Segmental Effect of Lumbar Epidural Hydromorphone: A Case Report

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Postoperative analgesia after esophagogastrectomy was achieved by administration of epidural narcotics. Incisional pain was relieved by hydromorphone (Dilaudid*), 1.5 mg in 10 ml saline, but this dose did not have any apparent effect on pain originating from the diaphragm. By increasing the volume of saline to 15 ml on subsequent epidural administrations, we were able to provide complete analgesia while still using the same total dose of narcotic.

REPORT OF A CASE

A 60-year-old, 86-kg man with biopsy-proven adenocarcinoma of the gastroesophageal junction underwent transthoracic esophagogastrectomy. The patient was premedicated with diazepam, 15 mg, po, 1 h prior to surgery. On arrival in the operating room, an iv and radial artery line were inserted. An epidural catheter was inserted into the L₃₋₄ interspace. Five minutes after administration of a 5-ml test dose of 1.5% lidocaine, 20 ml 1.5% lidocaine was injected through the epidural catheter. Ten minutes later the patient had a sensory level of T₄ bilaterally to pin prick. General anesthesia then was induced with thiopental, 500 mg iv, followed by succinylcholine, 100 mg iv to facilitate intubation of the trachea. A 39 F left-sided double-lumen endobronchial tube was inserted without difficulty. Anesthesia was maintained with isoflurane (0.5%) and O₂, and pancuronium bromide was used for muscle relaxation. General anesthesia was supplemented with intermittent doses of 0.5% bupivacaine administered via the epidural catheter approximately every 90 min during the operation. Surgery was performed through a transverse thoracoabdominal incision beginning at the T₄₋₅ interspace at the tip of the left scapula and extending across the abdomen. A wide diaphragmatic incision was made in order to provide exposure for esophagogastrectomy and splenectomy. The left lung selectively was collapsed for 100 min during surgery. The patient was hemodynamically stable throughout the procedure, which lasted 4 h.

Fifty minutes prior to the completion of the operation, hydromorphone, 1.5 mg in 10 ml normal saline was administered via the epidural catheter. At the completion of surgery, the trachea was extubated in the operating room.

On the first postoperative day, he received two additional doses of epidural hydromorphone (1.5 mg in 10 ml saline) at 6-h intervals. Following each epidural injection of hydromorphone, he denied pain in the area of his skin incision but complained of left shoulder pain in the scapular region. This pain partially was relieved with a total of 15 mg morphine, iv, given in incremental doses over 20 h. On the second postoperative day he was given hydromorphone, 1.5 mg in 15 ml saline via the epidural catheter, which relieved both his incisional and shoulder pain. This dose and volume of epidural narcotic was repeated on two other occasions that day. He required only 2 mg of morphine, iv, during the entire second postoperative day. On the third postoperative day, immediately prior to transfer from the intensive care unit, the epidural catheter was removed. He experienced incisional and shoulder pain for the next several days, and iv morphine provided only partial relief. He went home on the eleventh postoperative day.

DISCUSSION

The site of action of epidurally administered narcotics is believed to be at opiate receptors within the substantia gelatinosa of the spinal cord.¹ Postthoracotomy analgesia can be obtained from narcotics by direct absorption across the dura when given through an epidural catheter placed at a thoracic level or from rostral spread in the cerebrospinal fluid (CSF) when administered through an epidural at a lumbar level.²

It is unclear whether the analgesia from epidural narcotics is mainly segmental in nature³ or whether rostral spread within the CSF makes the actual level of injection unimportant. Narcotic analgesics administered into the lumbar epidural space successfully have relieved the pain of thoracotomy.⁴ Narcotics with low lipid solubility, such as morphine, demonstrate greater diffusion through the CSF and are effective for postthoracotomy pain when administered at either a lumbar or thoracic level.⁴ We used hydromorphone, a drug with greater lipid solubility than morphine. Therefore, the actual level of injection with apparent segmental effects we observed may be of greater significance when this agent is used than when less lipid-soluble narcotics are administered.

