴ showed that better post operative analgesia with fewer side effects could be achieved. The objectives of the present study were to determine the haemodynamic effects of intrathecal meperidine, used alone and mixed in lower doses with bupivacaine. Comparisons were made with a standard dose of intrathecal bupivacaine.

Methods

We studied 42 ASA II or III elderly Chinese patients, who required spinal anaesthesia (SA) for elective transurethral prostate or bladder tumour surgery. Local Ethical Committee approval was obtained before commencing the study and informed written consent was obtained from each patient. Patients were excluded from the study if their New York Heart Association dyspnoea class was III or IV or if their heart rate (HR) was irregular, because in patients with irregular heart rates stroke volume varies between heart beats. Patients with a baseline central venous pressure (CVP) of less than 0–2 cm H₂O were considered to be dehydrated and excluded from the study.

Patients were studied following an overnight fast, as in the routine in our hospital. Oral diazepam, 5–10 mg, was given one hour before surgery.

Patient monitoring were attached and baseline measurements made after a ten minute stabilization period. Systolic (SAP) and mean (MAP) arterial pressures were measured every minute using an automated oscillotonometer (Dinamap 1846SX, Critikon, Florida, USA) and data were recorded on the attached printer. The CVP measurements were made using a manometer attached to a 16 gauge cannula inserted into the right internal jugular vein with the zero taken at the mid axillary line and 4–6th intercostal space. Cardiac output was monitored non-invasively by transthoracic electrical bioimpedance (TEB) using the BoMed NCCOM3-R7S

(BoMed Medical Manufacturing Ltd., Irvine, CA, U.S.A.). It was connected to the patient according to the manufacturer's instructions, using four neck and four lower thoracic electrodes with two chest electrodes for detecting the ECG.¹⁵ The method used to derive cardiac output has been presented elsewhere and is discussed later.^{16,17} Data were indexed to the patient's body surface area and the average of 16 beats recorded. The variables recorded were cardiac index (CI), stroke index (SI) and HR. Using custom written software, these data were recorded continuously by an IBM-compatible lap-top computer onto a spreadsheet file (Microsoft Excel, Version 4.0, Microsoft Corporation, USA) for analysis at a later date.

Data from the Dinamap and BoMed were collected for three to five minutes before the patient was turned into the lateral position for subarachnoid block and continued for at least 25 min afterwards. The CVP measurements were made before turning the patient laterally and at five-minute intervals after starting the subarachnoid block.

Using sealed numbered envelopes, containing previously randomized instructions, patients were elected to receive one of the three spinal anaesthetics solutions: 0.8 mg·kg⁻¹ of meperidine 5% (Pethidine Injection B.P., Antigen Pharmaceuticals Ltd., Roscrea, Ireland), a mixture of 0.4 mg·kg⁻¹ of meperidine plus 1.5 ml of bupivacaine (Marcain Spinal 0.5% Heavy, Astra, North Ryde, NSW, Australia) or 3 ml bupivacaine. Dural puncture was performed with a 22 gauge spinal needle at the L₃₋₄ or L₄₋₅ interspace with the patient in the lateral position. The study drug was drawn up and injected over ten seconds. The patient was then returned to the supine position and remained horizontal for the duration of the study. Data were collected over 25 min by an investigator who was blinded to the study drug. The level of sensory block to pin prick and the degree of motor block, using a modified Bromage score (0 = no motor block; 1 = just able to flex knees; 2 = unable to flex knees; and 3 = unable to dorsiflex feet)¹⁸ were assessed at five minute intervals during the study.

During the study we aimed to maintain MAP > 75% of the pre-block value. If MAP decreased below this limit on two consecutive readings, a rapid infusion of Haemaccel 8 ml·kg⁻¹ was given and if this failed to restore MAP a further 4 ml·kg⁻¹ was given. In cases where Haemaccel failed to restore MAP metaraminol (0.5 mg·ml⁻¹) was given as a bolus of 0.25 mg followed by an infusion of 5 mg·h⁻¹. Bradycardias (<50 beat·min⁻¹) were treated with atropine 0.6 mg *iv*.

Once the data collection was completed, surgery was allowed to start. Inadequate block was treated with analgesics or general anaesthesia as appropriate.

