Anesth Prog 37:46–48 1990

Ineffective Ventilation During Conscious Sedation Due to Chest Wall Rigidity After Intravenous Midazolam and Fentanyl

William E. Ackerman, MD, James C. Phero, DMD, and Gregg T. Theodore, MD
Department of Anesthesia, University of Cincinnati College of Medicine, Cincinnati, Ohio

Chest wall rigidity has been reported after the administration of high-dose intravenous fentanyl. This case report supports the observation that low-dose intravenous fentanyl may also cause chest wall rigidity. The treatment of chest wall rigidity with naloxone or neuromuscular blocking agents is controversial. A discussion of the management of fentanyl-induced chest wall rigidity is presented.

Benzodiazepines and fentanyl are frequently used in combination for out patient surgical procedures because of their combined anxiolytic and analgesic effects. Unfortunately, intravenous fentanyl administration may be associated with chest wall rigidity that renders patient ventilation difficult, if not impossible. Truncal rigidity has been reported after administration of high-dose intravenous fentanyl.[1–4] Additionally, Vaughn and Bennett[5] have reported a case of a 22-year-old female who developed chest rigidity after administration of only 100 μg (2 ml) of intravenous fentanyl over 12 minutes.

Chest wall rigidity is a serious complication that requires immediate diagnosis and treatment. This rigidity may be severe enough to make bag and mask ventilation ineffective until pharmacologically managed.[6] Practitioners administering intravenous fentanyl should be familiar with airway management and the use of both narcotic antagonists and neuromuscular blocking agents.

CASE REPORT

Emergency anesthesia consultation was requested after an obstetrician administered 1 mg (1 ml) of midazolam and 100 μg (2 ml) of fentanyl intravenously in divided doses over five minutes to a 19-year-old female for sedation during dilation and curettage with a pudendal block for an incomplete abortion. The patient became apneic after the second 50-μg dose of fentanyl was administered. The consulting anesthesiologist was unable to ventilate the patient with a bag and mask system. The patient was immediately given 0.2 mg of intravenous naloxone. Within 30 seconds of the naloxone administration it was possible to ventilate the patient without difficulty. This patient was a healthy A.S.A. I female without positive history of pulmonary, cardiac, or anesthesia problems. She was not taking any medications before hospital admission. She was admitted to the recovery room and was discharged to next morning without sequelae.

DISCUSSION

Hypoxia and hypercarbia have been noted with the combination of narcotics and benzodiazepines for intravenous sedation. A recent study by Sokoll et al.[7] reported that when compared to midazolam or fentanyl administered alone, the combination of a benzodiazepine and fentanyl produced significantly more hypoxemia, suggesting that a benzodiazepine could augment the effects of fentanyl.

Fentanyl administered intravenously can produce marked truncal and abdominal muscle rigidity.[4] The site of action of fentanyl induced rigidity has not been specifically identified. Blasco et al., in 1972, reported that fentanyl-induced rigidity originates at higher centers than the spinal cord reflex arc.[8] There is evidence in laboratory animals that intravenous opioid-induced rigidity may be centrally mediated related to dopamenergic neurons within the basal ganglia.[9,10]

Data published by Ellenbroek et al. indicated that fen-

Received March 21, 1989; accepted for publication December 28, 1989.
Address correspondence to William E. Ackerman, MD, Department of Anesthesia, University of Cincinnati, College of Medicine, 231 Bethesda Avenue, Cincinnati, OH 45267-0531.

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ISSN 0003-3006/90/$3.50

tanyl could cause glottic rigidity, resulting in glottic closure with subsequent upper airway obstruction.[9] However, of more recent concern is the evidence that benzodiazepines may decrease genioglossus activity in humans possibly resulting in increased upper airway resistance.[11] Additionally, with the benzodiazepine, midazolam, a more pronounced depressive effect was noted on pharyngeal rather than on inspiratory muscle activity.[12] In this case presentation, benzodiazepine-induced upper airway resistance would not explain the fact that this patient was reversed with naloxone. It is unlikely that naloxone reverses central effects caused by benzodiazepines,[13] and thus it appears that midazolam did not cause the respiratory depression observed. In other instances of chest wall rigidity reported by various authors, a benzodiazepine was administered in combination with fentanyl.[14–16] However, in the report by Vaughn and Bennett 100 μg of fentanyl was administered without a benzodiazepine before the onset of truncal rigidity.[5]

Recommended treatment for fentanyl-induced rigidity is with either the intravenous administration of naloxone or neuromuscular blockers.[14] When considering the use of naloxone for chest wall rigidity, one should be reminded of the complications related to naloxone administration. In mice, naloxone even in low doses elevates brain tissue concentrations of dopamine within minutes after administration.[17,18] Sudden antagonism of fentanyl with naloxone has caused adverse changes in cardiovascular hemodynamics secondary to catecholamine activity.[19] Naloxone in low doses may increase heart rate, cardiac index, blood pressure, and ultimately myocardial oxygen consumption.[19] As a result, the practitioner should use care in the administration of naloxone to patients who have a history of ischemic cardiovascular disease and/or mitral or aortic valve prolapse. In these patients increased heart rate or systemic vascular resistance could have serious effects on the myocardial oxygen supply and demand interactions in these patients.

When considering the use of neuromuscular blockers to decrease chest wall rigidity after fentanyl administration, one must consider when the patient last ingested solids and/or liquids. If ingestion occurred within reasonable proximity to the time of fentanyl administration (≤6 hours), one must consider the possibility of a full stomach and subsequently perform rapid sequence intubation using succinylcholine. Nondepolarizing muscle relaxers have been shown to be effective in attenuating fentanyl-induced truncal rigidity.[20] When using metocurine or pancuronium, the disappearance of fentanyl-induced rigidity was shown to occur before any observable change in the train-of-four response.[20] When compared to succinylcholine, the problems with using a nondepolarizing muscle relaxant are slow onset of action and significantly longer duration of action. If a patient has a history of a difficult intubation or appears to be a potentially difficult airway, it may be prudent not to administer a muscle relaxant.

The actual mechanism of fentanyl-induced chest wall rigidity remains unanswered. However, practitioners who choose to administer fentanyl intravenously must be aware that benzodiazepines potentiate the effect of fentanyl. One must also consider that naloxone administration may cause severe adverse hemodynamic effects in patients with occlusive cardiovascular disease or incompetent valvular disease. Additionally, because naloxone has a shorter elimination half-life than fentanyl, patients who develop truncal rigidity and are treated with naloxone must be carefully observed for several hours after the naloxone administration.[16,21]

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