Anesthesiology
83:96–100, 1995

Local Anesthetic Test Dose as a Predictor of Effective Epidural Opioid Analgesia

Sandra R. Weitz, M.D., Kenneth Drasner, M.D.†*

Background: **When local anesthetic is used to produce epidural anesthesia intraoperatively, epidural catheter placement is confirmed. However, when epidural catheters are placed intraoperatively only to provide postoperative opioid analgesia, correct catheter placement may not be confirmed by administration of a local anesthetic. The current study tests the hypothesis that the extent of sensory blockade produced by a 10-ml dose of 1.5% lidocaine can be used to predict the adequacy of epidural opioid analgesia.**

Methods: **Forty-nine patients undergoing major abdominal surgery in whom a lumbar epidural catheter was placed intraoperatively were studied, but no more than 3 ml 1% lidocaine had been injected. Placement of the epidural catheter was assessed in the postanesthesia care unit by administration of a 10-ml dose of 1.5% lidocaine. The extent of sensory blockade was determined using the pinprick technique: All dermatomes, T2 and below, were assessed and scored using 1 point per dermatome per side from L1 to T2 to a maximum of 24 points. Scores were arbitrarily divided into three groups, where group 1, 0–7 points; group 2, 8–15 points; and group 3, 16–24 points. Epidural morphine infusion was initiated independently of the extent of the sensory blockade and adjusted using predetermined guidelines. Adequacy of opioid-induced analgesia was determined using the visual analog scale.**

Results: **Significantly lower visual analog scale scores for pain at rest and with movement from epidural morphine infusion were associated with sensory blockade score of 16–24 points. Seven patients failed to obtain a detectable sensory block. No patient requested alternative analgesia. None of the epidural catheters was removed because of inadequate pain relief, even in patients who failed to obtain a detectable sensory block.**

Conclusions: **Extensive sensory block from 10 ml 1.5% lidocaine was associated with excellent epidural opioid analgesia. Extent of analgesia after a 10-ml test dose of 1.5% lidocaine can be used to predict the adequacy of analgesia resulting from an epidural opioid infusion. The failure of a local anesthetic dose to produce sensory blockade does not necessarily predict a failure to produce analgesia from an epidural opioid infusion, as indicated by the presence of analgesia in several patients without detectable sensory block. (Key words: Analgesia: epidural; opioid. Anesthetic techniques, epidural: continuous infusion. Anesthetics, local: lidocaine. Opioids: morphine. Regional anesthesia.)**

EPIDURAL administration of local anesthetic produces anesthesia primarily by direct action on the nerve roots,[1] whereas epidural opioids bind to receptors in the substantia gelatinosa of the dorsal horn of the spinal cord.[2] The current experiment tests the hypothesis that, despite different sites and mechanisms of action of these two classes of drugs, the extent of sensory blockade produced by a local anesthetic test dose can be used to predict the adequacy of epidural opioid analgesia.

Methods

With approval from our Institutional Committee on Human Research, we conducted a prospective, observational study of 49 patients recovering from major abdominal surgery in whom epidural catheters were placed solely to provide postoperative analgesia. Lumbar epidural catheters (Braun Perifix epidural catheter kits containing an 18-G Tuohy needle and a 20-G epidural catheter) were placed preoperatively at either the L2–L3 or the L3–L4 interspace, and a 3-ml test dose of 1.5% lidocaine (with 1:100,000 epinephrine) was administered to exclude unintentional intravenous or intrathecal injection of local anesthetic. Intraoperative anesthetic management proceeded at the discretion of the attending anesthesiologist. As is routine practice at our institution, approximately 2 h before the end of surgery, a bolus of 2.5 mg morphine was administered through the epidural catheter. Patients receiving local anesthetic intraoperatively were excluded from study.

* Assistant Professor.

† Associate Professor.

Received from the Department of Anesthesia, University of California, San Francisco, San Francisco, California. Submitted for publication May 2, 1994. Accepted for publication April 4, 1995. Presented in part at the annual meetings of the American Society of Regional Anesthesia, Seattle, Washington, 1993, and the American Society of Anesthesiologists, Washington, D.C., 1993.