Patients were also assessed during the surgery and until discharge from recovery by verbal questioning, unless stated otherwise, at 20 min intervals for: (i) nausea and vomiting, (ii) sedation; defined by a Ramsay score of greater than four (patient asleep but responds to a light glabellar tap or a loud auditory stimulus,¹⁹) (iii) respiratory depression; defined as a respiratory rate of less than eight breaths per minute or a pulse oximetry reading of less than 90% when giving supplementary oxygen, 4 L · min⁻¹, via a simple face mask and (iv) pruritus.

Data analysis and statistics

The haemodynamic data collected before SA was averaged to give the baseline values. Subsequent TEB data were divided into minute intervals and data within each interval averaged to give mean values for each minute during SA. The systemic vascular resistance index (SVRI) was calculated from the formula:

$$\text{SVRI} = (\text{MAP} - \text{right atrial pressure}) \times 80 / \text{CI}$$

CVP values were substituted for right atrial pressure.

The magnitudes of the haemodynamic changes that followed intrathecal injection in each group but not including the effects of rescue treatment were calculated. First the mean percentage changes compared with baseline for SAP, MAP, SVRI, CI, SI and HR or the numerical difference from baseline for CVP were calculated for each minute interval. Then means for each variable for all the calculated percentages between 10–25 min, but excluding data collected after giving rescue treatment, were calculated.

The maximum height of sensory block, the time for the sensory block to reach the tenth thoracic dermatome level and the time for the motor block to reach a Bromage score of 3 were found in each patient. In those patients where the block was of insufficient height and failed to reach these levels a time of 30 min was used for the purpose of statistical analysis.

Statistical analysis was performed using the programme Statview 4.01 (Abacus Concepts, Inc., USA). Patient data, baseline haemodynamic data, height and onset times of block and incidence of complications were compared using analysis of variance (ANOVA), Kruskal Wallis or Chi square tests as appropriate. Haemodynamic data collected during subarachnoid block were compared within each group and between groups using analysis of variance for repeated measures (ANOVA-RM). Multiple paired Student *t* tests were used to make comparisons with baseline and identify when within group changes became significant. *P* < 0.05 was regarded as significant. Results are presented as mean (SD).

The power of the study was assessed using the nomo-

TABLE I Patient characteristics (mean (range or SD)) in the three patient groups

	<i>Bupivacaine</i> (<i>n</i> = 14)	<i>Mixed</i> (<i>n</i> = 14)	<i>Meperidine</i> (<i>n</i> = 14)
Age (yr)	72 (61–79)	73 (62–87)	72 (59–82)
Weight (kg)	57 (9)	55 (8)	60 (9)
Height (cm)	164 (9)	165 (8)	166 (5)
Sex (M/F)	13/1	13/1	14/0
ASA status (II/III)	12/2	10/4	13/1

No significant difference between the groups.

TABLE II Baseline haemodynamic variables (mean (SD)) in the three patient groups

	<i>Bupivacaine</i> (<i>n</i> = 14)	<i>Mixed</i> (<i>n</i> = 14)	<i>Meperidine</i> (<i>n</i> = 14)
SAP (mmHg)	144 (22)	153 (23)	154 (20)
MAP (mmHg)	102 (9)	107 (12)	109 (16)
SVRI (dyn · sec · cm ⁻⁵ · m ⁻²)	3035 (729)	3121 (771)	3145 (837)
CVP (cm H ₂ O)	5.9 (2.8)	7.4 (2.1)	6.6 (2.5)
CI (L · min ⁻¹ · m ⁻²)	2.7 (0.8)	2.7 (0.8)	2.7 (0.6)
SI (ml · m ⁻²)	34 (14)	36 (14)	38 (11)
HR (beat · min ⁻¹)	82 (12)	78 (14)	74 (13)

SAP = systolic arterial pressure, MAP = mean arterial pressure, SVRI = systemic vascular resistance index, CVP = central venous pressure, CI = cardiac index, SI = stroke index and HR = heart rate. No significant difference between the groups.

gram method described by Altman.²⁰ We wished to detect changes of 5% between haemodynamic data. This value was used for the standardized difference in the power analysis. As ANOVA-RM examines the changes within each set of data, rather than differences between actual values, we used the coefficient of variation of the measured variable, which is a measure of repeatability, for our estimate of standard deviation in the power analysis. For Dinamap measurements, arterial pressure is measured with an accuracy of ±10% (confidence limits)²¹ or coefficient of variation of 5%. For BoMed measurements, the coefficient of variation for stroke volume and cardiac output is 4.7%.²² Based on these figures the required study size was 14 to 16 patients per group.

