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Postthoracotomy Pain and Pulmonary Function Following Epidural and Systemic Morphine

Mark Shulman, M.D., Alan N. Sandler, M.Sc., M.B., Ch.B., F.R.C.P.(C).,†*
John W. Bradley, B.Sc., M.D., F.R.C.P.(C).,† Patricia S. Young, R.N.,‡
John Brebner, M.D., Ph.D., F.R.C.P.(C).§

Thirty patients undergoing thoracotomy for lung resection were entered in a randomized, double-blind trial comparing the effects of epidural (E) *versus* intravenous (iv) morphine on postoperative pain and pulmonary function. Postoperatively the patients were given repeated doses of either 5.0 mg of morphine epidurally or 0.05–0.07 mg/kg morphine intravenously until there were no further spontaneous complaints of pain. Two, 8, and 24 h postoperatively, the following indices were measured: linear analogue pain score, somnolence score, vital signs, arterial Pa_{O_2}, Pa_{CO_2} and *p*H, forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), and peak expiratory flow rate (PEFR). Patients receiving epidural morphine had significantly less pain at 2 h ($P < 0.01$) and 8 h ($P < 0.004$) postoperatively. There was no difference in vital signs except for significantly slower respiratory rates at 2 h ($P < 0.04$), 8 h ($P < 0.02$) and 24 h ($P < 0.01$) in the epidural group. No significant differences occurred in the somnolence scores or blood–gas measurements, which were within normal limits. The epidural morphine group has significantly less decrease in both FVC at 2 h (E -1.8 ± 2 l, iv -2.5 ± 0.2 l, $P < 0.03$), 8 h (E -1.4 ± 0.2 l, iv -2.1 ± 0.2 l, $P < 0.01$), and 24 h (E -1.2 ± 0.2 l, iv -2.0 ± 0.2 l, $P < 0.02$), and FEV_1 at 2 h (E -1.3 ± 0.2 l, iv -1.9 ± 0.2 l, $P < 0.04$), 8 h (E -1.0 ± 0.2 l, iv -1.7 ± 0.2 l, $P < 0.01$), and 24 h (E -0.8 ± 0.1 l, iv -1.5 ± 0.2 l, $P < 0.01$). In addition, the epidural morphine group had significantly less decrease in PEFR at 24 h (E -134 ± 29 l·min^{-1}, iv -238 ± 30 l·min^{-1}, $P < 0.03$). The authors conclude that lumbar epidural morphine is highly effective in alleviating pain and improving respiratory function in postthoracotomy patients. (Key words: Analgesics: morphine. Anesthesia: thoracic. Anesthetic techniques: epidural, morphine. Lung: pulmonary function. Pain: postoperative.)

PATIENTS UNDERGOING THORACOTOMY experience severe postoperative pain and marked respiratory impairment.[1–3] Several studies have demonstrated that epidural morphine produces prolonged analgesia without sedation, interference with neuromuscular function, or depression of the sympathetic nervous system.[4–7]

However, evidence obtained primarily from nonthoracic surgery presents conflicting views from both double-blind[8–13] and noncontrolled studies[14–18] as to whether epidural narcotics provided better postoperative pain relief than parenteral narcotics.

Narcotics administered via the lumbar approach in the epidural space have been shown to provide postoperative analgesia[19] and improve respiratory mechanics after thoracotomy.[14]

This study was undertaken to assess which route of administration of morphine, either epidural or intravenous, produced improved postoperative pain relief and pulmonary function.

Methods

Thirty patients were chosen for the study, and all gave informed consent. The protocol was approved by the Human Experimentation Committee of the University of Toronto. The patients ranged in age from 37 to 73 yr. Patients under 45 kg and over 100 kg were excluded. The study was randomized with the use of a table of random numbers. A sealed copy of the master code for the randomization was available to the investigators in the event of severe respiratory depression. Personnel taking measurements and personnel injecting drugs were kept separate.

The epidural morphine was prepared as 5 mg powdered base in 20 ml preservative-free normal saline. Patient and observer blinding was achieved by injecting via both the intravenous and epidural routes whenever the patients required analgesia postoperatively. For the group receiving intravenous narcotics, 0.05–0.07 mg/kg morphine was injected intravenously and 20 ml of normal saline was injected epidurally, whereas the epidural group received 5 mg of morphine in 20 ml normal saline epidurally and 0.05–0.07 ml/kg of normal saline intravenously.

