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Respiratory Effects of Pain in a Child after Thoracotomy

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Postoperative pain management in children has not been extensively studied, and several reports have indicated that pain management is often inadequate.^{1,2} Two arguments may be advanced in support of the need for improved pain relief in children. One is that relief of suffering is the responsibility of all physicians and humanitarian duty to patient calls for better pain relief. The second is that inadequately treated pain can lead to postoperative complications. Although it is often said that postoperative pain leads to splinting of the chest, which can lead to atelectasis and/or pneumonia, documentation of this statement in children is lacking. However, some may express the opinion that pain stimulates the patient to move and that opiates will lead to an increased risk of postoperative respiratory complications. I recently cared for a patient in whom there was clear documentation of an adverse respiratory effect of pain and the resultant splinting and improvement when adequate pain therapy was instituted.

REPORT OF A CASE

The patient was 14 mo old when she presented for resection of a cystic adenomatoid malformation of the right upper and right middle lobes of her lung. She had been previously healthy, and at 7 mo of age a chest radiograph revealed cystic lesions in her lung. Surgery was electively planned.

She was premedicated with rectal sodium thiamylal, and venous and arterial catheters were inserted. A caudal catheter was inserted 4 cm into the caudal canal but was not immediately injected. Anesthesia was

induced with halothane, muscle relaxation was achieved with pancuronium, and the patient's trachea was intubated. Mechanical ventilation was begun, and the patient was positioned in the right lateral decubitus position. A right posterolateral thoracotomy was performed through the 5th intercostal space, and the right upper lobe was resected. Two chest tubes were left in place.

Approximately 1.25 h prior to the end of surgery, 0.75 mg of preservative-free (PF) morphine was injected *via* the caudal catheter (75 $\mu\text{g}/\text{kg}$, patient weight = 10 kg). At the end of the procedure atropine and edrophonium were administered to reverse the residual neuromuscular blockade, and the patient's trachea was extubated while she was still deeply anesthetized, and she was taken to the recovery room. One hour later the patient was still sleepy, but airway reflexes were intact, and there was no evidence of pain. The patient was transferred to the intensive care unit.

Five hours and 30 min after the caudal administration of morphine, the patient was noted to be crying and splinting her chest and was unable to be consoled by her parents. She was using accessory muscles of respiration during exhalation. Arterial blood gas tensions while breathing 1 l/min oxygen were as follows: $\text{pH} = 7.21$; $\text{P}_{\text{CO}_2} = 64$ mmHg; $\text{P}_{\text{O}_2} = 94$ mmHg. Because the position of the caudal catheter had not been confirmed and because the duration of pain relief was somewhat shorter than that expected after caudal morphine,³ a total of 10 ml of 0.25% bupivacaine was injected *via* the caudal catheter. After 20 min splinting stopped and the patient appeared comfortable and also appeared to have a larger tidal volume. A pinprick sensory level at T6 was demonstrated. Arterial pH was 7.31, P_{CO_2} was 47 mmHg, and P_{O_2} was 149 mmHg. One milligram of PF morphine was administered *via* the caudal catheter with subsequent pain relief lasting for 8 h. When pain returned, the arterial pH was 7.28, P_{CO_2} was 52 mmHg, and P_{O_2} was 158 mmHg. PF morphine was again injected *via* the catheter, and after pain relief had been reestablished, pH was 7.33, P_{CO_2} was 44 mmHg, and P_{O_2} was 146 mmHg. Morphine was then administered *via* the caudal catheter every 8-12 h until it was removed on the third postoperative day. Satisfactory analgesia was maintained with acetaminophen with codeine, and the patient was discharged home on the fourth postoperative day.

DISCUSSION

This case presentation documents clear adverse respiratory effects of pain, with resolution after pain control was reestablished. When the patient was seen in the ICU, she was in pain, with subjective respiratory effects of

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splinting and diminished tidal volume, and objective elevation of P_{CO_2} and increased $A-aDO_2$. Both changes were reversed when demonstrable sensory analgesia was established. The respiratory changes were again seen prior to the next dose of PF morphine. In this patient inadequate analgesia interfered with respiratory function.

Extensive data on the respiratory effects of surgery and/or pain are not available in children, but it has been noted that there is a depression of the ventilatory response to CO_2 following surgery in children.^{4,5} In adults the ventilatory depression is explained by alterations in the mechanics of breathing induced by surgery and pain and by reflex changes in diaphragmatic function that are accentuated by pain.⁶⁻⁸

There are reasons to think that the respiratory effects of surgery and/or pain may be more significant in children because of differences in respiratory physiology. Because the chest wall is compliant,^{9,10} the infant must stabilize the chest wall at the same time as the diaphragm contracts to create negative pressure within the thorax. Active muscle contraction may be necessary, and attempts by the patient to stabilize the chest wall may result in pain from use of muscles that have been injured by surgery. Inability to stabilize the chest wall may lead to inadequate respiration. Such a phenomenon has been seen during anesthesia with halothane, which inhibits the chest wall component of respiration.¹¹ In the patient presented here, the hypercapnia may be attributed to a combination of mechanical instability accentuated by pain and diaphragmatic dysfunction.

The increased $A-aDO_2$ may be attributed to small areas of collapsed lung brought about by breathing at a low tidal volume. Return to larger tidal volumes after pain relief was established reduced the $A-aDO_2$. If pain resulting from movement of the chest wall is blocked, the patient is able to cough and deep breathe, and mechanical stabilization of the chest wall may be less inhibited. Treatment of pain in this patient reduced the use of accessory expiratory muscles and could have led to maintenance of a higher functional respiratory capacity. Areas of collapsed lung could have been reexpanded by the return to a more adequate tidal volume.

The decision to use a local anesthetic to provide pain relief was based on the fact that it had never been clearly established that the caudal catheter was correctly positioned and the clinical need of the patient for rapid pain relief. Furthermore, the patient's somnolence in the recovery room led to concern that the initial dose of caudal morphine was relatively large for the patient and that a repeat dose within 6 h of the first might have resulted in respiratory depression.

In summary, a case is reported in which a child with pain after thoracotomy had documented respiratory dysfunction, which was reversed with adequate pain therapy.

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