MUSCLE RIGIDITY WITH FENTANYL: A CASE REPORT†

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Fentanyl (Sublimaze[R]) is a potent narcotic analgetic of rapid action and short duration, therefore highly suitable for outpatient dental anesthesia. A side effect, namely rigidity of jaw, neck, chest and abdominal musculature associated with fentanyl-containing compounds, first noted by Hamilton and Cullen[1], is not uncommon. Chest wall rigidity can be a serious complication and must be promptly diagnosed and treated when administering fentanyl to avert respiratory and cardiovascular complications subsequent to hypercarbia and hypoxemia. Fentanyl should not be administered by those without training in anesthesia which would include the use of muscle relaxants and airway management.

Case Report

A 53 year old, 72 kg., white male was brought to the operating room for hemorridectomy with no significant medical problems. The patient denied any allergies or medications and had smoked 25 pack years. Laboratory data including chemistries (*Na* 140, K 4.4., Cl 101, CO_2 28) and hematocrit (42.1) were within normal limits. Chest x-ray and EKG were also normal.

The patient was premedicated with 10 mg. morphine sulphate, 10 mg. diazepam and .4 mg. atropine intramuscularly. An 18 gauge intravenous catheter was placed and 500 cc. of 5% dextrose was started.

Fentanyl (.05 mg.) was slowly injected over a one minute period. Oxygen and nitrous oxide (2:4 liters) was administered by mask, and a test dose of 50 mg. thiopental injected followed by a sleep dose of 250 mg. thiopental. Fentanyl (.10 mg.) was then injected over a five minute period. The respiratory frequency was 9 per minute and no difficutly was encountered in maintaining an airway.

Three minutes after the last dose of fentanyl the patient stopped breathing and could not be ventilated. An oral airway was inserted. The pharynx was expanding with controlled ventilation, but no chest movements nor breath sounds could be detected.

The vocal cords were viewed by direct laryngoscopy and there was no evidence of laryngospasm. Succinlycholine 20 mg. IV. was injected, and after 30 seconds the patient could be easily ventilated. Arterial blood gases were pH 7.32, $p0_2$ 90, and pCO_2 49 at that time.

Five minutes later rigidity again developed. The larynx was again visualized and no evidence of laryngospasm could be detected. Succinylcholine (25 mg.) was again injected, the patient ventilated, and intubated without difficulty with a #9.0 cuffed endotracheal tube. The case proceeded without incident with controlled ventilation. Arterial blood gases were pH 7.40, $p0_2$ 130, pCO_2 39.

Fentanyl (.05 mg.) again was slowly injected towards the end of the procedure and muscle rigidity developed immediately which was reversed with 4 mg. naloxone (Narcan[R]). Ten minutes later with the respiratory frequenty of 15/min., inspiratory force over—35 cm. H_2O, tidal volume of 600 cc. and the patient responding to commands, the patient was extubated. No further complications were observed in the recovery room or during the next two days on the floor.

Discussion

The onset of muscle rigidity after intravenous administration of fentanyl or fentanyl- containing compounds is not uncommon[2,3,4,5]. This rigidity is similar to the

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"lead pipe" rigidity produced in rats with morphine and seen occasionally in man with morphine in high doses combined with nitrous oxide[1]. Electromyographic studies have demonstrated that many narcotics produce an increase in abdominal muscle tone especially when supplemented with nitrous oxide[5,6].

Kallos et al[7] demonstrated that fentanyl-containing compounds reduced total lung compliance and functional residual capacity (FRC). The reversal of these phenomena by succinlycholine indicated that increased expiratory muscle activity was present even in the absence of clinically evident rigidity. They concluded that in patients where reduction in FRC may be undesirable, fentanyl should be administered with caution.

Sensitization of the stretch reflex arc ("H" reflex) is one of the mechanisms proposed for the increased muscle tone seen with fentanyl, but recent work has cast some doubt on this theory.[5,6,8] It appears that the origin of rigidity must lie outside the stretch reflex arc and in some other yet undetected area of the spinal cord or higher area of the central nervous system.[5,9]

All narcotics depress respiration and can produce apnea. Foldes et al[10,11] have shown a close correlation between the onset of apnea and ventilatory difficulties with fentanyl, of these, rigidity has been observed in patients adequately ventilated with oxygen rich mixtures.[12]

Chest wall rigidity has been observed with rapid intravenous injection of fentanyl-containing compounds.[11,13] Small doses of fentanyl injected slowly has been advocated by Foldes et al[11] to reduce the incidence of rigidity. Others suggest continuous dilute intravenous drip techniques to surmount this problem.[14] Grell et al[4] reported that muscular rigidity in their patients did not occur until a minimum of .25 mg. of fentanyl had been injected, but in our experience rigidity has occurred at lower doses.

Short-acting, depolarizing muscle relaxants (succinylcholine 20-40 mg.) overcome chest wall rigidity, but as this case report indicated, the rigidity may reappear after the action of the relaxant has ceased. In a study of 500 cases, no rigidity developed in patients intubated with thiopental, succinylcholine, and large doses of fentanyl. It was postulated that the residual blood level of succinylcholine prevented rigidity initially, but offered no protection against recurrance of rigidity with later repeat doses of fentanyl.[4]

In the case presented and in dental outpatient anesthesia not utilizing an endotracheal intubation technique, succinylcholine is not employed and one might hypothesize a higher incidence of muscle rigidity. It is important to note that only a small dose of succinylcholine is necessary to adequately ventilate the patient. Succinlycholine administered to a hypercarbic and hypoxic patient can cause cardiac arrthymias, most commonly bradycardia, hypotension, and even cardiac arrest, making early detection of paramount importance.

The muscle rigidity and respiratory depressent effects of fentanyl are reversible with the administration of small doses of narcotic antagonist (naloxone .2-.4 mg.). It must be remembered that in most cases analgesia and sedation are also reversed.

Nondepolarizing muscle relaxants, such as curare and pancuronium bromide (Pavulon®) can also overcome chest wall rigidity and allow the patient to be ventilated, but their onset is much slower than succinylcholine and one is left with a curarized patient who must be ventilated and adequately reversed. The rapid onset of action and ability to maintain anesthetic depth make succinylcholine the treatment of choice for muscle rigidity. It is also important to note that succinylcholine will relieve both rigidity and laryngospasm without removing the cause of laryngospasm, but will not alleviate bronchospasm which to the untrained may mimic rigidity.

Despite the normal arterial blood gases and the slow rate of injection of fentanyl at the end of the case, rigidity developed with a small increment (.05 mg.) demonstrating that despite all precautions this complication must be anticipated.

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Letters to the Editor

McKESSON ISSUES ALERT

February 4, 1977

Dear Sir:

The McKesson Company would like to bring to your attention a very serious and urgent problem that exists in the equipment used for anesthesia and analgesia. This equipment manufactured prior to the adoption of standard in 1959 regulating the sizes and connection configuration, referred to as Diameter Index Safety System (DISS), poses a dangerous hazard. This problem is not limited to any one manufacturer but exists on all such equipment manufactured prior to the adoption of the DISS standard. We strongly urge you to use the power of communication available through your organization and your publications to inform your members of this serious problem. The solution can be accomplished at very low cost. All that is required is the removal from the equipment of the existing standard pipe fittings and replacing them with new DISS fittings. This will also require new flexible hose connections from the machine to the wall outlets. This modification should be done by returning your machine to the original manufacturer. This modification will eliminate the inadvertent misconnection of the Nitrous gas to the oxygen supply line.

I am certain you will convey McKesson's concern on this matter to all your members to ensure the safety of their patients.

Yours truly,

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