J Anesth (1995) 9:357–359

Journal of Anesthesia

Intraperitoneum insufflation of carbon dioxide increases epidural pressure in laparoscopic cholecystectomy

HONG-LIN DU, MASAKAZU HAYASHITA, and KAZUO HANAOKA

Department of Anesthesiology, University of Tokyo, 7-3-1 Hongo, Tokyo, 113 Japan

Key words: Laparoscopic cholecystectomy, Epidural space, Cerebrospinal fluid pressure

Introduction

Laparoscopic cholecystectomy is being performed with increasing frequency because of its low level of postoperative pain, faster recovery, and shorter hospitalization [1], but data recorded during this procedure indicate that it affects some physiologic functions. For example, intraperitoneum insufflation with carbon dioxide (CO_2) often has a depressive effect on circulation and respiration [2, 3]. However, little is known about the effect of CO_2 pneumoperitoneum on intracranial pressure or cerebrospinal fluid pressure, partly because of the difficulty in measuring these parameters. In this study, we continuously measured epidural pressure after injecting a known volume of local anesthetic at a fixed speed. We used epidural pressure as an indirect measure of cerebrospinal fluid pressure when it was relatively stable after epidural injection [4–6]. The influence of CO_2 pneumoperitoneum on epidural pressure in laparoscopic cholecystectomy was evaluated.

Materials and methods

Eight patients (four men and four women) were involved in the study. They all had chronic cholecystitis and/or gallbladder stone, and were scheduled for laparoscopic cholecystectomy. Their mean age was 35 ± 12 (SD) years, and mean weight was 58 ± 11 kg, ASA I or II. Informed consent was obtained from each patient on the day before surgery. All patients were premedicated with intramuscular atropine ($0.01\,mg \cdot kg^{-1}$) and hydroxyzine ($1\,mg \cdot kg^{-1}$) 30 min before arriving in the operating room. Before the induction of general anesthesia, epidural puncture was performed at the T8–T9 interval with a 17 G Tuohy needle. That the epidural space had been entered was confirmed by loss of resistance to normal saline injection. An 18 G epidural catheter (Terumo, Tokyo, Japan; 0.985 mm inner diameter, 953 mm length) was advanced 3 cm cephalad. A 25 G needle (20 mm length) was connected to the outer end of the epidural catheter. A pressure transducer was then connected with the 25 G needle through a three-way stopcock. Using this stopcock, the epidural catheter could be connected either to the transducer for monitoring epidural pressure or to the local anesthetic injector for injecting anesthetic. General anesthesia was induced with thiopentone ($5\,mg \cdot kg^{-1}$ i.v.) and endotracheal intubation was performed with the facilitation of intravenous vecuronium ($0.2\,mg \cdot kg^{-1}$). The patient's respiration was mechanically controlled with a tidal volume of $12\,mg \cdot kg^{-1}$ at a rate of 10 breaths·min^{-1}. General anesthesia was maintained by 0.5%–1.5% isoflurane with 40% oxygen in nitrogen. One per cent mepivacaine was injected into the epidural space when necessary.

After anesthesia induction, the operating table was adjusted to the horizontal position. The first 5 ml mepivacaine was injected into the epidural space at $0.5\,ml \cdot s^{-1}$. Epidural pressure was monitored continuously. It increased immediately after epidural injection and then decreased gradually. It remained relatively stable from 3–5 min to 20 min after the injection. Epidural pressure was sampled every minute from 6 min to 20 min after epidural injection to calculate its coefficient of variation.

The second 5 ml mepivacaine was injected into the epidural space at $0.5\,ml \cdot s^{-1}$ 6 min before intraperitoneum insufflation. Epidural pressure was monitored continuously for 20 min after the injection. The peritoneal cavity was insufflated with CO_2 and intraperitoneal pressure was maintained at 12–15 mmHg throughout the procedure. For statistical comparison

Address correspondence to: H.-L. Du

Received for publication on December 13, 1994; accepted on April 27, 1995

between epidural pressure and intraperitoneal pressure, the values immediately before and 5 min after the start of the insufflation were used. Arterial blood was sampled and $PaCO_2$ was measured immediately before and 5 min after the start of intraperitoneum insufflation.

Epidural pressure was referenced at the level of the outer ear tract. Intraperitoneal pressure was measured with a differential pressure transducer. Both pressures were read at the end of expiration. Data were presented as mean ± SD. Comparison between before and 5 min after the start of intraperitoneum insufflation was conducted by paired *t*-test. Statistical significance was accepted at $P < 0.05$.

Results

The coefficient of variation of epidural pressure sampled every minute from 6 min to 20 min after the first epidural injection was 2.4 ± 0.3%. Intraperitoneum insufflation with CO_2 resulted in simultaneous increases in epidural pressure and intraperitoneal pressure (Fig. 1). Epidural pressure remained elevated during the 15 min of CO_2 pneumoperitoneum when it was being monitored. When we compared the increases in intraperitoneal and epidural pressure 5 min after the start of CO_2 pneumoperitoneum, it was found that the increase in epidural pressure was about one-third of the increase in intraperitoneal pressure (Table 1). $PaCO_2$ did not change significantly 5 min after the start of CO_2 pneumoperitoneum when compared with that before CO_2 pneumoperitoneum.