Preoperative and postoperative pulmonary function was assessed using a Wright disposable peak flow meter (Wright Peak Flow Meter®; Ohio Medi-Shield®) to measure peak expiratory flow rate (PEFR), and a Breon model 2400 Spirometer® (Breon Laboratories Inc. New York) to measure forced vital capacity (FVC) and forced expiratory volume in the first second (FEV_1). All pulmonary function tests were done with the patients sitting upright in a vertical position with nose clips applied.

* Research Fellow. Current position: Assistant Professor, Stanford University Hospital.
† Lecturer, University of Toronto.
‡ Research Assistant, University of Toronto.
§ Associate Professor, University of Toronto.

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Address correspondence to Dr. A. N. Sandler, Department of Anesthesiology, University of Toronto, Toronto General Hospital, 101 College Street, Toronto, Ontario, M5G 1L7, Canada.

Vital signs and arterial blood gases also were measured preoperatively. Postoperatively, pain was evaluated using a horizontal linear analogue score (0 = no pain, 10 = severe pain).[20] Somnolence was graded by using a modification of a previously determined scale, which used a five-point rating system[21]: 1 = oriented and initiates conversation; 2 = responds to all forms of stimulation, is well oriented but feels sleepy and does not initiate conversation; 3 = responds to verbal command and painful stimulation but is disoriented and does not initiate conversation; 4 = responds to painful stimulation but not to verbal command; 5 = unresponsive to verbal command or painful (pinprick) stimulation.

Side effects such as pruritis, nausea, and vomiting were noted. The incidence of urinary retention could not be determined, because all patients were catheterized intraoperatively.

Mean arterial pressure (MAP), heart rate (HR), respiratory rate (RR), pulmonary function, arterial blood gases, pain, and somnolence were measured at 2 h, 8 h, and 24 h postoperatively. Twenty-two patients (12 in the epidural group and ten in the intravenous group) returned at 6 weeks postoperatively for pulmonary function tests only.

Preoperatively, an epidural catheter was placed in either the L_{2-3} or L_{3-4} interspace. Ten milliliters 2% carbonated lidocaine was injected to test placement of the epidural catheter. Analgesia levels ranged between T_{10} and T_4.

Thirty minutes later, each patient was given either intravenous saline and epidural morphine or intravenous morphine and epidural saline. The patients did not receive any other preoperative or intraoperative narcotics. Twenty-two patients did not receive any premedication, while eight patients were given 5 mg diazepam iv immediately before catheter insertion to allay anxiety. No other medications were given preoperatively. General anesthesia was induced with sodium thiopental, and either succinylcholine or pancuronium was administered to facilitate endotracheal intubation. All patients were intubated with either a Robertshaw® double-lumen tube or an endotracheal tube and bronchial blocker. All patients had radial artery catheters placed.

Anesthesia was maintained with nitrous oxide, oxygen, and enflurane. No intraoperative narcotics were given other than the single dose of study drug. After reversal of muscle relaxants, all patients were extubated and taken to the recovery room. Each time the patient complained of pain postoperatively, they were given both epidural and intravenous preparations according to their group, with a minimum interval of 30 min between treatments.

All patients were transferred from the recovery room to an intensive care unit (ICU) 2 hours postoperatively. The epidural and intravenous preparations were given for the first 24 h and then the epidural catheter was removed. After the epidural catheter was removed, the patients were given either intravenous or intramuscular morphine for analgesia. The patients were then kept in the ICU for at least another 24 h to allow them to watch for possible delayed onset of respiratory depression.

Statistical Analysis

All results are presented as the mean ± SEM. Unpaired *t* tests were used to show that preoperative values for both randomized groups were comparable. The change in each variable, at 2 h, 8 h, and 24 h and 6 weeks postoperatively was measured by subtracting the value for each patient from his own preoperative value. Unpaired *t* tests were used to compare the epidural and intravenous mean changes in the postoperative period. Analysis of variance (two-way) and Tukey's test were used to do paired comparisons for each variable between times (preoperatively, 2 h, 8 h, and 24 h postoperatively). This was done separately for each treatment group. Somnolence scores were compared using a chi-square. Paired *t* tests were used to compare the 6-week postoperative, and preoperative FVC, FEV_1, and PEFR in 22 of the 30 subjects. $P < 0.05$ was taken to indicate significant differences in all cases.

