Spinal clonidine produces less urinary retention than spinal morphine

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Summary
We have conducted a double-blind, randomized study in two groups of 20 patients each, undergoing hip surgery during spinal anaesthesia, to compare the incidence of urinary retention after spinal morphine or clonidine. Patients received 0.5 % spinal bupivacaine 15 mg combined with either clonidine 75 μg or morphine 0.2 mg. After operation, patients were examined for micturition, bladder distension, or both; if they failed to void, they received naloxone 0.2 mg, and if bladder distension persisted, a catheter was inserted. At 12 h, all patients in the morphine group but only five in the clonidine group had bladder distension, and at 24 h this was present in seven and one patient in the morphine and clonidine groups, respectively (P < 0.001). Naloxone was given in 16 and one, and a catheter was placed in one and six patients in the morphine and clonidine groups, respectively (P < 0.001). We conclude that spinal clonidine impaired bladder function to a lesser extent than morphine. (Br. J. Anaesth. 1996; 76: 872–873)

Key words

Urinary retention is common after spinal administration of opioids [1, 2]; it may be distressing and is a serious limitation to the use of spinal opioids in daily practice, especially in orthopaedic surgery where patients do not routinely have a bladder catheter.

Spinal clonidine strengthens local anaesthetic block and produces postoperative analgesia, with side effects such as hypotension, bradycardia and sedation, but there are no reports of urinary retention [3]. If spinal clonidine is unlikely to promote urinary retention in postoperative patients, it could be an alternative to spinal morphine when bladder catheterization is considered harmful. In this study we tested the hypothesis that spinal bupivacaine with clonidine resulted in a lower frequency of urinary retention than spinal bupivacaine with morphine.

Methods and results
After obtaining informed consent and Ethics Committee approval, we studied 40 ASA II–III patients undergoing hip surgery. Patients were excluded if they were receiving drugs which may interfere with clonidine or morphine, or if they had pathology of the urinary tract.

Patients were premedicated with flunitrazepam 1 mg orally, and allocated randomly to one of two groups. All received spinal 0.5 % isobaric bupivacaine 15 mg (3 ml); in addition, patients were given spinal morphine 0.2 mg (group M) or clonidine 75 μg (group C). Maximal extension of the anaesthetic block was evaluated by pinprick.

Ringer’s lactate solution was infused at a rate of 15 ml kg⁻¹ h⁻¹ during surgery and at 3 ml kg⁻¹ h⁻¹ after operation for 24 h. Blood loss was replaced during surgery with colloids and autologous blood. When hypotension (decrease in systolic arterial pressure >30 % of control value), or bradycardia (heart rate < 50 beat min⁻¹) occurred, doses of ephedrine 6–12 mg i.v. were administered. Atropine was not used. Postoperative pain treatment included propacetamol i.v.

After operation, patients were examined at 6, 12 and 24 h by a blinded observer. At each time, micturition within the preceding hours was noted, and the bladder was examined for distension. When bladder distension was noted, patients were asked to void. If they failed, or if a second examination revealed persistent bladder distension after incomplete micturition, they received naloxone 0.2 mg. If naloxone did not result in complete voiding, the bladder was catheterized transiently.

Results are expressed as mean (sd). Data were analysed using one-way ANOVA and a Student’s t test, and chi-square test and Yates’ correction and Fisher’s exact test where appropriate.

There were 20 patients in each group. The two groups were comparable in age (group C, 64.8 (range 41–77) yr and group M, 61.2 (44–70) yr), weight (77.6 (sd 13.3) kg and 72.9 (11.4) kg) and height (169 (5) cm and 166 (6) cm) (ns). Group C included 16 men and four women and group M, 13 men and seven women. Maximum extension of block was comparable in the two groups. Duration of surgery was 95.8 (8.7) min and 100.9 (15.7) min in groups C and M, respectively (ns). Mean amounts of i.v. infusions were comparable in the peroperative period.

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(2684 (730) ml and 2875 (428) ml in groups C and M, respectively, and after operation (6055 (1392) ml and 6500 (875) ml in groups C and M). Ephedrine was given to five patients in group C and to three in group M.

At 6 h no patient had experienced spontaneous micturition. At 12 h, 17 patients in group C but none in group M had experienced spontaneous micturition. At 24 h, 20 patients in group C and 12 in group M reported micturition within the preceding 12 h (P < 0.001).

Bladder distension was noticed in two patients in group M and in none in group C at 6 h (table 1). At 12 h, all patients in group M had bladder distension compared with only five patients in group C; at 24 h, seven patients in group M and one in group C had bladder distension (P < 0.001) (table 1). All patients were able to void when they had bladder distension but micturition was incomplete in one patient in group C and in 14 in group M at 12 h (P < 0.05), and in two patients in group M at 24 h. Consequently, naloxone was given to 16 and one patient in groups M and C, respectively (P < 0.001).

Bladder catheterization was performed after naloxone in only one patient who failed to empty his bladder at 12 h (group C), while four and two patients were catheterized at 12 h and 24 h, respectively, in group M (P < 0.001) (table 1).

### Comment

We have found that the incidence of bladder dysfunction was less when spinal clonidine, instead of morphine, was given with spinal bupivacaine.

A difference in urinary output between the two groups might have explained the difference in urinary retention. We did not measure urinary volumes but previous studies indicated that spinal morphine may decrease urinary output by release of arginine vasopressin [4] and conversely that clonidine has a diuretic effect [5]. Such effects would have favoured bladder distension in group C whereas we observed the opposite effect.

### Spinal and extradural opioids

Spinal and extradural opioids are considered risk factors for urinary retention [2]. Extradural morphine induces difficulties in micturition in healthy volunteers [6]. Urinary retention ranges from 42 % to 62 % in patients who have received spinal or extradural opioids [1, 2].

Activation of the parasympathetic pathways to the detrusor and relief of the somatic input to the external bladder sphincter induce micturition. Activation of the sympathetic outflow to the urinary tract promotes an increase in urethral resistances and depresses detrusor contraction, favouring urinary retention. Spinal opioids acting on spinal or supraspinal mu receptors may depress efferent parasympathetic outflow to the bladder, inhibiting detrusor contractions and micturition reflex for several hours after spinal administration.

Spinal administration of clonidine results in less difficulties in micturition. Alpha2 adrenergic agonists have complex and potentially opposite effects on bladder emptying. Peripheral α2 adrenergic receptors are documented in the bladder wall, especially at the level of the trigone [2]. Their stimulation induces contraction of the smooth muscle fibres of the internal sphincter tone and therefore increases urethral resistances and impairs voiding. As a small dose was given intrathecally, a peripheral effect of clonidine is unlikely. Spinal clonidine decreases sympathetic tone through a supraspinal and spinal effect (on preganglionic sympathetic neurones) which could facilitate micturition. Indeed, administration of clonidine stimulates bladder motility in cats by depressing sympathetic outflow to the bladder [7].

### References