Address reprint requests to Dr. Weitz: Department of Anesthesia, University of California, San Francisco, 521 Parnassus Avenue, San Francisco, California 94143-0648. Address electronic mail to: sandyweitz@vaxine.ucsf.edu.

Study Protocol

Baseline blood pressure, heart
(visual analog scale, VAS) were
anesthesia care unit. When patien
ficiently to respond to command
1.5% lidocaine with epinephrine
there was no evidence of intrath
injection after 3 min, an additiona
without epinephrine was given.
the second injection, we assessed
sory blockade by pinprick and obt
for pain at rest and pain with mo
The extent of sensory blockade
point per dermatome per side fro
imum of 24 points. All lumbosac
matomes to T2 were assessed by
(S.W.) using the pinprick technic
skin of each dermatome with a
extending only to L2 or below we
Before the study began, a decisio
the points indicating extent of blo
three groups, where group , tho
score was 0–7 points; group 2, 8–
3, 16–24 points.

Independently of the extent of
epidural infusion of morphine (
of 0.010 $mg \cdot kg^{-1} \cdot h^{-1}$ was initia
Acute Pain Service attending ane
to the extent of sensory blockad
score data and managed the epidu
rates. VAS scores were obtained
morning rounds. Patients were ask
at rest and greatest pain with m
period. Pain was scored using
ranging from no pain to worst pa
The epidural infusion rate was a
were based on both VAS score for
effects to allow patients to distinguish
side effects predominated, *e.g.*,
creased in patients who indicat
(pruritus, nausea, vomiting) wer
than pain. Specifically, infusion
or decreased by 0.003 $mg \cdot kg^{-1}$.
lowing guidelines: (1) if VAS = 0
decreased; (2) if VAS = 1–3 and
than side effects, infusion rate wa
VAS = 1–3 and side effects seem
infusion rate was decreased; (4)
seemed worse than side effects,
creased; and (5) if VAS > 3 and

Study Protocol

Baseline blood pressure, heart rate, and pain scores (visual analog scale, VAS) were obtained in the postanesthesia care unit. When patients had recovered sufficiently to respond to commands, a 3-ml test dose of 1.5% lidocaine with epinephrine was administered. If there was no evidence of intrathecal or intravascular injection after 3 min, an additional 7 ml 1.5% lidocaine without epinephrine was given. Twenty minutes after the second injection, we assessed the level of the sensory blockade by pinprick and obtained VAS pain scores for pain at rest and pain with movement.

The extent of sensory blockade was scored using 1 point per dermatome per side from L1 to T2 for a maximum of 24 points. All lumbosacral and thoracic dermatomes to T2 were assessed by a single investigator (S.W.) using the pinprick technique, *i.e.,* touching the skin of each dermatome with a 19-G needle. Blocks extending only to L2 or below were assigned 0 points. Before the study began, a decision was made to assign the points indicating extent of blockade arbitrarily into three groups, where group 1, those in whom blockade score was 0–7 points; group 2, 8–15 points; and group 3, 16–24 points.

Independently of the extent of sensory blockade, an epidural infusion of morphine (0.1 mg/ml) at a rate of 0.010 $mg \cdot kg^{-1} \cdot h^{-1}$ was initiated.

Acute Pain Service attending anesthesiologists blinded to the extent of sensory blockade collected all pain score data and managed the epidural morphine infusion rates. VAS scores were obtained once daily during morning rounds. Patients were asked to score least pain at rest and greatest pain with movement over a 24-h period. Pain was scored using the 10-cm VAS scale ranging from no pain to worst pain imaginable.