Results

The intravenous morphine group included 11 men and five women with an average age of 59.6 yr and an average weight of 72.9 kg. The epidural morphine group had 11 men and three women with an average age of 61.3 yr and an average weight of 73.8 kg. In the intravenous group there were 11 single lobectomies, one bi-lobectomy, three wedge resections, and one thoracotomy without lung resection, whereas the epidural group consisted of 12 single lobectomies and two wedge resections. All preoperative measurements in both randomized groups were comparable and within normal limits.

The first dose of epidural or intravenous morphine was given within 15 min of starting the anesthetic induction. Operating time ranged from 3 to 5 h for all procedures, and thus the time from the first dose of epidural or intravenous morphine given to each patient to arrival of the patient in the recovery room was 3–5 h. Of the 30 patients, 28 completed the study. Two patients requested removal from the study, one at 9 h and the other at 18 h, because of poor analgesia. Both subsequently were found to be in the intravenous morphine group. Only seven of 14 patients in the epidural group required analgesia within 30 min after reaching

TABLE 1. Vital Signs

Mean Arterial Pressure (mmHg)	Epidural ($\bar{X}_E \pm$ SEM)	Intravenous ($\bar{X}_I \pm$ SEM)	Epidural ($\bar{\Delta}_E \pm$ SEM)	Intravenous ($\bar{\Delta}_I \pm$ SEM)	
Preoperative	99 ± 3	96 ± 3			NS
2 h	94 ± 3	94 ± 4	−5 ± 4	−2 ± 3	NS
8 h	89 ± 2*	94 ± 3	−10 ± 4	−2 ± 3	NS
24 h	90 ± 3	94 ± 2	−9 ± 5	−2 ± 3	NS
Heart rate (min^{-1})					
Preoperative	78 ± 3	81 ± 2			NS
2 h	79 ± 4	83 ± 3	+1 ± 4	+2 ± 4	NS
8 h	86 ± 5	85 ± 2	+8 ± 4	+4 ± 3	NS
24 h	82 ± 5	91 ± 3*	+4 ± 4	+10 ± 4	NS
Respiratory rate (min^{-1})					
Preoperative	21 ± 1	20 ± 1			NS
2 h	19 ± 1	23 ± 1	−2 ± 1	+3 ± 1	$P < 0.04$
8 h	19 ± 1	21 ± 1	−2 ± 1	+1 ± 1	$P < 0.02$
24 h	19 ± 1	23 ± 1	−2 ± 1	+3 ± 1	$P < 0.01$

$\bar{X}_E \pm$ SEM = mean ± SEM for epidural group; $\bar{X}_I \pm$ SEM = mean ± SEM for intravenous group; $\bar{\Delta}_E \pm$ SEM = difference between preoperative and postoperative means for epidural group; $\bar{\Delta}_I \pm$ SEM = difference between preoperative and postoperative means for intravenous group; NS = not significant between groups; P = significant differences between groups.

* Significantly different from preoperative value within groups ($P < 0.05$).

the recovery room, whereas in the intravenous group 13 of 16 patients required analgesia within 30 min of arriving in the recovery room. The epidural morphine group required on average, four pain treatments in the 24-h period, whereas the intravenous morphine group required, on the average, seven pain treatments. The total dose of morphine given the epidural group (19.6 mg ± 5.7) was significantly less than the intravenous morphine group (35 mg ± 11.6) ($P < 0.001$).

MAP was decreased significantly at 8 h in the epidural group, but no significant changes occurred in the intravenous group. There were no significant differences in MAP between the groups at 2 h, 8 h or 24 h (table 1). The epidural group had no significant changes in HR in the postoperative period, whereas the intravenous group had significantly increased HR at 24 h postoperatively. Between-group analysis revealed no significant differences at 2 h, 8 h, or 24 h (table 1).

