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Anesthesiology  
63:199-201, 1985

## Postoperative Rigidity Following Sufentanil Administration

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Chest wall rigidity after administration of narcotics first was reported by Hamilton and Cullen<sup>1</sup> in 1953. Morphine,<sup>2</sup> meperidine,<sup>3</sup> fentanyl,<sup>4-5</sup> fentanyl-droperidol<sup>3</sup> (Innovar®), and sufentanil<sup>5</sup> can produce decreased chest wall compliance and ventilatory difficulty. Following high-dose fentanyl (70-115 µg/kg), chest wall rigidity may occur several hours after surgery.<sup>6</sup> We report a patient in whom chest wall rigidity developed in the recovery room 3 hrs after administration of 4 µg/kg of sufentanil.

### REPORT OF A CASE

A previously healthy 36-year-old, 75-kg ASA physical status I man was scheduled for an elective lumbar laminectomy for disc herniation. He was premedicated with hydromorphone 1 mg and scopolamine 0.43 mg im, and on arrival to the operating room was awake and cooperative. Awake endotracheal intubation was accomplished after topical application to the tongue and hypopharynx of lidocaine 10% spray, bilateral superior laryngeal nerve blocks with lidocaine 4%, and 4 ml intratracheal lidocaine 4% under direct vision. Diazepam 5 mg iv in divided doses was required for additional sedation during the intubation. Sufentanil 0.5 µg/kg (37 µg) iv was then given to provide analgesia for bladder catheterization, during which the patient was somnolent, spontaneously ventilating, and analgetic.

After insertion of the Foley catheter, the patient was easily aroused. He was able to move himself onto the operating room table into the prone position and then gave the "thumbs-up" sign. General anesthesia was induced with sufentanil 3.5 µg/kg (263 µg) and pancuronium 5 mg iv and maintained with nitrous oxide/oxygen

70/30%. Surgery proceeded uneventfully; additional pancuronium 2 mg was given 1 h before the end of surgery. The operation concluded 2.5 h after the induction dose of sufentanil; no additional narcotic had been given since induction. Following iv administration of neostigmine 3.5 mg, physostigmine 1 mg, and glycopyrrolate 0.4 mg iv, the patient was awake and alert. Sustained tetanus (100 Hz) was demonstrated with a peripheral nerve stimulator. His vital capacity was 700 ml, and his respiratory rate was 12 breaths/min. He was strong enough to lift himself above the Wilson frame for 10 s, and after he turned to the supine position, his trachea was extubated. Immediately after extubation he was alert and asked questions about his surgery. In the recovery room, BP was 156/80 mmHg, HR 70 bpm, respiratory rate 12 breaths/min, and temperature was 35.2° C. His ventilation was judged to be "adequate" by the recovery room personnel, and cyanosis was not evident. Oxygen, 5 l/min was administered via nasal cannula. At no time was any narcotic or narcotic antagonist administered.

Over the next 22 min his respiratory rate and depth gradually decreased until he required verbal stimulation to breathe. His blood pressure was 120/80 mmHg, and his heart rate was 68 bpm. He then became rigid, cyanotic, and unresponsive. It was impossible to open his mouth or to ventilate him by mask, even with a nasal airway in place. He was given succinylcholine 100 mg iv, and within seconds his chest wall compliance increased. His trachea was reintubated, and, although arousable, he was somnolent. His ventilation via T-piece was inadequate, due to insufficient effort, and mechanical ventilation was required for three additional hours. During this time he exhibited strong hand grip, sustained head lift and arousal with stimulation. Arterial blood gas (ABG) before the second extubation (30 min while breathing through a T-piece with an O<sub>2</sub> flow rate of 6 ls/min) showed  $pH_a$  7.37,  $PaO_2$  130 mmHg,  $PaCO_2$  48 mmHg, and base excess +2 mEq/l. Although still somnolent, his ventilation had improved; after extubation his arterial  $pH_a$  was 7.36,  $PaCO_2$  48 mmHg,  $PaO_2$  135 mmHg, and base excess +1.9 mEq/l. His first requirement for analgesic medication was 15 h postoperatively.