The epidural infusion rate was adjusted daily. Criteria were based on both VAS score for pain at rest and side effects to allow patients to distinguish whether pain or side effects predominated, *e.g.,* infusion rate was increased in patients who indicated that side effects (pruritus, nausea, vomiting) were more troublesome than pain. Specifically, infusion rates were increased or decreased by 0.003 $mg \cdot kg^{-1} \cdot h^{-1}$ based on the following guidelines: (1) if VAS = 0–1, infusion rate was decreased; (2) if VAS = 1–3 and pain seemed worse than side effects, infusion rate was maintained; (3) if VAS = 1–3 and side effects seemed worse than pain, infusion rate was decreased; (4) if VAS > 3 and pain seemed worse than side effects, infusion rate was increased; and (5) if VAS > 3 and side effects seemed worse than pain, infusion rate was maintained. In addition, patients reporting pain (VAS > 3 and/or requested additional pain medication) received 2-mg intravenous morphine. More than one report of pain during a 12-h nursing shift was treated by an increase in the epidural infusion rate. Increases in the epidural infusion rate were permitted as frequently as every 4 h. Study was concluded on postoperative day 3.

Patients whose epidural infusion was discontinued before the conclusion of the study were included in the statistical analysis.

Statistical Analysis

The Kruskal-Wallis test was used for the analysis of pain scores, epidural infusion rates, and supplemental morphine requirements. $P < 0.05$ was considered significant.

Results

Forty-nine patients were studied (27 men and 22 women), ranging in age from 34 to 72 yr. Injection of the 3-ml test dose of 1.5% lidocaine with epinephrine suggested neither intravascular nor intrathecal catheter placement in any of the patients. Seven patients had no demonstrable sensory block of any dermatome, including those below L1, and thus a score of 0. Fourteen patients had a sensory blockade score of 0–7 points (group 1), and 14 patients, a score of 8–15 points (group 2). Twenty-one patients achieved a blockade score of 16–24 points (group 3). Groups were similarly distributed with regard to type of surgery (table 1).

Epidural infusion was discontinued in ten patients before postoperative day 3 in response to the surgeon's

Table 1. Distribution of Surgical Procedures

Type of Surgery	Group 1	Group 2	Group 3	Total
Colectomy	3	3	3	9
Radical prostatectomy	1	2	3	6
Radical cystectomy	2	1	0	3
Hepatic lobectomy	3	2	2	8
Radical hysterectomy	0	1	3	4
Whipple procedure	1	1	1	3
Cholecystectomy	1	0	1	3
Bowel resection	2	2	4	8
Abdominal aortic aneurysm	0	1	1	2
Aortofemoral bypass graft	0	0	1	1
Gastrectomy	1	1	0	2

request or postoperative catheter displacement. On postoperative day 1, the catheter was removed in one patient in group 1 and two patients in group 3; on postoperative day 2, one patient in each of groups 1 and 2, and five patients in group 3. None was removed due to inadequate pain relief.

VAS pain scores for pain at rest and with movement were significantly less for patients in group 3 (figs. 1 and 2). The epidural morphine infusion rates for the three groups on all three postoperative days are seen in figure 3. Supplemental intravenous morphine requirements did not differ significantly among the three groups (fig. 4). Pain scores did not differ significantly according to the day of catheter removal.

Discussion

Our data indicate that, despite different sites and mechanisms of action, the extent of sensory blockade obtained from a 10-ml test dose of 1.5% lidocaine may predict the adequacy of analgesia resulting from an epidural opioid infusion. Extensive sensory blockade was associated with excellent epidural opioid analgesia (fig. 1). This finding confirms the clinical impression that patients experiencing sensory blockade after local anesthetic administration have a correctly placed epidural catheter, which also provides excellent analgesia after opioid administration *via* these catheters.

