THE FENTANYL-INDUCED COUGH AT INDUCTION

- The Influence of Premedication and Smoking -

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Summary

The history of smoking and premedication did not influence the proportion of patients who had a cough response to fentanyl when administrated as first agent during induction in anesthesia.

Introduction

Intravenous administration of fentanyl is a common practice to reduce the hyperdynamic response to tracheal intubation during induction of anesthesia.

However, this may be accompanied by cough[1]. Coughing upon induction of anesthesia is undesirable in some patients, e.g open eye injury or increased intracranical pressure. In these situations, fentanyl may not be suitable as the first drug of anesthetic induction sequence.

In the present study we evaluated the influence of smoking history and premedication of fentanyl-induced during induction of anesthesia.

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Material and methods

After IRB approval, 50 patients (M/F: 22/28), ASA I-II, scheduled to undergo elective surgery were included. Patients were randomly allocated in two groups. In group A (n = 24) patients received oral premedication with temazepam 20 mg two hours before induction and in group B (n = 26) patients without premedication In the operating room venous access was established. Standard monitoring included non-invasive blood pressure measurement, ECG and pulse oximetry.

In both groups, IV fentanyl 2-3 μg/kg was administrated as first agent during induction in anesthesia.

Parameters recorded included presence or absence of cough for the first two minutes after the IV injection of fentanyl and presence of history of smoking (>10 cigarettes/day).

Results

There were no demographic differences between groups. Fentanyl-induced coughing on induction of anesthesia presented in 5/24 (21%) of premedicated patients, and in 6/26 (23%) of non premedicated patients, two-tailed p = 0.27 (non statistical) significant, Fisher's exact test). The smokers were totally 23/50 (46%) of patients, 12/25 (48%) in group A and 11/25 (44%) in group B. Concerning the influence of smoking on history, fentanyl-induced coughing on induction of anesthesia presented in 6/23 (26%) of smokers and in 6/27 (22.2%) of nonsmokers (non statistical significant, Fisher's exact test).

Discussion

Our study demonstrated that history of smoking and premedication did not influence the proportion of patients who had a cough response to fentanyl when administrated as first agent during induction in anesthesia. Phua and Agarwal reported similar results with an incidence 28% of cough following a dose of injected fentanyl between 1,5-2 $\mu g/kg^{-1}$ [2,3].

However, Bohrer observed a 45% incidence of cough when fentanyl 7 $\mu g/kg^{-1}$ was given through a central venous line and Lui reported a 43% incidence of fentanyl-induced cough using a dose of 5 $\mu g/kg^{-1}$ [4,6]. The differences in the incidence of cough may be ascribed to the doses and routes of administration.

Various mechanisms have been proposed to explain fentanyl-induced cough. Opioid induced coughing is elicited within seconds of drug injection. This phenomenon can be explained by the presence of a pulmonary chemoreflex, mediated by C-fiber receptors (also known as J-receptors is thought to underlie this phenomenon). Opioid receptors have been shown in smooth muscles of the trachea and bronchi and in alveolar walls, but not in the small airways. Alveolar wall opioid receptors may be associated with J receptors. Fentanyl constricts the tracheal smooth muscle and hence the irritant receptors nearby may be stimulated secondary to deformation of the tracheobronchial wall. These receptors, when stimulated can trigger the cough reflex. Irritant receptors, also known as rapidly adapting or cough receptors, appear to be the more likely candidate for cough because they are highly concentrated in the walls of proximal tracheobronchial airway and found superficially within the mucosa.

Bohrer speculated that pulmonary C-fibre receptors or J-receptors with its nonmyelinated afferent fibres are most likely involved in the mediation of the pulmonary chemoreflex that leads to cough evoked by fentanyl[6]. This possibly occurs because J-receptors are readily accessible via pulmonary circulation and are more sensitive to chemical irritants.

Bailey stated that opioid-induced coughing is frequently more noticeable in patients who smoke[1]. However, in our study the incidence of smokers and non smokers among the patients who presented cough after IV administration of fentanyl, was similar (26% and 22.2% respectively).

The cough reflex opioid injection is not likely to be vagally medicated because atropine pretreatment does not affect it[2]. However, pretreatment with inhaled $ß_2$-adrenergic bronchodilator (terbutaline) or with corticosteroid (beclomethasone) that reduce bronchial

hyperrirritability, mucosal edema and suppress the inflammatory response to trigger stimuli, can reduce the cough evoked by fentanyl[3,4].

Bailey stated that fentanyl-induced cough is brief and self-limited. But Tweed presented a case-report of explosive, spasmodic coughing after peripheral IV injection of fentanyl to a child 7 years old that was relieved after induction or general anesthesia[5].

Another possible mechanism of fentanyl-induced cough is histamine release, though this appears very unlikely. Fentanyl, in contrast to morphine, rarely causes histamine release in mast cells of human lung. Release of neuropeptides, by activation of pesynaptic sensory C-fibres, can also induce bronchoconstriction and cough. In the study of Agarwal et al. inhalation of sodium chromoglycate decreased the incidence of cough[3]. In conclusion no influence of smoking and premedication was found on fentanyl-induced coughing during induction in anesthesia.

References

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