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Received from the Department of Anesthesiology, Hahnemann University Hospital, Broad and Vine Streets, Philadelphia, Pennsylvania 19102. Accepted for publication March 6, 1985.

Key words: Anesthetics, intravenous; sufentanil. Complications: rigidity.

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### DISCUSSION

Chest wall rigidity during narcotic administration, whether for small doses used for sedation or large doses used for anesthetic induction, is not uncommon. The incidence, however, is disputed. Lunn *et al.*<sup>7</sup> either failed

to mention chest wall rigidity or state that in 18 patients "... decreased chest wall compliance (occurred with fentanyl) but not to the point of rigidity. . . ." However, Waller *et al.*<sup>4</sup> found chest wall rigidity occurred before the loss of consciousness in all of 12 patients given fentanyl. Twenty of 21 patients studied by Comstock *et al.*<sup>18</sup> had rigidity develop with fentanyl, as did seven of 21 patients anesthetized with fentanyl by Stoelting *et al.*<sup>9</sup> Quintin *et al.*<sup>10</sup> and Wynands *et al.*<sup>11</sup> administered pancuronium 1 mg before giving fentanyl and observed no chest wall rigidity. De Lange *et al.*<sup>15</sup> reported a 28% incidence of chest wall rigidity on induction of anesthesia with sufentanil infused at  $300 \mu\text{g} \cdot \text{min}^{-1}$ .

Factors that allegedly contribute to rigidity include concomitant use of nitrous oxide, though it is not a prerequisite.<sup>2,3</sup> Our patient undoubtedly had a negligible alveolar concentration of nitrous oxide when his rigidity occurred because the nitrous oxide had been discontinued 35 min previously. Fentanyl has been shown to be absorbed and resecreted by the gastric mucosa<sup>12</sup>; Christian *et al.* suggest this mechanism to explain the postoperative rigidity seen in their patients.<sup>6</sup>

The cause of narcotic-induced muscular rigidity is not understood. Freund *et al.*<sup>2</sup> compared abdominal muscle electromyographic activity with activity of local stretch-receptor reflex arcs. Because EMG activity was increased while reflex arc activity was decreased, they suggested that the source of rigidity is supraspinal. Benthuyssen *et al.*<sup>13</sup> and Freund<sup>2</sup> found that rigidity occurred in all muscle groups. However, Scamman<sup>14</sup> reported that the cause of narcotic-induced inability to ventilate was rigidity of the glottic musculature, which caused glottic closure.

Rigidity does not seem to occur, or is reversed, when narcotic agonist-antagonist drugs such as pentazocine and levallorphan are used.<sup>15</sup> Perhaps one of the four different proposed narcotic agonist receptors<sup>16</sup> ( $\mu$ ,  $\kappa$ ,  $\sigma$ ,  $\delta$ ) is the central receptor that causes the rigidity, and antagonism at that receptor would eliminate the rigidity. This hypothesis has not been tested during clinical use of narcotics, since it is either undesirable to reverse the narcotic effect (as during a narcotic induction) and because the syndrome causes such a complete inability to ventilate the patient that immediate paralysis is required (as happened to our patient).

We believe that the most likely cause of the striking rigidity observed in our patient was the residual effect of sufentanil. It is possible that our patient's rigidity was due to progressive respiratory depression and hypoxia. Becker *et al.*<sup>17</sup> noted a biphasic depression of the respiratory response to  $\text{CO}_2$  with fentanyl, with the second decline in  $\text{CO}_2$  responsiveness occurring about 150 min after the initial dose of narcotic. However, the degree

and quality of the rigidity we observed resembled the rigidity usually seen shortly after the administration of a narcotic. When this case occurred, sufentanil in the dose range of  $2\text{--}6 \mu\text{g}/\text{kg}$  was purported to have a recovery time "comparable to enflurane, isoflurane, and fentanyl."<sup>‡</sup> With an elimination half-time ( $t_{1/2\beta}$ ) of 140 min at a dose of  $5 \mu\text{g}/\text{kg}$  of sufentanil<sup>18</sup> versus 219 min for fentanyl,<sup>19</sup> and a potency widely believed to be 10 times that of fentanyl,<sup>5</sup> our patient probably had a serum sufentanil concentration compatible with both respiratory depression and narcotic induced muscle rigidity.