We also found that patients without detectable sensory block achieved some degree of opioid-induced analgesia. Despite relatively high VAS scores, none of these patients requested alternative forms of pain medication. The presence of analgesia with epidural morphine despite absence of a detectable sensory block with local anesthetic might reflect inaccuracy of sensory assessment in the immediate postoperative period, *i.e.,* some patients may not have recovered sufficiently to permit meaningful determination of sensory blockade. This could result in an underestimate of the extent of blockade, particularly in patients having a sensory block of a relatively restricted distribution. In addition, differences in the sensitivity of the two measures used to assess analgesia might have affected our results. That is, pinprick was used to determine the presence of a

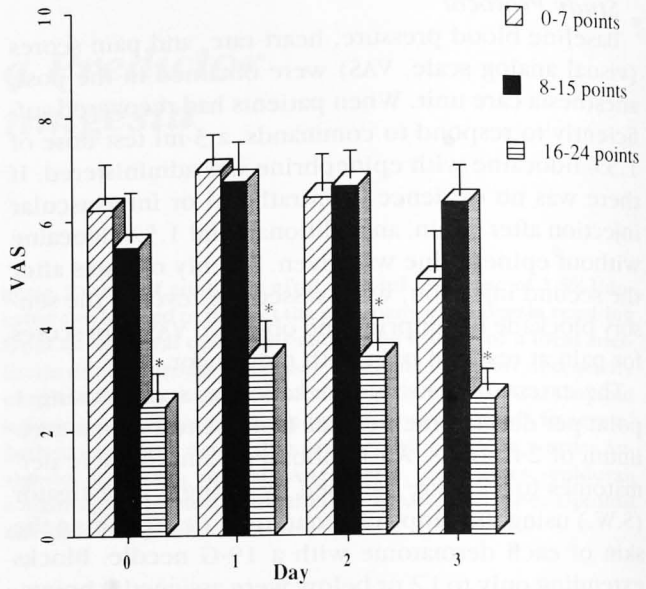

Fig. 2. Patient's self-report of greatest pain with movement as mean visual analog score (VAS) ± SEM. The extent of the sensory block was scored using 1 point per dermatome per side from L1 to T2 for a maximum of 24 points. Point ratings are arbitrarily divided into three groups: 0–7, 8–15, and 16–24 points. Postoperative day 0 represents the VAS ± SEM obtained 20 min after administration of 10 ml 1.5% lidocaine. Patients in the group with 16–24 had significantly less pain; *$P < 0.05$.

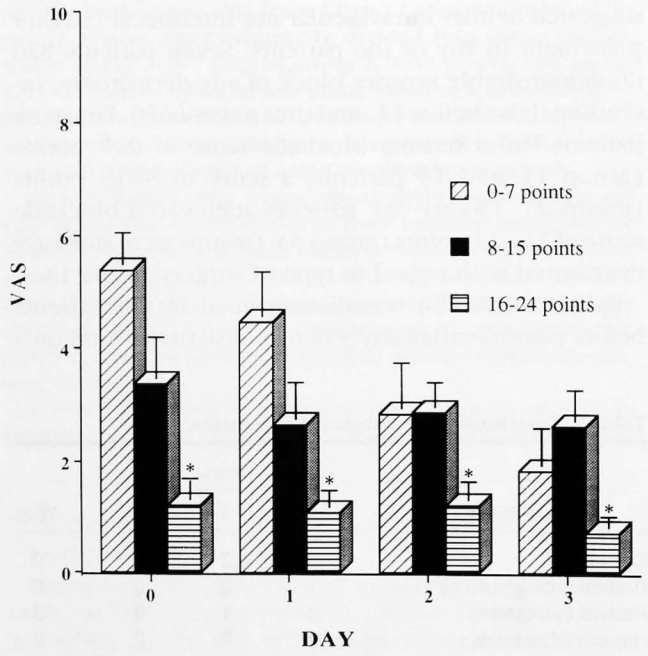

Fig. 1. Patient's self-report of least pain at rest as mean visual analog score (VAS) ± SEM. The extent of the sensory block was scored using 1 point per dermatome per side from L1 to T2 for a maximum of 24 points. Point ratings are arbitrarily divided into three groups: 0–7, 8–15, and 16–24 points. Postoperative day 0 represents the VAS ± SEM obtained 20 min after administration of 10 ml 1.5% lidocaine. Patients in the group with 16–24 had significantly less pain; *$P < 0.05$.

