Clinical Reports

Respiratory depression associated with meperidine spinal anaesthesia

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Intrathecal meperidine administration can provide surgical anaesthesia and postoperative analgesia for about two to six hours. We have observed two cases of respiratory depression associated with meperidine spinal anaesthesia. An 81-yr-old woman received 50 mg intrathecal meperidine for inguinal hernia repair. Supplemental oxygen was administered at 3 L·min⁻¹ by nasal prongs. About 40 min later, the patient's oxygen saturation decreased from 97% to 87% and she was asked to take big breaths. She responded immediately and oxygen saturation returned to 97%. Two more similar episodes followed in the next five minutes. Naloxone 0.1 mg iv was administered and the oxygen saturation remained at 96–97% until completion of surgery about 15 min later. She had an uneventful recovery. A 24-yr-old healthy woman presented for tubal ligation eight hours after vaginal delivery of an infant. The surgical procedure was performed under spinal anaesthesia produced by 50 mg meperidine. During surgery, midazolam 2 mg iv was given for anxiolysis. About five minutes after admission to the postanesthesia care unit, the patient's respiratory rate decreased to ten breaths per minute and oxygen saturation decreased to 89%. Supplemental oxygen at 3 L·min⁻¹ was administered by nasal prongs. The patient was encouraged to take big breaths and the arterial oxygen saturation rapidly increased to 98–99%. Forty minutes later, nasal oxygen was discontinued. The patient maintained her oxygen saturation while breathing room air. She was then discharged to the ward and had an uneventful recovery course. We recommend that a patient's respiratory variables and oxygenation be closely mon-

itored for at least one hour after intrathecal meperidine administration.

La méperidine rachidienne procure généralement une anesthésie chirurgicale et une analgésie postopératoire d'une durée approximative de deux à six heures. Nous avons observé deux cas de dépression respiratoire associés à l'anesthésie rachidienne à la méperidine. Une femme âgée de 81 ans reçoit de la méperidine 50 mg en rachianesthésie pour une cure de hernie inguinale. On lui administre de l'oxygène 3 L·min⁻¹ par sonde nasale. Après 40 minutes, la saturation d'oxygène baisse de 97% à 87% et on lui demande de prendre des respirations profondes. Elle s'exécute immédiatement et la saturation revient à 97%. Deux épisodes identiques surviennent pendant les cinq minutes suivantes. De la naloxone 0,1 mg iv est administrée et la saturation se maintient à 96–97% jusqu'à la fin de la chirurgie, environ 15 minutes plus tard. Elle récupère sans séquelles. Une femme de 24 ans en bonne santé est programmée pour une ligature tubaire huit heures après un accouchement normal. La chirurgie est réalisée sous rachianesthésie à la méperidine 50 mg. Pendant la chirurgie, on administre du midazolam 2 mg pour calmer l'anxiété. Cinq minutes après l'admission à la salle de réveil, la fréquence respiratoire baisse à dix respirations à la minute et la saturation en oxygène à 89%. On administre de l'oxygène 3 L·min⁻¹ par canule nasale. On incite la patiente à prendre des respirations profondes et la saturation en oxygène remonte à 98–99%. Quarante minutes plus tard, on arrête l'administration de l'oxygène nasal. La patiente maintient sa saturation en respirant de l'air. Elle retourne au service et récupère sans incident. Nous recommandons un monitoring étroit des signes respiratoires et de l'oxygénation pendant au moins une heure après l'administration rachidienne de méperidine.

Key words

ANAESTHESIA, LOCAL: meperidine;

ANAESTHETIC TECHNIQUES, SPINAL: meperidine.

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Subarachnoid administration of meperidine can produce spinal anaesthesia profound enough for surgical procedures.^{1–5} One of the major benefits of meperidine spinal anaesthesia is postoperative analgesia for two to six hours,

even after return of tactile sensation and motor function.^{5,6} Although respiratory depression is recognized as a possible complication as with any epidural/spinal opioid administration, there are few reports of such complications with meperidine spinal anaesthesia.^{3,7} Since the introduction of this technique into our anaesthesia practice over the last year, we have observed two cases of respiratory depression associated with spinal anaesthesia induced with 50 mg intrathecal meperidine.