Although our patient's thoracoabdominal incisional pain was relieved on the first postoperative day with hydromorphone, 1.5 mg in 10 ml saline administered at the L₃₋₄ level, he still experienced referred diaphragmatic pain in his shoulder severe enough to require additional parenteral narcotics. On the second postoperative day, the patient still complained of thoracoabdominal and shoulder pain, which was relieved completely after a larger volume, but same total dose, of hydromorphone was given.

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Received from the Departments of Anesthesia and Surgery, Stanford University School of Medicine, Stanford, California 94305. Accepted for publication July 23, 1984.

Key words: Anesthetic technique: epidural narcotic.

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Surgery had included a wide dissection of the diaphragm. It is likely that the shoulder pain was from this and was not vagal in origin. The innervation of the central portion of the diaphragm is by the phrenic nerve, which contains nerve fibers from C₃₋₅. The periphery of the diaphragm is innervated by intercostal nerves from T₆₋₉ or T₁₀. When the volume of the narcotic solution administered at the lumbar level was increased from 10 ml to 15 ml, relief of diaphragmatic pain from nerves originating in the cervical spinal cord was achieved along with relief of pain originating from the thoracic and lumbar spinal cord levels. Presumably, by using a larger volume of solution, there was either increased spread of the hydromorphone in the CSF or in the epidural space itself. Since relief of the referred shoulder pain occurred very shortly after injection of the larger volume, the latter explanation appears more probable. It is unlikely that a cumulative effect from the narcotics occurred, since the patient experienced incisional pain as well as shoulder pain before each “top-up” dose of epidural hydromorphone was given.

Since respiratory depression can occur if epidurally administered narcotics reach the brainstem, we were hesitant to use larger volumes initially. Our patient was monitored in the intensive care while receiving epidural narcotics and did not have any evidence of respiratory depression with either 10 or 15 ml of narcotic solution.

In summary, 10 ml of hydromorphone (1.5 mg) administered via an epidural catheter inserted at the L₄₋₅ level provided thoracoabdominal incisional pain relief but did not provide analgesia for pain originating from the diaphragm. By increasing the volume to 15 ml, we were able to achieve complete analgesia. The larger volume of solution enabled the hydromorphone to reach the cervical spinal cord levels needed for complete analgesia in our patient, levels that apparently were not reached with 10 ml of solution administered at the lumbar level.

REFERENCES

Noninvasive Detection of Profound Arterial Desaturations Using a Pulse Oximetry Device

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Current monitoring of blood oxygenation in patients with respiratory failure requires analysis of arterial blood gases, which is invasive, expensive, and provides only intermittent information. Furthermore, this type of analysis takes several minutes (depending on institutional facilities), which could prove to be crucial in a given clinical situation. Devices like the pulse oximeter,¹ which purport to measure oxygenation noninvasively, are attractive because they provide continuous information that may result in improved patient care. Pulse oximetry is similar to classical oximetry in that discreet wavelengths of light are used to measure optical density of hemoglobin but is unique in that it can distinguish arterial blood from venous blood and tissue. Pulse oximeters are essentially multiple-wavelength plethysmographs. The pulse amplitude detected is a function of the arterial distension, hemoglobin oxygen saturation of the inflow of arterial blood, and the wavelength of light. When two wavelengths are utilized, the pulse amplitude of the two wavelengths will change relative to each other as the arterial hemoglobin oxygen saturation changes. A ratio of one pulse amplitude to the other will change directly as arterial hemoglobin oxygen saturation changes.¹ We evaluated finger pulse oximetry as a measure of arterial hemoglobin oxygen saturation in critically ill patients with respiratory distress or failure.

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Received from the Department of Anesthesia, Stanford University School of Medicine, Stanford, California 94305. Accepted for publication July 30, 1984. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, Atlanta, Georgia, October 1983.

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Key words: Equipment; pulse oximetry. Oxygen: tension; saturation; blood levels.