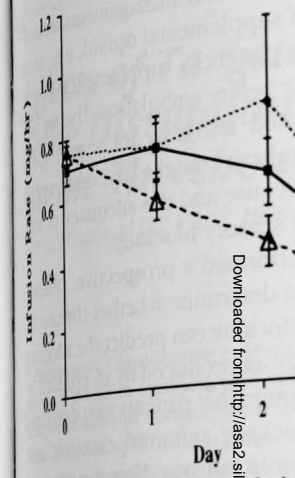

Fig. 3. Mean epidural morphine infusio
of the sensory block was scored using
per side from L1 to T2 for a maxim
ratings are arbitrarily divided into t
and 16–24 points. Postoperative day
SEM obtained 20 min after administ
docaine.

sensory block and self-assessment
ity of postoperative analgesia. P
sensitive to the quality of their p
detect subtle differences in analge
ferences in pinprick. Thus, the di
sensitivity of these "instruments"
to greater detection of pain and
Another possible explanation is
from systemic absorption of
anecdotal experience from patie
dural catheters are not in the ep
that it is unlikely that adequate an
either systemic absorption or a
over, preliminary data from a st
patient-controlled analgesia after
inal surgery suggest that the dose
required to achieve the pain sc
current study may be more than
tered—patients required, on ave
venous morphine over a 24-h p
day 1) to obtain a mean VAS sco
2.5.[3] Thus, although systemic abs
administered morphine might co
it is unlikely a systemic effect al
analgesia obtained. Although in
group receiving epidural saline an
would have provided additional
observational study of our standar

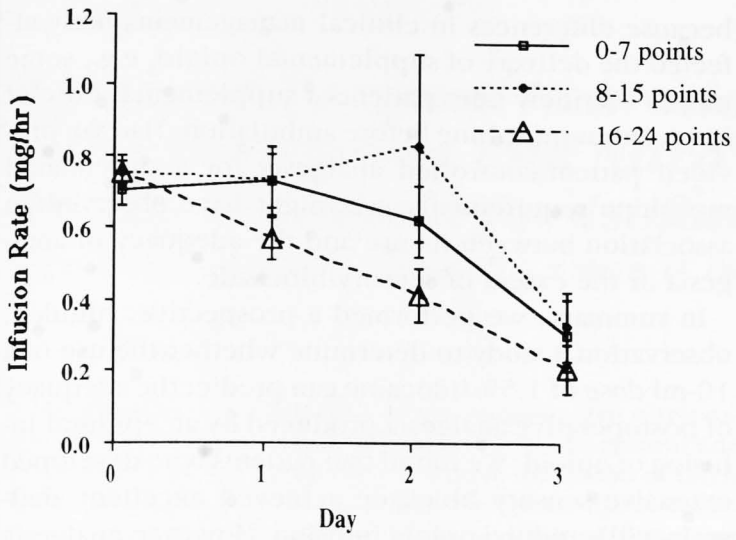

Fig. 3. Mean epidural morphine infusion rate ± SEM. The extent of the sensory block was scored using 1 point per dermatome per side from L1 to T2 for a maximum of 24 points. Point ratings are arbitrarily divided into three groups: 0–7, 8–15, and 16–24 points. Postoperative day 0 represents the VAS ± SEM obtained 20 min after administration of 10 ml 1.5% lidocaine.

sensory block and self-assessment to evaluate the quality of postoperative analgesia. Patients may be more sensitive to the quality of their pain and better able to detect subtle differences in analgesia than to detect differences in pinprick. Thus, the difference between the sensitivity of these "instruments" may have contributed to greater detection of pain and analgesia.