Results

Fourteen patients were studied in each group. The three groups were similar with respect to age, weight, height, sex, ASA status, maximum level of sensory block (Table I) and baseline haemodynamic data (Table II). No patient was found to be dehydrated. The speeds of onset of sensory and motor blocks were slower in the meperidine group than in both the bupivacaine and the mixture groups (*P* < 0.05) (Table III).

The haemodynamic effects of spinal block, in all

TABLE III Maximum sensory level (thoracic (T) or lumbar (L) dermatome) of block to pin prick (median (range)) and time for sensory block to reach T₁₀ and motor block to reach a Bromage score of three (mean (SD)) in the three patient groups

	Bupivacaine (n = 14)	Mixed (n = 14)	Meperidine (n = 14)
Max. sensory level	T _{5.5} (T ₂ -T ₁₁)	T ₇ (T ₃ -L ₄)	T ₇ (T ₃ -L ₂)
Onset times (min)			
- Sensory block	10 (8)	10 (9)	17 (11)*
- Motor block	12 (10)	14 (7)	21 (6)*

Significant difference compared with other groups: **P* < 0.05.

patients, and including the effects of rescue treatment for hypotension, are shown in Figure 1. The magnitudes of the haemodynamic effects of block, before rescue treatment for hypotension or bradycardia, are shown in Table IV.

In the bupivacaine group decreases compared with baseline occurred in SAP after two minutes, in MAP after one minute and in SVRI after two minutes (all ANOVA-RM; *P* < 0.001) and there was an increase in HR between 3–12 min (ANOVA-RM; *P* = 0.0001). In the mixture group decreases, compared with baseline, occurred in SAP after three minutes, in MAP after three minutes, in SVRI after three minutes (all ANOVA-RM; *P* < 0.001) and in CVP between 5–10 min (ANOVA-RM; *P* = 0.01). In the meperidine group decreases, compared with baseline, occurred in SAP after three minutes, in MAP after five minutes, in SVRI after five minutes (all ANOVA-RM; *P* < 0.001) and in CVP at five minutes (ANOVA-RM; *P* = 0.03). The decreases in SAP, MAP and SVRI at five minutes were similar in each group. No within or between group differences were found in CI or SI. There was an increase in HR in the bupivacaine group compared with the other groups (ANOVA-RM; *P* = 0.03). All patients were included in the ANOVA-RM analysis (Figure 1).

Nine patients in the meperidine group, seven patients in the mixed group and six patients in the bupivacaine group required treatment for hypotension, after 14 (7), 13 (6) and 7 (2) min respectively and included in these patients were six meperidine group and four mixed group patients who also developed bradycardias requiring treatment with intravenous atropine (Figure 2). The maximum height of sensory block in all patients receiving treatment was above the seventh thoracic dermatome (range T₂-T₇).

More patients in the meperidine group, 6/14, required general anaesthesia for inadequate block than in the bupivacaine group, 0/14, (*P* < 0.01). The reasons for general anaesthesia in the meperidine group included two low blocks (T₁₂ and L₂), one unexpected delay of