There were no significant within-group changes in RR in either the epidural or intravenous group in the postoperative period. However, the epidural group had a decrease in RR at 2 h, 8 h, and 24 h, whereas the intravenous group had an increase in RR at 2 h, 8 h, and 24 h. These between-group differences were significant at 2 h, 8 h, and 24 h (table 1).

Both the epidural and intravenous groups had significantly lower arterial pH at 2 h and 8 h after the procedure. There were no significant differences in

TABLE 2. Arterial Blood Gases

	Epidural ($\bar{X}_E \pm$ SEM)	Intravenous ($\bar{X}_I \pm$ SEM)	Epidural ($\bar{\Delta}_E \pm$ SEM)	Intravenous ($\bar{\Delta}_I \pm$ SEM)	
pH					
Preoperative	7.42 ± 0.01	7.42 ± 0.01			
2 h	7.35 ± 0.01*	7.34 ± 0.01*	−0.07 ± 0.01	−0.08 ± 0.01	NS
8 h	7.36 ± 0.01*	7.38 ± 0.01*	−0.06 ± 0.01	−0.04 ± 0.01	NS
24 h	7.40 ± 0.01	7.41 ± 0.01	−0.02 ± 0.01	−0.01 ± 0.01	NS
Pa_{O_2} (mmHg)					
Preoperative	79 ± 2	80 ± 3	+32 ± 8		
2 h	111 ± 6*	122 ± 9*	+32 ± 8	+42 ± 8	NS
8 h	106 ± 6	109 ± 6*	+27 ± 7	+29 ± 6	NS
24 h	91 ± 10	79 ± 4	+12 ± 11	−1 ± 5	NS
Pa_{CO_2} (mmHg)					
Preoperative	34 ± 1	33 ± 1			
2 h	42 ± 1*	40 ± 1*	+8 ± 2	+7 ± 2	NS
8 h	40 ± 2*	37 ± 1*	+6 ± 2	+4 ± 2	NS
24 h	37 ± 1	35 ± 1	+3 ± 2	+2 ± 1	NS

$\bar{X}_E \pm$ SEM = mean ± SEM for epidural group; $\bar{X}_I \pm$ SEM = mean ± SEM for intravenous group; $\bar{\Delta}_E \pm$ SEM = difference between preoperative and postoperative means for epidural group; $\bar{\Delta}_I \pm$ SEM = difference between preoperative and postoperative means for intravenous group; NS = not significant between groups.

* Significantly different from preoperative value within groups ($P < 0.05$).

TABLE 3. Pulmonary Function Tests

	Epidural ($\bar{X}_E \pm$ SEM)	Intravenous ($\bar{X}_I \pm$ SEM)	Epidural ($\bar{\Delta}_E \pm$ SEM)	Intravenous ($\bar{\Delta}_I \pm$ SEM)	
FVC (l)					
Preoperative	3.4 ± 0.3	3.6 ± 0.2			NS
2 h	1.6 ± 0.2*	1.1 ± 0.1*	−1.8 ± 0.2	−2.5 ± 0.2	$P < 0.03$
8 h	2.0 ± 0.2*	1.5 ± 0.1*	−1.4 ± 0.2	−2.1 ± 0.2	$P < 0.01$
24 h	2.2 ± 0.2*	1.6 ± 0.1*	−1.2 ± 0.2	−2.0 ± 0.2	$P < 0.02$
6 weeks	3.1 ± 0.3	3.1 ± 0.3	−0.3 ± 0.3	−0.5 ± 0.2	NS
FEV_1 ($l \cdot sec^{-1}$)					
Preoperative	2.4 ± 0.3	2.7 ± 0.2			NS
2 h	1.1 ± 0.1*	0.8 ± 0.1*	−1.3 ± 0.2	−1.9 ± 0.2	$P < 0.04$
8 h	1.4 ± 0.1*	1.0 ± 0.1*	−1.0 ± 0.2	−1.7 ± 0.2	$P < 0.01$
24 h	1.6 ± 0.2*	1.2 ± 0.1*	−0.8 ± 0.1	−1.5 ± 0.2	$P < 0.01$
6 weeks	2.1 ± 0.3	2.3 ± 0.2	−0.3 ± 0.1	−0.4 ± 0.2	NS
PEFR ($l \cdot min^{-1}$)					
Preoperative	404 ± 44	430 ± 29			NS
2 h	175 ± 23*	130 ± 11*	−229 ± 30	−300 ± 27	NS
8 h	230 ± 30*	188 ± 13*	−174 ± 30	−242 ± 22	NS
24 h	270 ± 30*	192 ± 15*	−134 ± 29	−238 ± 30	$P < 0.03$
6 weeks	358 ± 44	402 ± 30	−46 ± 27	−28 ± 22	NS