We would feel more certain of the diagnosis of narcotic-induced rigidity if naloxone rather than succinylcholine had been used to treat the rigidity. Christian *et al.* did successfully use naloxone to treat rigidity in one of his three patients.<sup>6</sup> We suggest careful postoperative surveillance for the occurrence of chest wall rigidity and respiratory depression after the stimulation of surgery has ended for patients who have received sufentanil in the dose range employed here.

‡ Sufenta® package insert, Janssen Pharmaceutica, Inc., Piscataway, New Jersey, 1984.

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Anesthesiology  
63:201-202, 1985

## Anesthetic Considerations in Cri Du Chat Syndrome: A Report of Three Cases

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Limited consideration has been given to the anesthetic management of patients with cri du chat syndrome.<sup>1,2</sup> This is a syndrome associated with a unique mewing quality like that of a kitten and is characterized by a partial deletion of the short-arm of chromosome number 5.<sup>3</sup> A recent report<sup>4</sup> on home-reared children indicates that the prognosis of these patients is more favorable than previously described in studies of institutionalized patients. Thus, we may see an increasing number of these patients requiring anesthesia and surgery.

We report our anesthetic experiences on three patients with cri du chat syndrome.

### REPORT OF THREE CASES

**Patient 1.** This 2,600-g infant was born to a 29-year-old mother after an uncomplicated gestation. The infant was mildly depressed at birth (Apgar 6) but responded quickly to suctioning and oxygen

given via a mask. Because of characteristic facial features (microcephaly, rounded face, hypertelorism, oblique palpebral fissures, epicanthus, low-set ears, and micrognathia) and cry, the diagnosis of cri du chat syndrome was made. Subsequent chromosomal analysis confirmed this clinical diagnosis. Other findings on physical examination included the following: simian crease, umbilical hernia, prolapsus recti, dislocation of the hip, and lumbosacral meningocele. The operation was scheduled for repair of the meningocele at 7 weeks of age, at which time body weight was 3,890 g.

After the patient received scopolamine 0.05 mg im, anesthesia was induced and maintained with halothane, nitrous oxide, and oxygen. A 2.5-mm ID endotracheal tube was inserted into the trachea without the use of muscle relaxants. No apparent abnormalities were noticed in the larynx. Repair of lumbosacral meningocele was completed without incident. The trachea was extubated uneventfully shortly after surgery. Mild respiratory distress (tachypnea, retractions) was noticed approximately 6 h later; this resolved over the next 2 h, and the remainder of the postoperative course was uneventful.

**Patient 2.** This 3,020-g product of an uncomplicated gestation was mildly asphyxiated at birth but responded to oxygen given via a mask. Shortly after birth, the infant was clinically diagnosed as having cri du chat syndrome. In addition, physical examination revealed a heart murmur. Cardiac catheterization revealed a patent ductus arteriosus (PDA) and pulmonary hypertension. The infant was scheduled for ligation of her PDA.

After premedication with scopolamine 0.03 mg im, anesthesia was induced with halothane, nitrous oxide, and oxygen. The trachea was intubated (tube size ID 3.5 mm) with the aid of succinylcholine 2.5 mg iv. Because of retromicrognathia and a long, curved, floppy epiglottis, visualization of the vocal cords was limited. For surgery the infant was placed in the right lateral position. Several minor ventilatory problems occurred during surgery: left upper lobe atelectasis, which seemed to respond to increase in peak airway pressure; and bloody secretions requiring vigorous suctioning. The trachea remained intubated for 4 h postoperatively until the cardiovascular system was stable. The postoperative chest roentgenogram was unremarkable. The infant's subsequent course was uneventful until day four, when she died after aspiration of milk, probably attributable

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Received from the Department of Anesthesiology, University of Hirosaki School of Medicine, Hirosaki 036, Japan, and the Department of Anesthesiology, Ohita Medical College, Ohita 879-56, Japan. Accepted for publication March 6, 1985.

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Key words: Cri du chat syndrome. Airway: anatomy. Intubation: complications.