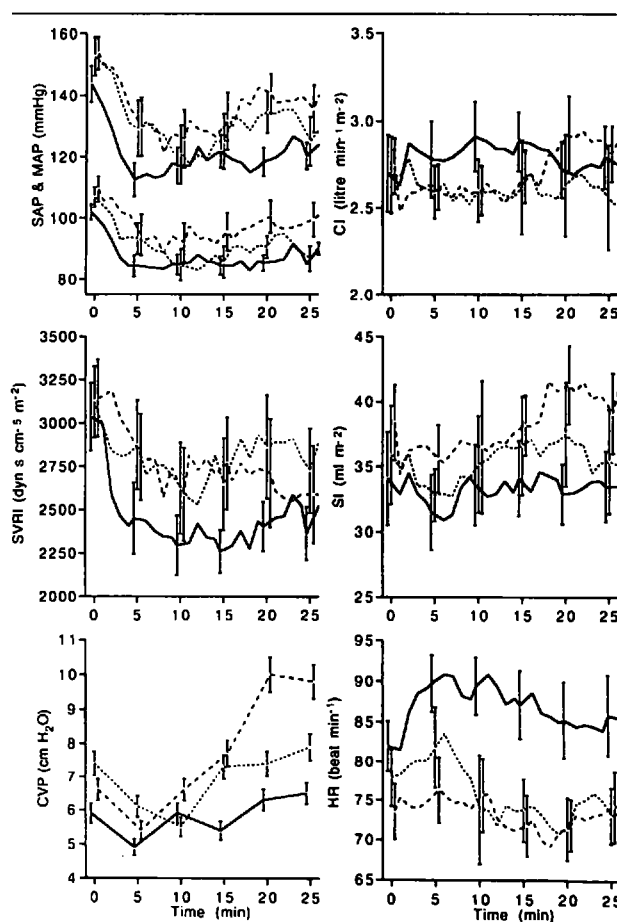


FIGURE 1 Mean (SEM) values for haemodynamic variables at baseline (0 min) and during the first 25 min of subarachnoid block for the three patient groups. Data from all patients included. — bupivacaine, mixture and --- meperidine.

TABLE IV Percentage change (Δ) and numerical difference (diff.) compared with baseline values for haemodynamic variables during subarachnoid block, measured between 10–25 min after intrathecal injection, in the three patient groups (mean (SD)). Data collected after treatment for hypotension or bradycardia has been excluded.

	Bupivacaine (n = 14)	Mixture (n = 14)	Meperidine (n = 14)
Δ SAP (%)	-23 (13)†	-25 (19)†	-24 (23)†
Δ MAP (%)	-21 (12)†	-23 (16)†	-24 (20)†
Δ SVRI (%)	-22 (16)†	-18 (17)†	-17 (22)*
CVP diff. (cm H ₂ O)	-1.5 (1.7)†	-1.5 (2.6)	-0.8 (1.6)
Δ CI (%)	+5 (18)	-7 (14)	-8 (14)
Δ SI (%)	-6 (16)	-5 (14)	-11 (16)*
Δ HR (%)	+12 (15)†	-1 (15)	+5 (14)

SAP = systolic arterial pressure, MAP = mean arterial pressure, SVRI = systemic vascular resistance index, CVP = central venous pressure, CI = cardiac index, SI = stroke index and HR = heart rate. Significant changes compared with baseline: **P* < 0.05; †*P* < 0.01.

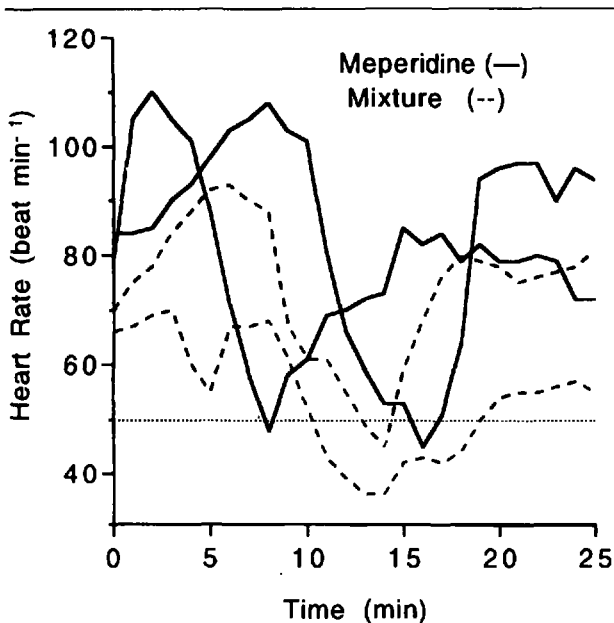


FIGURE 2 Examples of sudden decreases in HR following SA in four patients receiving either intrathecal meperidine or the mixture. Treatment with intravenous atropine was given when HR decreased $<50 \text{ beat} \cdot \text{min}^{-1}$. Subsequent treatment for hypotension was also given.

TABLE V Incidence of side effects, rescue treatment (colloid only/colloid plus vasopressor) and inadequate block (analgesics/general anaesthetic) in the three patient groups.