$\bar{X}_E \pm$ SEM = mean ± SEM for epidural group; $\bar{X}_I \pm$ SEM = mean ± SEM for intravenous group; $\bar{\Delta}_E \pm$ SEM = difference between preoperative and postoperative means for epidural group; $\bar{\Delta}_I \pm$ SEM = difference between preoperative and postoperative means for intravenous group; NS = not significant between groups; P = significant differences between groups.

* Significantly different from preoperative value within groups ($P < 0.05$).

arterial *p*H between the groups in the postoperative period (table 2). Pa_{O_2} was significantly increased at 2 h in the epidural group and at 2 h and 8 h in the intravenous group. There were no significant Pa_{O_2} differences between the two groups at 2 h, 8 h, and 24 h (table 2). However, Pa_{O_2} values cannot be compared meaningfully as the patients were on variable inspired oxygen concentrations at the time the arterial blood samples were drawn. Pa_{CO_2} was significantly elevated at 2 h and 8 h within both groups. However, no significant differences occurred between the two groups at 2 h, 8 h or 24 h (table 2).

FVC, FEV_1, and PEFR were not significantly different at 6 weeks postoperatively in 22 of the 30 patients when compared with their preoperative values (table 3). Eight patients did not complete the six-week follow-up. Because of this evidence of return to preoperative pulmonary function within 6 weeks of surgery, all the 2 h, 3 h, and 24 h measurements of FVC, FEV_1, and PEFR were compared with the preoperative values. Within the epidural group and the intravenous group, FVC was decreased significantly at 2 h, 8 h, and 24 h. However, the decrease in FVC was significantly less in the epidural group than in the intravenous group at 2 h, 8 h, and 24 h (table 3). Similarly FEV_1 was significantly decreased within both groups at 2 h, 8 h, and 24 h. However, the epidural group again had significantly less decrease in FEV_1 at 2 h, 8 h, and 24 h (table 3). PEFR also showed significant decreases in both groups at 2 h, 8 h, and 24 h postoperatively. The epidural group had significantly smaller decrease in PEFR at 24 h.

TABLE 4. Linear Analogue Pain Score

Pain (cm)	Epidural ($\bar{X}_E \pm$ SEM)	Intravenous ($\bar{X}_I \pm$ SEM)	
2 h	4.0 ± .58	6.4 ± .65	$P < 0.01$
8 h	2.6 ± .37*	4.8 ± .59	$P < 0.004$
24 h	3.1 ± .58	4.9 ± .69	NS

$\bar{X}_E \pm$ SEM = mean ± SEM for epidural group; $\bar{X}_I$ SEM = mean ± SEM for intravenous group; NS = not significant between groups; P = significant differences between groups.

* Significantly different from preoperative value within groups ($P < 0.05$).

The epidural group had significantly better pain relief at 2 h and 8 h as determined by the linear analogue pain score (table 4). There was no significant differences between pain in the two groups at 24 h, although the P value was close to the significance limit ($P < 0.06$). In addition, the epidural group demonstrated maximum pain relief 8 h postoperatively, the 8 h measurement being significantly better than the 2 h value but no different from the 24-h value (table 4). The intravenous group had no significant differences in pain at either 2 h, 8 h, or 24 h.

There were no significant differences at 2 h, 8 h, or 24 h in somnolence scores between the two groups (table 5). One patient had a somnolence score of 4 at 2 h, and another patient had a score of 3 at 8 h. Both patients were given 1 mg of physostigmine, which totally reversed their drowsiness. Neither patient had a Pa_{CO_2} greater

than 50 mmHg or a respiratory rate less than 12 min^{-1} at the time. Both patients later were found to be receiving epidural morphine. Neither patient had received valium or any other drug as a premedication.

The length of hospital stay was not significantly different, being 9.9 days for the epidural group and 9.6 days for the intravenous group.

