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Short communications

Effects of epidural buprenorphine on bowel movement following gynecological surgery

YOSHITAKA TOYODA[1], HIROSHI MORISAKI[2], TOMOKO YOROZU[1], and TOSHIYUKI SHIGEMATSU[1]

[1] Department of Anesthesia, Tokyo Metropolitan Otsuka Hospital, 2-8-1 Minami-otsuka, Toshima-ku, Tokyo, 170 Japan
[2] Department of Anesthesiology, School of Medicine, Keio University, 35 Shinanomachi, Shinjuku-ku Tokyo, 160 Japan

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While epidural administration of opioids has been widely employed to relieve either acute or chronic pain [1], this method likely disturbs physiological bowel motility [2,3]. The question as to whether or not buprenorphine epidurally administered modifies the bowel movement after abdominal surgery has not yet been answered. We compared the effects of postoperative lumbar epidural infusion and intermittent intramuscular administration of buprenorphine, on bowel motility following gynecological surgery.

After obtaining approval from the hospital ethics committee and informed consent from all participants, 30 adult patients (ASA grade 1 to 2) undergoing elective abdominal total hysterectomy under the diagnosis of myoma uteri were included in the study. None of the patients had a history of either abnormal bowel habits, taking any drugs known to influence gastrointestinal motility, or abdominal surgery. All patients were given ranitidine 150 mg orally at 9:00 p.m. on the day before surgery, and hydroxyzine 50 mg with atropine 0.5 mg, i.m. 60 min before surgery. The patients were then randomly allocated to two groups according to the postoperative analgesic management: control group (n = 15) or epidural group (n = 15). In the latter group, an epidural catheter was inserted at the T12/L1 or L1/2 level prior to the induction of general anesthesia. The epidural space was identified by the loss-of-resistance method, followed by injection of 5 mL of 1% mepivacaine.

Address correspondence to: H. Morisaki
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All patients received a standardized inhalational general anesthetic. General anesthesia was induced by intravenous administration of thiopental 3–4 mg/kg, followed by intravenous injection of succinylcholine 1 mg/kg to facilitate tracheal intubation. Anesthesia was maintained with 67% nitrous oxide in oxygen with sevoflurane in both groups. Muscle relaxation during surgery was achieved with intermittent injection of vecuronium bromide. At the end of anesthesia, residual muscle relaxation was fully antagonized with the injection of neostigmine 2.5 mg and atropine sulfate 1.0 mg. Postoperatively, the epidural catheter in the epidural group was connected to a continuous infusion syringe pump (Terfusion, Terumo, Tokyo, Japan), set at an infusion rate of 0.008 mg/h of buprenorphine in normal saline. The patients in the control group were administered buprenorphine 0.1 mg, i.m. twice during the first 24 h. As a supplemental analgesic, indomethacin suppository (50 mg) was given on request. Twenty-four hours following surgery, blood samples were taken to measure serum potassium and buprenorphine concentration. Serum potassium was measured by a flame-photometer (Hitachi-710, Tokyo, Japan), and the buprenorphine concentration was measured by radioimmunoassay [4]. The intra- and inter-assay coefficients of variation of the latter method were 6.4% and 19.3%, respectively. The time to the first passage of flatus was recorded by the ward nurses. Student's unpaired t-test and the Mann-Whitney U-test were employed for statistical analysis of the results where appropriate. A P value of less than 0.05 was considered statistically significant.

The demographic data of the patients were comparable, as shown in Table 1. Eight patients in the epidural group and two in the control group required indomethacin suppository during the first 24 h. The plasma concentration of buprenorphine at 24 h following the surgery in the epidural group was significantly higher than those in the control group. The patients

Table 1. Patient characteristics and plasma buprenorphine concentration 24 hours following surgery

	Age (years)	Body weight (kg)	Duration of surgery (h)	Serum potassium (mEq/L)	Plasma buprenorphine concentration (ng/mL)
Epidural group	45.6 ± 5.0	52.6 ± 7.3	71.4 ± 12.2	4.2 ± 0.3	0.24 ± 0.06*
Control group	42.8 ± 7.7	52.0 ± 5.1	65.2 ± 19.5	4.1 ± 0.4	<0.1 (less than assay limits)

Data are expressed as mean ± SD.
* $P < 0.05$ vs. control group.

in the epidural group required a significantly longer duration before the first passage of flatus compared with the control group (50.7 ± 10.6 h vs. 35.7 ± 8.4 h; $P < 0.05$).

Previous studies showed that the first passage of flatus and/or feces is reliable and correlates well with the return of bowel movements, measured by radiopaque markers [5–7]. This study demonstrates that the epidural buprenorphine likely delayed the return of bowel motility following gynecological surgery more so than intramuscular injection. While we chose gynecological surgery to obviate the confounding factors of the duration of intestinal handling, the difference between these two routes of administration might be augmented in cases of colonic surgery. Other factors such as anesthetic agents, anticholinergic drug, and serum potassium level, which could modify bowel movement, were given to similar amounts in this study [8–11]. Among several mechanisms to account for the inhibition of gastrointestinal motility following abdominal surgery [3], the most accepted hypothesis is the activation of spinal reflexes originating from the abdominal cavity, involving sympathetic efferent nerves to the bowel [12,13]. Thus, buprenorphine, a highly lipophilic substance, could be absorbed into the spinal cord with ease, resulting in modification of the spinal response [14]. While the plasma concentration at one spot may not directly reflect the overall effects of buprenorphine, systemic intramuscular injection would be more assured method than *ia* epidural route to obtain both postoperative pain relief and the earlier return of bowel movement following gynecological surgery. Although a combination of epidural bupivacaine with morphine is unlikely to have shortened the duration of postoperative ileus [15], further studies are warranted to examine the effects of local anesthetics.

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