Table 1. Effects of CO_2 pneumoperitoneum on intraperitoneal pressure, epidural pressure and $PaCO_2$

	Before insufflation	5 min after start of insufflation
Intraperitoneal pressure	0	13.9 ± 11*
Epidural pressure	8.4 ± 1.3	13.2 ± 1.6*
$PaCO_2$	33.2 ± 2.1	36.9 ± 4.5

The unit of pressure is mmHg; $n = 8$; mean ± SD.
* $P < 0.01$ vs. before insufflation.

Discussion

This study showed that the increase in intraperitoneal pressure caused by intraperitoneum insufflation with CO_2 resulted in a simultaneous increase in epidural pressure. The increase in epidural pressure was about one-third of the increase in intraperitoneal pressure.

Epidural pressure after epidural injection may be affected by factors such as the volume injected and the injecting speed. By controlling these factors, epidural pressure can be measured with good reproducibility and may remain stable for several minutes [4–6]. Then as the injected fluid is absorbed or fluxes out of the epidural space, the epidural pressure gradually decreases. In this study, we found that by controlling the volume injected and the injecting speed, the epidural pressure remained relatively stable from 6 min to 20 min after injection of local anesthetic into the epidural space (coefficient of variation 2.4 ± 0.3%). Influence of intraperitoneum insufflation with CO_2 on epidural pressure could therefore be investigated during this period.

This stable epidural pressure has been shown to have a good relationship with cerebrospinal fluid pressure in both humans and animals [4–6]. This finding is acceptable because there is only elastic dura mater between the epidural space and the subarachnoid space. Epidural pressure and cerebrospinal fluid pressure may reach equilibration through the dura mater under stable conditions when fluid exists in the epidural space to transmit pressure [4]. Therefore, our results suggest that caution should be taken when CO_2 pneumoperitoneum is conducted in patients with low intracranial compliance.

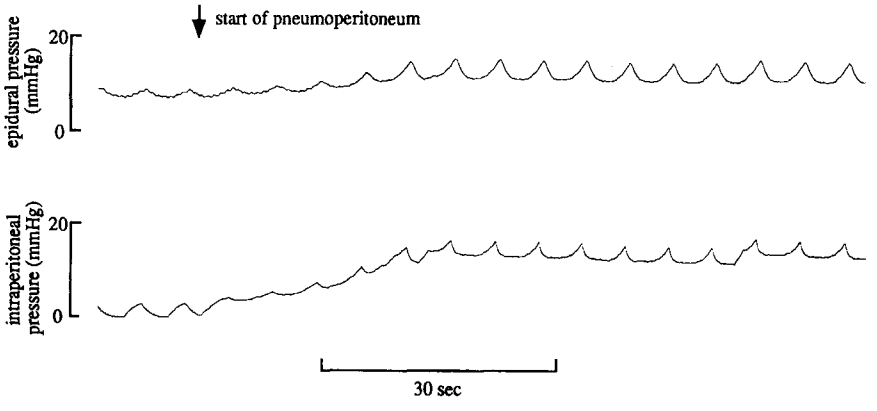

Fig. 1. Responses of intraperitoneal pressure and epidural pressure to CO_2 pneumoperitoneum

The reason for the increase in epidural pressure caused by CO_2 pneumoperitoneum is not well known. The elevated intraperitoneal pressure produced by intraperitoneum insufflation with CO_2 may transmit directly to the epidural space through intervertebral foramina, and/or indirectly increase the epidural pressure by interfering with the venous return of the epidural space vein [7]. As $PaCO_2$ did not change significantly 5 min after the start of insufflation, the increase in epidural pressure cannot be attributed to the $PaCO_2$ change.

In summary, we have found that the increase in intraperitoneal pressure caused by intraperitoneum insufflation with CO_2 resulted in a simultaneous increase in epidural pressure. Since epidural pressure reflects cerebrospinal fluid pressure, it is suggested that caution should be taken when CO_2 pneumoperitoneum is conducted in patients with low intracranial compliance.

References

1. Wolfe BM, Gardiner B, Frey CF (1991) Laparoscopic cholecystectomy. A remarkable development (Editorial). JAMA 265: 1573–1574
2. Critchley LAH, Critchley JAJH, Gin T (1993) Haemodynamic changes in patients undergoing laparoscopic cholecystectomy: Measurement by transthoracic electrical bioimpedance. Br J Anaesth 70:681–683
3. Nyarwaya JB, Mazoit JX, Samii K (1994) Are pulse oximetry and end-tidal carbon dioxide tension monitoring reliable during laparoscopic surgery? Anaesthesia 49:775–778
4. Du HL, Suwa K, Hanaoka K (1994) Estimation of cerebrospinal fluid pressure via the lumbar epidural space by the equilibration method. J Anesth 8:182–187
5. Asano M, Kosaka Y (1986) Changes of epidural pressure and cerebrospinal fluid pressure during abdominal surgery (in Japanese with English abstract). Masui (Jpn J Anesthesiol) 35:1694–1700
6. Asano M, Oh-Oka T, Kosaka Y (1987) Relation between epidural pressure and cerebrospinal fluid pressure in dogs (in Japanese with English abstract). Masui (Jpn J Anesthesiol) 36:89–93
7. Usubiaga JE, Moya F, Usubiaga LE (1967) Effect of thoracic and abdominal pressure changes on the epidural space pressure. Br J Anaesth 39:612–618