Four patients had pruritis in the epidural group, while no patients had pruritis in the intravenous group ($P < 0.05$). All patients with pruritis responded to 25–50 mg diphenhydramine, and naloxone was not required. Three patients in the epidural group and two patients in the intravenous group had nausea and vomiting (NS). One patient in the intravenous group died 48 h postoperatively. Autopsy later showed a severe narrowing of the left anterior descending coronary artery and multiple small pulmonary emboli.

TABLE 5. Somnolence Score

	Epidural ($\bar{X}_E \pm$ SEM)	Intravenous ($\bar{X}_I \pm$ SEM)	
2 h	1.4 ± 0.51	1.6 ± 0.85	NS
8 h	1.4 ± 0.50	1.3 ± 0.85	NS
24 h	1.0 ± 0	1.0 ± 0	NS

$\bar{X}_E \pm$ SEM = mean ± SEM for epidural group; $\bar{X}_I \pm$ SEM = mean ± SEM for intravenous group; NS = not significant between groups.

Discussion

Several different methods have been utilized in an attempt to reduce pain and improve pulmonary mechanics postthoracotomy. These include epidural blocks using local anesthetics[22,23] and intercostal blocks.[24,25] Both have been shown to provide excellent relief with relative preservation of lung volumes in the postoperative period, and both have disadvantages. Intercostal blocks are time consuming, uncomfortable for the patient, and may cause a pneumothorax.[26,27] Epidural local anesthetics may cause hypotension and motor blockade of lower extremities.

It has been reported that continuous thoracic epidural fentanyl and lumbar epidural morphine when compared with parenteral narcotics lessen the impairment of pulmonary function during the first 24 h postoperatively.[14,17] Another study at the 24 h postoperative time showed no significant difference between parenteral and thoracic epidural morphine in the control of pain or postoperative pulmonary function in patients who underwent upper-abdominal surgical procedures.[18] There have been no reported double-blind controlled studies comparing the effects of lumbar or thoracic epidural morphine with parenteral morphine on postoperative pain and pulmonary mechanics in patients undergoing thoracotomy.

Our study design followed the normal ward routine, in which patients are given postoperative analgesics on demand for pain relief. Under these circumstances, epidural morphine produced significantly better pain relief than intravenous morphine at 2 and 8 h postoperatively. In addition, although lung volumes and expiratory flow rates were decreased markedly, the epidural morphine group had significantly less decrease than the intravenous group for the first 24 h postoperative period. We were able to use preoperative pulmonary function as a baseline in spite of lung resection in most of the patients, because pulmonary function had returned to normal by six weeks postoperatively. This compensatory hyperinflation after lobectomy has been documented previously.[28]

Small but significant differences in respiratory rate between the two groups were present, the epidural group having a slower postoperative rate and the intravenous group having a faster postoperative rate. Both groups showed significant increases in Pa_{CO_2} at 2 h and 8 h, with corresponding decreases in arterial pH. The increases in Pa_{CO_2}, however, were within the normal physiologic range, and there were no significant differences between the two groups.

The potential problem of respiratory depression in patients receiving epidural narcotics has been a recurring one.[29–34] In our study, we gave no narcotics preoperatively or intraoperatively other than the initial single dose of either parenteral or epidural morphine following induction. Using this regimen, we had no incidence of marked respiratory depression. One of the possible causes for the late onset of respiratory depression may be the use of combinations of epidural and parenteral narcotics. In the nationwide Swedish survey of more than 6,000 patients, only three patients who received epidural narcotics and no other parenteral narcotics had any sign of respiratory depression.[35] Even without profound respiratory depression, patients in the postoperative period have a variable ventilatory response to carbon dioxide if they have received only epidural narcotics.[36,37] For this reason and also because some patients may receive small supplemental doses of parenteral narcotics, we feel that all patients receiving epidural narcotics should be monitored in an intensive care setting or with an apnea monitor.