Case 1

An 81-yr-old woman was admitted for right inguinal hernia repair. The patient had two previous myocardial infarctions 13 and 14 yr before the current admission. She had occasional exertional angina that was readily relieved by sublingual nitroglycerine. The patient smoked about 20 cigarettes per day until her first myocardial infarction and then changed to smoking 1–2 cigarettes per day. Her height was 162 cm and weight was 73 kg. Physical examination on admission did not reveal any abnormality. Her ECG showed low T voltage in limb leads, V₅ and V₆ along with nonspecific ST changes. After discussion of anaesthetic options, meperidine spinal anaesthesia was agreed upon.

After receiving 10 mg diazepam *po* as a preoperative sedative, the patient was conscious and cooperative on arrival in the operating room. Monitors were applied and supplemental oxygen was started at 3 L · min⁻¹ via nasal prongs. With the patient in the right lateral position, a #25 Whitacre needle was inserted into the subarachnoid space at the L₃–L₄ interspace and 50 mg preservative free meperidine (5%) with an equal volume of 10% dextrose solution was administered over ten seconds. After remaining on her side for two minutes, the patient was returned to the supine position. Ten minutes later, sensory loss was found to have extended to T₈ and surgery started. Five minutes later, the blood pressure decreased from 150/90 to 100/65. After administration of intravenous fluid and 10 mg ephedrine *iv*, blood pressure was 120/70 mmHg and remained at that level for the remainder of the case.

About 40 min after subarachnoid meperidine administration, the patient's pulse oximeter showed a decrease of oxygen saturation from 97% to 87%. As respiration was noted to be shallow, the patient was instructed to take big breaths. She responded immediately and the oxygen saturation returned to 97% in less than a minute. Two more similar episodes followed in the subsequent five minutes. Naloxone 0.1 mg *iv* was administered and the patient's oxygen saturation remained at 96–97% until completion of surgery about 15 min later. The patient did not receive any other sedative or analgesic medication during surgery. She closed her eyes at times but re-

sponded immediately to voice and she cooperated with commands to take big breaths.

After surgery, the patient was observed in the post-anaesthesia care unit (PACU). During the first hour, she maintained oxygen saturation in the 95–97% range except for one brief decrease to 89% associated with a reduction in respiratory rate to 12 L · min⁻¹ from the 14–16 · min⁻¹ range. No intervention was required apart from reminders to take big breaths.

Supplemental oxygen was administered for 24 hr after the end of surgery by nasal prongs. The patient did not complain of pain until 6.5 hr after the end of surgery and that was successfully treated with 30 mg ketorolac *im*. Another dose of ketorolac was needed 6.5 hr later. The next day, the patient's surgical pain was controlled by oral plain acetaminophen tablets. The rest of her hospital course was uneventful.

Case 2

A 24-yr-old woman presented for tubal ligation eight hours after vaginal delivery. She smoked about 10 cigarettes per day. Her only medication during pregnancy was iron and vitamin supplements. The patient's height was 157 cm and weight was 70 kg. After discussion of anaesthetic options, meperidine spinal anaesthesia was agreed upon.

After applying monitors and with the patient in the right lateral position, a #25 Whitacre needle was inserted into the subarachnoid space at the L₃–L₄ interspace and 50 mg preservative free meperidine (5%) with an equal volume of 10% dextrose solution was administered over ten seconds. Ten minutes later, loss of sensation was found to extend to T₇ and surgery started. Five minutes later, the patient complained of feeling dizzy. The blood pressure had not changed. Midazolam 2 mg *iv* was given for anxiolysis. Surgery was completed ten minutes later and the patient was transferred to PACU where she was fully conscious, her blood pressure was 110/58 mmHg, respiratory rate was 16 min⁻¹ and heart rate was 78 · min⁻¹. Five minutes later, her respiratory rate decreased to ten breaths per minute and her arterial oxygen saturation decreased to 89%. Supplemental oxygen at 3 L · min⁻¹ was administered by nasal prongs and the patient was encouraged to take big breaths with which her arterial oxygen saturation rapidly increased to 98–99%. Forty minutes later, with the patient's respiratory rate at 16 · min⁻¹, nasal oxygen was discontinued. She maintained her oxygen saturation for the next hour while breathing room air. She was then discharged to the ward and had an uneventful recovery course.

