Postoperative Respiratory Failure Following Intercostal Block

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The postoperative pain of an abdominal incision can produce hypoventilation. In the presence of preexisting lung disease, respiratory failure may result. Postoperative intercostal nerve blocks have the advantage of providing analgesia without the central respiratory depression usually associated with narcotic analgesia.1 Thus intercostal nerve blocks might represent a more desirable technique in patients with preexisting lung disease. These patients, however, often depend on their intercostal muscles for adequate ventilation, and the theoretical advantages of intercostal nerve blocks may be overshadowed.

Report of a Case

A 60-year-old, 63-kg male required emergency reduction of a prolapsed ileostomy. He had a history of hypertension and angina on exertion, chronic obstructive pulmonary disease (dyspnea on walking one block), and peptic ulcer disease. He used an Aerolone吸入 inhaler and sublingual nitroglycerin on a p.r.n. basis. Physical examination revealed an apparently uncomfortable, thin white male with an arterial blood pressure of 160/80 mm Hg and a regular heart rate of 100 beats/min. He had an increased anteroposterior diameter of the chest with minimal respiratory excursions. Breath sounds were distant, and bilateral pleural effusions were present. Laboratory examination included a chest roentgenogram showing hyperinflation of both lungs, an electrocardiogram showing sinus tachycardia, and a ratio of forced expiratory volume in one second to forced vital capacity of 1.5 liters to 3.7 liters (40 per cent). With an FIO2 of 0.21, Pao2 was 58 mm Hg, Paco2 46 mm Hg, and PH 7.50. Initial serum creatinine was 4.2 mg/dl but fell to 3.0 mg/dl following infusion of 3 liters of 0.45 per cent saline solution.

On arrival in the anesthetic induction room, he complained of typical anginal pain, which responded to one sublingual nitroglycerin tablet. One- and one-half inches of nitroglycerin paste were applied to the patient's chest, and fentanyl, 75 µg, and diazepam, 5 mg, were administered intravenously (iv). A catheter was inserted into the radial artery. Propranolol, 0.5 mg, was given intravenously. Following thiopental, 320 mg, iv, two per cent enflurane was given via mask for 10 min. Following the iv administration of metocurine, 4 mg, the trachea was intubated. Additional metocurine, 6 mg, was given and ventilation was controlled at a tidal volume of 750 ml and a rate of 9 times/min. At the termination of surgery, neuromuscular blockade was reversed with neostigmine, 5.5 mg, and atropine, 1 mg, to produce a normal train-of-four response on a peripheral nerve stimulator. Before extubation of the trachea, lateral intercostal nerve blocks at the 6th to the 12th ribs were performed bilaterally using 0.25 per cent bupivacaine with 1:200,000 epinephrine, 4 ml at each rib.

On arrival in the recovery unit, the patient was unresponsive to voice, but was breathing with a clear airway and taking shallow breaths. With 6 liters of oxygen via nasal prongs, Pao2 was 79 torr, Paco2 76 torr, PH 7.32, and bicarbonate 29.9 mEq/l. Chest roentgenogram at this time showed good expansion bilaterally without pneumothorax. Phystostigmine, 2 mg, and naloxone, 0.1 mg, were given intravenously to reverse sedative medication. The patient awoke and sat up on his stretcher. He was noted to be tachycardic and recruiting accessory muscles on inspiration. Repeat Pao2 was 61 torr, Paco2 71 torr, PH 7.25, and bicarbonate 29.9 mEq/l. Although not subjectively weak, lower intercostal retractions and a scaphoid abdomen were evident. Inspiratory force measured through a mouthpiece was a negative 10 cm H2O. Fifty minutes after his arrival in the recovery unit, nasotracheal intubation was performed and ventilation was controlled overnight. The next morning he was allowed to breathe spontaneously via a T-piece attachment to the endotracheal tube, and on an FIO2 of 0.5, Pao2 was 90 torr, Paco2 54 torr, and PH 7.42. Following extubation of the trachea, the Pao2 was 81 torr, Paco2 43 torr, and PH 7.41. He continued to do well and was discharged from the hospital on the sixth postoperative day.

Discussion

As postoperative pain and narcotic analgesia have been documented to produce decreases in tidal volume and functional residual capacity, 2,3 regional block may offer a useful method for improving the patient's ability to maintain adequate ventilation and good cough effort. Our patient is an example of a paradoxical response in whom chest-wall anesthesia may have precipitated carbon dioxide retention and progressive hypoxemia. Other drugs may have contributed to his respiratory failure. He received diazepam, 7.5 mg, and fentanyl, 125 µg, 2.5 hours prior to his episode of failure. Surgery was performed.
under enflurane and nitrous oxide. These agents may all produce respiratory depression, but the length of time following the induction, his alertness, and subjective dyspnea and tachypnea prior to nasotracheal intubation would suggest minimum contribution of drug-induced central respiratory depression. Also, his ability to sit unassisted and recruit accessory muscles of respiration without subjective weakness argue against significant residual paralysis from muscle relaxants. Chest wall motor blockade with loss of the contribution of intercostal and rectus muscles to inspiration and exhalation appears to be the major cause of respiratory failure in this patient.

The loss of abdominal and lower intercostal muscle power can compromise ventilation, even in normal subjects. Eisele et al. found that four patients without preexisting pulmonary disease who were given spinal or epidural anesthesia to the T-1 motor level, had a 42–80 per cent reduction in inspiratory capacity.4 Freund et al. have similarly found that spinal anesthesia to the T-5 level also reduces expiratory reserve volume by 48 per cent.5 The work of Jakobson and Ivarsson suggested that functional residual capacity is reduced following intercostal block.6,7 Sharp et al. have further shown that patients with chronic obstructive disease may depend more on the rib-cage muscles than the diaphragm to produce tidal ventilation compared to healthy patients.8 Fourteen of their 20 patients produced maximal forced inspiration mostly by the use of rib-cage muscular motion. In 5 to 20 patients, they saw an unusual decrease in abdominal girth in normal tidal breathing on inspiration, and concluded that patients with COPD are liable to have abnormal diaphragmatic function and dependence on rib-cage musculature.

Thus, in patients with chronic pulmonary disease, the intercostal muscles may play a crucial role for ventilation. Intercostal nerve block following surgery may enhance the development of respiratory failure in this group. Patients with preexisting pulmonary disease would profit from a critical clinical appraisal of the contribution of their intercostal musculature to their "normal" ventilatory pattern before considering an intercostal nerve block. If an intercostal block is performed, an assessment of arterial blood gases and ventilatory flow measurements should be performed to assure the adequacy of ventilation.

REFERENCES

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The Case of the Errant Epidural Catheter

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Continuous lumbar epidural analgesia via catheter for analgesia during labor is a popular and effective technique. Occasional difficulty during removal of the catheter is usually resolved by flexing the patient's back and applying steady traction to the catheter. We present an instance of an epidural catheter which knotted itself around a strand of ligamentum flavum.

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