Somnolence after the use of narcotics previously has been reported in association with respiratory depression.[38] The two patients in our study who had somnolence scores greater than two showed no evidence of a decreased respiratory rate or increased Pa_{CO_2}. Both were reversed completely with physostigmine. Physostig-

mine was used instead of naloxone because we only wanted to decrease somnolence. Naloxone has been reported to diminish analgesia when given to patients receiving epidural narcotics.[12]

We used a large volume of solution (20 ml) epidurally to obtain a higher segmental level. In a previous study, it was demonstrated that the initial segmental distribution of morphine hypalgesia corresponds closely to the spread of analgesia provided by the local anesthetic dose used to test the correct placement of the epidural catheter.[39] We felt the larger volume would ensure a level up to the upper thoracic dermatones in all patients. At present, the optimal volume has not been determined.

A previous study in thoracotomy patients showed that analgesia with lumbar epidural morphine using 20 ml of morphine solution was adequate when the dose of morphine varied from 2 to 6 mg.[19] The increased dosage only provided a longer duration of analgesia. It appears that the dose of 5 mg of morphine is a good choice initially in these patients.

The mechanism causing the restrictive ventilatory defect after upper abdominal and thoracic surgery is not understood completely.[40] It has been shown that pain relief with the use of epidural local anesthetics[41,42] or intercostal blocks[24,25] will not restore vital capacity to preoperative levels. It is hypothesized that pain and surgical trauma lead to a decreased postoperative vital capacity.[40] Measures that decrease pain will increase vital capacity and improve the patient's ability to cough and take deep breaths. Deep breathing maneuvers prevent atelectasis and the resulting hypoxemia by helping to expand airways that have collapsed postoperatively. In addition, decreased pain aids early mobilization, with resultant decrease in deep vein thrombosis.

This study has shown that epidural morphine administered in the lumbar space offers an effective method of providing analgesia and improving respiratory function in the postoperative period in thoracotomy patients.

References

1. Loon WB, Morrison JD: The incidence and severity of postoperative pain. Br J Anaesth 39:695–698, 1967
2. Johnson WC: Postoperative ventilatory performance. Dependence upon surgical incision. Am Surg 41:615–619, 1975
3. Ali J, Weisel RD, Layug AB, Kripke BJ, Hechtman HB: Consequences of postoperative alterations in respiratory mechanics. Am J Surg 128:376–382, 1974
4. Behar M, Magora F, Olshwang D, Davidson JT: Epidural morphine in treatment of pain. Lancet i:527–528, 1979
5. Chayen MS, Rudick V, Borvine A: Pain control with epidural injection of morphine. ANESTHESIOLOGY 53:338–339, 1980
6. Cousins MJ, Mather LE, Glynn CJ, Wilson PR, Graham JR: Selective spinal analgesia. Lancet i:1141–1142, 1979
7. Torda TA, Pybus DA: Clinical experience with epidural morphine. Anaesth Intensive Care: 9:129–134, 1981
8. Chambers WA, Sinclair CJ, Scott DB: Extradural morphine for pain after surgery. Br J Anaesth 53:921–925, 1981
9. Anderson I, Thompson WR, Varkey GP, Knill RL: Lumbar epidural morphine as an effective analgesic following cholecystectomy. Can Anaesth Soc J 28:523–528, 1981
10. Lanz E, Theiss D, Reiss W, Sommer U: Epidural morphine for postoperative analgesia: A double blind study. Anaesth Analg 61:236–240, 1982
11. Carmichael FJ, Rolbin SH, Hew E: Epidural morphine for analgesia after caesarean section. Can Anaesth Soc J 29:359–363, 1982
12. Cohen SE, Woods WA: The role of epidural morphine in the postcaesarean patient. Efficacy and effects on bonding. ANESTHESIOLOGY 58:500–504, 1983
13. Jacobson L, Phillips PD, Hull CJ, Conacher ID: Extradural versus intramuscular diamorphine. Anaesthesia 38:10–18, 1983
14. Bromage PR, Camporesi E, Chestnut D: Epidural narcotics for postoperative analgesia. Anesth Analg 59:473–480, 1980
15. Rawal N, Sjöstrand UH, Dahlström B, Nydahl PA, Ostelius J: Epidural morphine for postoperative pain relief: A comparative study with intramuscular narcotic and intercostal nerve block. Anesth Analg 61:93–98, 1982
16. Holland AJC, Srikantha SK, Tracey JA: Epidural morphine and postoperative pain relief. Can Anaesth Soc J 28:453–458, 1981
17. Welchew EA, Thorton JA: Continuous thoracic epidural fentanyl. Anaesthesia 37:309–316, 1982
18. Klinck JR, Lindap JM: Epidural morphine in the elderly. Anaesthesia 37:907–912, 1982
19. Nordberg G, Hedner T, Mellstrand T, Dahlström B: Pharmacokinetic aspects of epidural morphine analgesia. ANESTHESIOLOGY 58:545–551, 1983
20. Revill SI, Robinson JO, Rosen M, Hogg MIJ: The reliability of a linear analogue for evaluating pain. Anaesthesia 31:1191–1198, 1976
21. Bidwai AV, Cornelius LR, Stanley TH: Reversal of Innovar®-induced postanesthetic somnolence and disorientation with physostigmine. ANESTHESIOLOGY 44:249–252, 1976
22. Shuman RL, Peters RM: Epidural anesthesia following thoracotomy in patients with chronic obstructive airway disease. J Thorac Cardiovasc Surg 71:82–88, 1976
23. James EC, Kolberg BS, Iwen GW, Gellatly TA: Epidural analgesia for post-thoracotomy patients. J Thorac Cardiovasc Surg 82:898–903, 1981
24. Fust RF, Nauss LA: Post-thoracotomy intercostal block: Comparison of its effects on pulmonary function with those of intramuscular meperidine. Anesth Analg 55:542–546, 1976
25. Toledo-Pereyra LH, Demeester TR: Postoperative randomized evaluation of intrathoracic intercostal nerve block with bupivacaine on post operative ventilatory function. Ann Thorac Surg 27:203–205, 1979
26. Engberg G: Single dose intercostal nerve blocks with etidocaine for pain relief after upper abdominal surgery. Acta Anaesthesiol Scand 60(Suppl):43–49, 1975
27. Moore DC: Intercostal nerve block for postoperative somatic pain following surgery of thorax and upper abdomen. Br J Anaesth 47:284–288, 1975
28. Berend N, Woollock AJ, Marlin GE: Effects of lobectomy on lung function. Thorax 35:145–150, 1980
29. Weddel SJ, Ritter RR: Serum levels following epidural administration of morphine and correlation with relief of postsurgical pain. ANESTHESIOLOGY 54:210–214, 1981