Discussion

Meperidine spinal anaesthesia has been reported to be

efficacious for surgical procedures of lower limb, perineum, inguinal region, and tubal ligation providing good surgical conditions and postoperative analgesia.¹⁻⁵ Since meperidine spinal anaesthesia does not require mixing an opioid with a local anaesthetic, it is easy to prepare and is cost-effective. Respiratory depression after intrathecal meperidine administration has been reported previously.^{3,7} A 21-yr-old female with a weight of 44.5 kg received 100 mg intrathecal meperidine. She became stuporous and had irregular respiration with less than ten breaths per minute. In that case, analgesia had extended to the T₂ dermatome when the complication was detected. The patient required intermittent positive-pressure ventilation for about 30 min.³

Brownridge *et al.* reported respiratory depression after accidental subarachnoid meperidine in a 24-yr-old woman after elective repeat Caesarean section and tubal ligation.⁷ Meperidine was administered through an epidural catheter. There was no complication after the first 50-mg dose. Four hours later, the patient requested more analgesia and a second 50-mg dose was administered. Shortly afterwards, the patient felt nauseated, vomited, and five minutes later, became drowsy, unresponsive, cyanosed, and had slow, deep gasping respiration. She regained consciousness after insertion of an airway, intermittent positive-pressure ventilation, and attempted endotracheal intubation. She was then given naloxone 0.4 mg *iv*. The patient recovered without further respiratory problems. The epidural catheter was subsequently documented by x-ray and contrast material to be in the subarachnoid space.

Both of these previous cases occurred after a total dose of 100 mg meperidine into the subarachnoid space and both were associated with a marked reduction in the level of consciousness and the need for positive-pressure ventilation. The two patients that we reported received only 50 mg meperidine and both remained conscious at all times. Respiratory depression was manifested by hypoventilation with resultant hypoxaemia. In the first case, a small intravenous dose of naloxone successfully corrected the respiratory depression without affecting surgical anaesthesia or postoperative analgesia. The second, a younger patient, required only supplemental oxygen and verbal instruction to take big breaths.

Our first patient was 81-yr-old and received 0.7 mg · kg⁻¹ meperidine intrathecally. Hypoxaemia occurred in spite of concurrent supplemental oxygen administration at 3 L · min⁻¹ by nasal prongs. Two previous studies of meperidine spinal anaesthesia have included patients aged up to 83 yr who had received 1 mg · kg⁻¹ intrathecal meperidine.^{1,2} There was no respiratory depression reported in either of these two series.

Our patients received midazolam sedation during

surgery. Midazolam has been shown to cause decreases in tidal volume and minute ventilation in patients who had spinal anaesthesia.⁸ Sangarlangkarn *et al.*³ have reported drowsiness in 77% of patients who received meperidine spinal anaesthesia. Further studies are needed to determine the interactions between benzodiazepine sedation and meperidine spinal anaesthesia.

In the two previous reports and our two patients, respiratory depression was noted within the first hour after subarachnoid injection. This is consistent with the data from Maurette *et al.*,⁹ who demonstrated that peak ventricular cerebral spinal fluid (CSF) meperidine concentrations occurred within one hour in three of the four subjects studied. They postulated that this peak was due to meperidine absorption into the systemic circulation and redistribution back into the CSF. No delayed secondary peak was demonstrated, and no report of delayed respiratory depression akin to that witnessed with intrathecal or epidural morphine has ever been reported.

We recommend that a patient's respiratory variables and oxygenation be closely monitored for at least one hour after intrathecal meperidine administration.

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