Another possible explanation is that analgesia resulted from systemic absorption of morphine. However, anecdotal experience from patients in whom the epidural catheters are not in the epidural space suggests that it is unlikely that adequate analgesia resulted from either systemic absorption or a placebo effect. Moreover, preliminary data from a study of patients using patient-controlled analgesia after similar major abdominal surgery suggest that the dose of systemic morphine required to achieve the pain scores obtained in the current study may be more than twice that administered—patients required, on average, 52.4 mg intravenous morphine over a 24-h period (postoperative day 1) to obtain a mean VAS score for pain at rest of 2.5.[3] Thus, although systemic absorption of epidurally administered morphine might contribute to analgesia, it is unlikely a systemic effect alone accounts for the analgesia obtained. Although inclusion of a control group receiving epidural saline and systemic morphine would have provided additional insight, this was an observational study of our standard practice of providing epidural analgesia, and we did not believe it justifiable to place epidural catheters in patients who would receive only a saline infusion.

Differences in the site of action of the administered drug also could contribute to a simultaneous failure to achieve detectable sensory blockade with local anesthetic and successful provision of analgesia with morphine. Local anesthetic sites of action include spinal nerves in the paravertebral space, dorsal root ganglia immediately adjacent to the dural cuff region, individual anterior and posterior spinal nerve roots within their dural root sleeves, spinal nerve rootlets, and peripheral regions of the spinal cord. After epidural injection of local anesthetic, diffusion of local anesthetic into intradural spinal nerve roots plays a major role in the early development of sensory blockade. Subsequent seepage of local anesthetic through the intervertebral foramina contributes to producing multiple paravertebral blocks. Among the local anesthetic sites of action, the spinal nerve roots, dorsal root ganglia, and spinal cord play a significant role,[4,5] as indicated by the segmental pattern of anesthesia that develops.[6,7] Spread within the epidural space is, therefore, a critical determinant of block.

In comparison, opioids injected into the epidural space produce analgesia principally by binding within the substantia gelatinosa in the dorsal horn of the spinal cord.[2] After diffusion of morphine across the spinal meninges,[8] significant spread occurs within the subarachnoid space. Thus, in comparison to the local an-

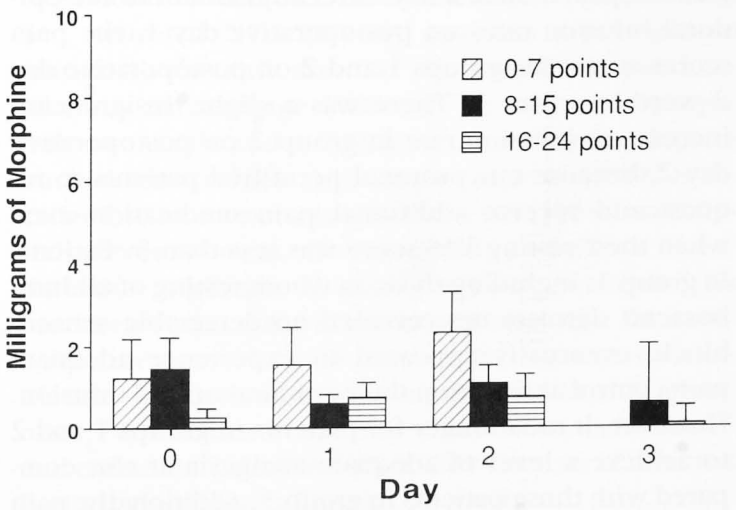

Fig. 4. Mean supplemental morphine doses ± SEM for postoperative days 0–3. The extent of the sensory block was scored using 1 point per dermatome per side from L1 to T2 for a maximum of 24 points. Point ratings were arbitrarily divided into three groups: 0–7, 8–15, and 16–24 points.

esthetics, initial spread within the epidural space is likely to be far less important.

Finally, it is possible that repetitive dosing of the epidural catheter may improve the spread of drug or penetration of the spinal meninges. This would explain the common clinical experience of failure to achieve sensory blockade with an initial dose, followed by success with subsequent doses. Possibly, analgesia may have been produced without detectable sensory blockade by our use of a continuous opioid infusion.

The VAS scores for postoperative day 0 reflect pain in the postanesthesia care unit before epidural infusion was initiated but after administration of the local anesthetic dose. Similarly, the infusion rate reported on postoperative day 0 is the initial epidural morphine infusion rate initiated after administration of the local anesthetic dose. Because patients in group 3 had a extensive sensory blockade from the 10-ml dose of 1.5% lidocaine when the VAS scores for postoperative day 0 were obtained, it is not surprising that patients in group 3 appeared to be significantly more comfortable from their opioid infusion on postoperative day 0 (figs. 1 and 2).