30. Boas RA: Hazards of epidural morphine. Anaesth Intensive Care 8:377–378, 1980
31. Christiansen V: Respiratory depression after extradural morphine. Br J Anaesth 52:841, 1980
32. McDonald AM: Complications of epidural morphine. Anaesth Intensive Care 8:490–491, 1980
33. Reiz S, Westberg M: Side-effects of epidural morphine. Lancet ii: 203–204, 1980
34. Yaksh TL: Spinal opiate analgesia: Characteristics and principles of action. Pain 11:293–346, 1981
35. Gustafsson LL, Schildt B, Jacobsen K: Adverse effects of extradural and intrathecal opiates: Report of a nationwide study. Br J Anaesth 54:479–485, 1982
36. Doblar DD, Muldoon SM, Albrecht PH, Baskoff J, Watson RL: Epidural morphine following epidural local anesthetic: Effect on ventilatory and airway occlusion pressure responses to CO_2. ANESTHESIOLOGY 55:423–428, 1981
37. Kafer ER, Brown RJ, Scott D, Findlay JWA, Butz RF, Teeple E: Biphasic depression of ventilatory responses to CO_2 following epidural morphine. ANESTHESIOLOGY 58:418–427, 1983
38. Paulus DA, Paul WL, Munson ES: Neurologic depression after intrathecal morphine. ANESTHESIOLOGY 54:517–518, 1981
39. Bromage PR, Camporesi EM, Durante PAC, Nielsen CH: Rostral spread of epidural morphine. ANESTHESIOLOGY 56:431–436, 1982
40. Craig DB: Postoperative recovery of pulmonary function. Anesth Analg 60:46–52, 1981
41. Wahba WM, Craig DB, Don HF, Becklake MR: The cardiorespiratory effects of thoracic epidural anesthesia. Can Anaesth Soc J 19:8–19, 1972
42. Spence AA, Smith G: Postoperative analgesia and lung function: A comparison of morphine with extradural block. Br J Anaesth 43:144–148, 1971