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REFERENCE

1. James FM: The anesthesiology triad in obstetrics. ANESTHESIOLOGY 56:335–336, 1982

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Epidural Morphine in a Terminally Ill Patient

To the Editor:—We read with interest the report by Woods and Cohen[1] on the use of high-dose epidural morphine in a terminally ill patient. In the summer of 1981, we provided long-term epidural morphine analgesia for a patient with intense rectal pain. He was a 63-year-old man who in 1970 had undergone a low anterior resection for adenocarcinoma of the rectosigmoid colon. Local recurrence had been treated with irradiation. However, extension into the sacrum caused intense rectal pain which was inadequately relieved by systemic narcotics. The patient disliked the somnolence and sedation caused by the narcotics. He was not expected to live for more than three or four months. Since we were using epidural morphine for analgesia in surgical patients, we were asked to help in the care of this patient.

Morphine, 5 mg in 10 ml preservative-free saline (supplied by A. H. Robins Company, Richmond, Virginia), injected through a percutaneous Portex epidural catheter, provided complete analgesia for more than 24 h without any systemic side effects. We injected the morphine while he was in the hospital and showed his wife how to do the same when he was home. Over the next two weeks, the frequency of injections increased from once, then twice, and finally three times a day. We had heard from Eltherington at the Annual Scientific Meeting of the California Society of Anesthesiologists in June 1981, that this tachyphylaxis could be reversed by an epidural injection of 10 ml 1% lidocaine.[2] We were pleasantly surprised to find that we could achieve this in our patient, and did so again four more times over the next two months. We also had to replace the epidural catheter on about six occasions because of leaks that developed in the catheter. Epidural morphine analgesia was provided from June 30 to September 10, 1981. An alcohol block of sacral nerves 2, 3, and 4 was then performed as it appeared he was going to live longer than we originally thought. He died October 9, 1981. An autopsy was not performed.

We agree with Woods and Cohen that long-term epidural morphine facilitates the care of terminally ill patients who are in pain. We were pleased that we were able to restore analgesic efficacy with 1% lidocaine injected epidurally whenever tachyphylaxis occurred.

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REFERENCES

1. Woods WA, Cohen SE: High-dose epidural morphine in a terminally ill patient. ANESTHESIOLOGY 56:311–312, 1982
2. Chayen MS, Rudick V, Borvine A: Pain control with epidural injection of morphine. ANESTHESIOLOGY 53:338–339, 1980

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Collapse after Epidural Injection Following Inadvertant Dural Perforation

To the Editor:—The interesting report by Dr. Hodgkinson[1] concerning the course of epidural block following dural puncture is of great interest to all obstetric anesthesiologists. Three of his patients developed apparent total spinal anesthesia, and another three showed a high level of sensory loss, presumably associated with massive epidural block.[2]

Our practice following inadvertant dural puncture

(59 cases, incidence 0.6%) was originally to insert the epidural catheter in the same interspace and inject the reduced volume of local anesthetic as mentioned by Dr. Hodgkinson. However, two cases of suspected massive subdural (extra-arachnoid) block, following the injection of 15 ml 0.5% bupivacaine, have caused us to abandon the technique. These two patients showed typical features of massive subdural block,[2,3] with a slow onset of symptoms over 30–40 min, only moderate hypotension, and progressive respiratory depression and incoordination, rather than the sudden collapse and apnea seen with a classical total spinal block. Complete recovery occurred over two hours. Dr. Hodgkinson may well have seen the same clinical situation, as the description of his second patient would seem more typical of a massive subdural than a total spinal block. In his third patient there was also a long delay between the full dose of bupivacaine and the patient's collapse.

In our last ten cases of dural puncture, we have used the adjacent cephalad interspace for catheter insertion, but, unfortunately another case of subdural block has occurred, similar to the other two. Due to various technical difficulties, we have been unable to demonstrate the classic radiologic appearances[4,5] of subdural injection in any of these three patients.

In conclusion, we would join Dr. Hodgkinson in recommending great caution when proceeding to epidural injection following dural puncture as it appears that one or more of three major complications may occur: total spinal, massive subdural, or massive epidural block, with onset times ranging from a few seconds to forty minutes post-block.

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REFERENCES

1. Hodgkinson R: Total spinal block after epidural injection into an interspace adjacent to an inadvertant dural perforation. ANESTHESIOLOGY 55:593–595, 1981
2. Bromage PR: Epidural Analgesia. Philadelphia, WB Saunders, 1978, pp 654–659
3. Collier CB: Total spinal or massive subdural block. Anaesth Intensive care 10:92–93, 1982
4. Boys JE, Norman PF: Accidental subdural analgesia. Br J Anaesth 47:1111–1113, 1975
5. Mehta M, Maher R: Injection in the extra-arachnoid subdural space. Anaesthesia 32:760–766, 1977

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Additional Causes of Postoperative Respiratory Complications in Premature Infants

To the Editor:—The complications Dr. Steward[1] found while anesthetizing premature infants are similar to our experience. Several mechanisms were proposed for postoperative apnea and we would add two more:

(1) *Fatigue resulting from spontaneous ventilation.* Most of the infants were permitted to breathe spontaneously during anesthesia. Regardless of the breathing circuit, endotracheal intubation or the presence of a mask on the face, work of breathing increases during anesthesia.[2] Certain breathing devices, such as inappropriately small endotracheal tubes or sticky valves, may exacerbate this increase in work of breathing. Because fatigue may be increased by spontaneous ventilation, perhaps this factor may contribute to these respiratory complications.

(2) *Postoperative hypoxemia.* Induction of anesthesia is associated with a decrease in FRC and an increase in ventilation-perfusion mismatch.[3] Infants anesthetized with halothane have low tidal volumes and may have airway closure at end expiration.[4] The usual mechanisms by which infants maintain FRC (*e.g.*, sighing, expiration against a closed glottis[5]) do not occur during anesthesia. Thus, atelectasis and hypoventilation may lead to hypoxemia in the postoperative period which may persist for an unknown time. The premature infant has an abnormal ventilatory response to hypoxia (*i.e.*, a decrease rather than an increase in ventilation[6]), which may explain the incidence of postoperative apnea.

In summary, we believe that spontaneous ventilation during anesthesia and hypoxemia during the postoperative period should be considered as two additional causes of respiratory complications in the premature infant.

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