The infusion rate was checked and adjusted (if necessary) daily. On postoperative days 1 and 2, VAS scores reflected the adequacy of analgesia provided by infusion rates on postoperative days 0 and 1. On postoperative day 1, patients in groups 1 and 2 had significantly more pain than those in group 3; VAS scores at rest were greater than three in groups 1 and 2. However, the epidural infusion was started on postoperative day 0 and adjusted once a day. After adjustment of the epidural infusion rates on postoperative day 1, the pain scores at rest for groups 1 and 2 on postoperative day 2 were less than 3. There was a slight, insignificant increase in infusion rate in group 2 on postoperative day 2, because our protocol permitted patients to request and receive additional pain medication even when their resting VAS score was less than 3. Patients in group 1, including those in whom testing of all lumbosacral dermatomes revealed no detectable sensory block, eventually appeared to experience adequate pain control at rest from their epidural opioid infusion. However, it took longer for patients in groups 1 and 2 to achieve a level of adequate analgesia at rest compared with those patients in group 3. Additionally, pain with movement was significant greater in groups 1 and 2 on all 3 postoperative days.

The average intravenous morphine requirement for each study group cannot be considered representative because differences in clinical management likely affected the delivery of supplemental opioid, *e.g.*, some nurses routinely offer patients a supplemental dose of intravenous morphine before ambulation. Had we provided patient-controlled analgesia for supplemental morphine requirements, we might have observed an association between its use and the adequacy of analgesia or the extent of sensory blockade.

In summary, we performed a prospective, blinded, observational study to determine whether the use of a 10-ml dose of 1.5% lidocaine can predict the adequacy of postoperative analgesia produced by an epidural infusion of opioid. We found that patients who developed extensive sensory blockade achieved excellent analgesia with epidural opioid infusion. However, analgesia also was obtained in the absence of sensory blockade, likely reflecting insensitivity of our assessment of blockade. Regardless, our results suggest that failure to achieve sensory blockade does not necessarily predict failure of the epidural opioid technique to provide postoperative analgesia. In the absence of sensory blockade, patients may, at times, achieve satisfactory analgesia. The degree of analgesia in this group, however, remains significantly less than epidural administration of morphine is capable of providing. In cases in which optimal analgesia may effect outcome, the epidural catheter should be replaced in patients in whom extensive sensory blockade does not occur after local anesthetic administration.

References

1. Cousins MJ, Bromage PR. Epidural neural blockade, Neural Blockade. 2nd edition. Edited by Cousins MJ, Bridenbaugh PO. Philadelphia, JP Lippincott, 1988, pp 306–307

2. Cousins MJ, Mather LE: Intrathecal and epidural administration of opioids. ANESTHESIOLOGY 61:276–310, 1984

3. Weitz SR, Drasner K, Cohen NH, Jain U, Mangano DT: PCA vs. epidural morphine: Comparison of perioperative myocardial ischemia (abstract). Reg Anesth 18:4S, 1993

4. Cusick JF, Myklebust JB, Abrams SE: Differential neural effects of epidural anesthetics. ANESTHESIOLOGY 53:299–306, 1980

5. Cusick JF, Myklebust JB, Abrams SE, Davidson A: Altered neural conduction with epidural bupivacaine. ANESTHESIOLOGY 57:31–36, 1982

6. Bromage PR: Physiology and pharmacology of epidural analgesia. ANESTHESIOLOGY 28:592–622, 1967

7. Bromage PR: Mechanisms of action of extradural analgesia. Br J Anaesth 47S:199–211, 1975

8. Bernards CM, Hill HF: The spinal nerve root sleeve is not a preferred route for redistribution of drugs from the epidural space to the spinal cord. ANESTHESIOLOGY 75:827–832, 1991