	Bupivacaine (n = 14)	Mixture (n = 14)	Meperidine (n = 14)
Bradycardia	0	4*	6†
Nausea/Vomiting	1	3	6*
Sedation	0	1	2
Rescue treatment (colloid/pressor)	5/1	5/2	6/3
Block inadequate (analgesia/GA)	0/0	0/2	2/6†

Number of patients shown. Significant difference compared with the bupivacaine group: * $P < 0.05$; † $P < 0.01$.

surgery by over one hour and three prolonged operations in which the duration block was insufficient (Table V). In the mixed group two patients required general anaesthesia because of low block (L_2 and L_4) (Table V). The incidence of nausea and vomiting was greatest in the meperidine group ($P < 0.05$) (Table V). One patient in the mixture group and two patients in the meperidine group became sedated. No patient in the study developed respiratory depression or pruritus.

Discussion

We found that intrathecal bupivacaine, the mixture and meperidine, in dosages used in this study, all produced

similar haemodynamic changes, with decreases in SAP, MAP, SVRI and CVP (Figure 1; Table IV). Bradycardias to $<50 \text{ bpm}$ requiring treatment with intravenous atropine occurred in those patients receiving meperidine or the mixture, but only when the height of sensory block was above T_7 . The use of meperidine was associated with a high incidence of nausea and vomiting, as well as failure to provide adequate analgesia in several cases. The rate of onset of sensory and motor block was slower in the meperidine group compared with the other groups.

The BoMed measures cardiac output to a high degree of repeatability, with a coefficient of variation of 4.7%.^{22,23} Thomas compared the BoMed with dye dilution measurements over a range of cardiac output and showed good trending ability, albeit in healthy volunteers.²⁴ However, the BoMed does not measure cardiac output accurately. When compared with thermodilution, limits of agreement range from an acceptable $\pm 22\%$ in otherwise healthy patients undergoing neurosurgery²³ to an unacceptable $\pm 50\%$ in critically ill patients.^{25,26} The inaccuracy in critically ill patients appears to be related to increases in lung water. Our patients had relatively normal cardiopulmonary physiology. However, accuracy was of limited importance in our study, because relative changes in cardiac output were analyzed.

Non-invasive arterial pressure monitoring was used in preference to direct intra-arterial pressure monitoring. Data was recorded at one minute intervals, which was sufficient to show the changes in SAP. Individual Dinamap readings have been shown to have a $\pm 10\%$ variability²¹ and in order to minimize errors, treatment was started only after two consecutive low readings.

The study was conducted over the first 25 min of block when the patients were undisturbed by surgical factors. Also, the main haemodynamic effects of block occurred within this period.

The most commonly studied dose of meperidine intrathecally is $1 \text{ mg} \cdot \text{kg}^{-1}$.^{4,5,7-11} However, Nguyen Thi *et al.*¹² have suggested recently that the safety margin of intrathecal meperidine is narrow and that respiratory depression and other side effects, such as sedation, nausea and vomiting, pruritus and urinary retention, can occur with doses as low as $0.5 \text{ mg} \cdot \text{kg}^{-1}$. Maurette *et al.*¹⁵ investigated meperidine concentrations in the ventricular cerebro-spinal fluid (CSF) and systemic circulation and found that concentrations in the CSF, and hence the respiratory centers, were mainly due to absorption from the systemic circulation, which peaked during the first hour after intrathecal injection. In order to limit the risk of respiratory depression and other side effects we reduced our dose of meperidine to $0.8 \text{ mg} \cdot \text{kg}^{-1}$. Few investigators have studied the use of

meperidine and local anaesthetic mixtures. Tauzin-Fin *et al.*⁹ compared intrathecal meperidine and prilocaine with meperidine alone and found that the mixture had a more rapid onset and longer duration of action. They also found that prilocaine increased the systemic absorption of meperidine from the CSF. Nguyen Thi *et al.*¹² found that adding meperidine to intrathecal bupivacaine improved postoperative analgesia. Hence, we also investigated a mixture of meperidine 0.4 mg·kg⁻¹ with heavy bupivacaine 7.5 mg, which had not been previously studied. By limiting the dosage of intrathecal drugs, we hoped to reduce the incidence of complications. We used hyperbaric bupivacaine 15 mg for our control group, because we had used this dosage in previous studies.²⁷⁻²⁹

Preloading before intrathecal injection was omitted from the study, as this would have affected our assessment of venous pressures. However, no patient was admitted to the study dehydrated and any hypotension was promptly treated. Treatment of hypotension was based on the findings of our previous studies.²⁷⁻²⁹ Most authors recommend treating hypotension when arterial pressure decreases by 20–30%. Colloid solution was chosen because of its relatively long half life in the circulation and previously we had shown²⁷ that the alpha agonist metaraminol was more effective than ephedrine in correcting hypotension in elderly patients.

Cozian *et al.*⁸ are the only investigators to have studied the haemodynamic effects of intrathecal meperidine. They measured radial arterial pressures and cardiac output by thermodilution in eight patients and found decreases in MAP, CVP and left atrial wedge pressure with no change in CI and HR. The SVRI decreased by 17% but this value did not reach statistical significance. Their level of sensory block was T₈ (range T₆–T₁₂), which was slightly lower than in the present study. Previously, we had reported that, following intrathecal hyperbaric bupivacaine,²⁸ there were decreases in SAP, MAP and SVRI, a moderate decrease in CVP of 1.5–2.0 cm H₂O, small decreases in SI and CI and increases in HR. These findings are similar to all three patient groups in the present study and the findings of Cozian *et al.*,⁸ suggesting that intrathecal meperidine causes a sympathetic block similar to that of intrathecal hyperbaric bupivacaine.

Our most important haemodynamic finding was that, following an initial increase in HR, severe bradycardia requiring treatment with intravenous atropine occurred in ten patients receiving meperidine or the mixture when the sensory block was above T₇ (Figure 2). This finding was absent from our previous study²⁸ and patients receiving hyperbaric bupivacaine in the present study, despite high levels of sensory block and hypotension.

Previous investigators have reported less severe decreases in HR following intrathecal meperidine.^{4,7,8} Cozian *et al.*⁸ suggested several possible mechanisms for the decrease in HR, of which complete cardiac sympathetic block seems unlikely because equally high sensory blocks with bupivacaine did not decrease HR. Their most plausible explanation was an opioid receptor effect, possibly related to central nausea and vomiting centers, acting through the vagus. However, regardless of the aetiology, the association of bradycardia with high intrathecal meperidine or mixed block is a very important clinical finding.

The block failed in two patients in the mixture group and in six patients in the meperidine group. Wide variations in level of sensory block were seen in both these groups (range T₃–L₄), the lower blocks being inadequate for surgery. This is in contrast to the bupivacaine group in which the sensory level of block was more consistent (T₂–T₁₁). In the present study, intrathecal injection was made with the patient in the lateral position, whereas in previous studies of meperidine the intrathecal injection was made with the patient sitting⁷⁻¹¹ and this may have affected the spread of meperidine in the CSF.

The duration of adequate meperidine block for surgery is reported to be 45–90 min.⁷⁻¹¹ Although the duration of block was not specifically measured in our study, it was too short in several patients in the meperidine group to allow time for the haemodynamic study before the intended surgery.

Nausea and vomiting is frequently reported with meperidine block^{4,5,7} and its incidence is higher than with local anaesthetic block.⁵ The height of meperidine block seemed to be a contributory factor to an incidence being 5% with low perineal blocks¹² and 17–55% with high blocks.^{4,5,7,12} In the study we had similar findings. As the incidence of nausea and vomiting was unrelated to other complication, such as hypotension, the most likely aetiology was a central opioid receptor effect.

The rate of onset of sensory and motor block was slower with meperidine than with bupivacaine, which is in keeping with previous studies.^{5,9} A potential advantage of intrathecal meperidine looked for in the study was a slower and more controllable onset of hypotension. However, the rate of onset of hypotension and, hence sympathetic block, with meperidine was not shown to be reduced.

In conclusion, we found no intraoperative advantages in using intrathecal meperidine or the mixture over heavy bupivacaine. Haemodynamically meperidine and the mixture were more unstable than hyperbaric bupivacaine with a high incidence of severe bradycardia, the extent of the block was less predictable and there was a high incidence of nausea and vomiting.

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