If all the determinations realized in the same individual are not independant as clearly shown, the bias is not affected and the standard deviation of the bias is underestimated.

Yours faithfully,

P. Bizouarn

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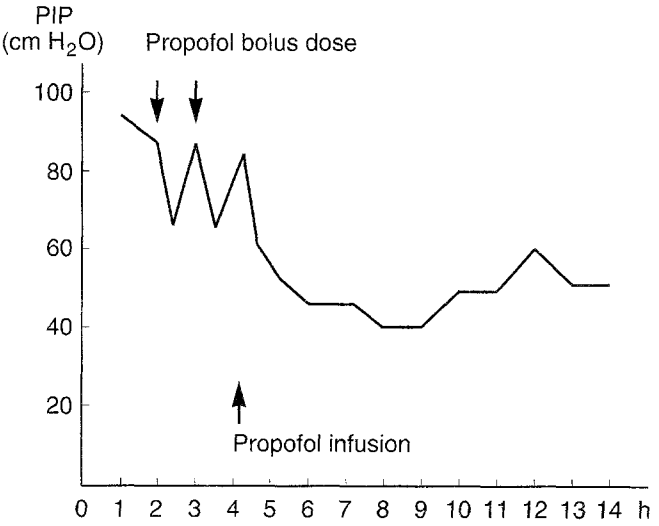

Fig. 1. Modifications of peak inspiratory pressure with unmodified tidal volume in the first 14 h of treatment

Propofol induces bronchodilation in a patient mechanically ventilated for status asthmaticus

Dear Sir,

Severe status asthmaticus (SA) is commonly observed in ICU and is treated with $\beta 2$ mimetic drug, aminophylline, steroids with associated mechanical ventilatory support. In the past, a large incidence of barotrauma has been reported in SA patients receiving mechanical ventilation; therefore SA patients are generally anesthetized, paralyzed and ventilated with low respiratory frequencies and low tidal volumes [1]. Heavy sedation is prescribed to obtain machine adaptation and respiratory drive reduction; however, the presence of intrinsic bronchodilation properties has never been reported for the sedatives of common clinical use. Propofol is currently used in ICU for long term sedation of mechanically ventilated adult patients [2]; it has been shown recently that propofol prevents fentanyl induced bronchoconstriction and induces bronchodilation, in patients with chronic hyperreactive disease and chronic obstructive pulmonary disease [3–5]. We report a patient with status asthmaticus, mechanically ventilated after heavy sedation, in wich the administration of propofol produced an important bronchodilaton.

A 23-year-old man was transferred to our ICU from the emergency room where he had been intubated and ventilated after a cardiac arrest due to status asthmaticus, unresponsive to conventional treatment (aminophylline 480 mg i.v., adrenaline 0.5 mg s.c. and methylprednisolone 2 g i.v.). On ICU admission he was manually ventilated, as mechanical ventilation was impossible (peak inspiratory pressure 100 cmH_2O), despite heavy sedation with flunitrazepam (5 mg i.v.) and paralysis with pancuronium bromide (8 mg i.v.). After 20 min of manual ventilation with oxygen 100%, he had a respiratory acidosis (pH 7.21, PaO_2 107, $PaCO_2$ 71, BE −5); connected to a Servo 900 C (Siemens) with the following settings: RR 8/min^{-1}; Vt 10 ml Kg^{-1}, I:E 1:2, square wave, no PEEP, FIO_2 0.5, he showed high peak inspiratory pressure (90 cmH_2O) and high values of intrinsic PEEP, (20 cmH_2O) evaluated with a prolonged tele-expiratory airway occlusion. Continuous infusion of salbutamol was started (0.6 mg Kg^{-1}) and sedation with flunitrazepam i.v. infusion was continued (0.1 mg/Kg/h); at this dose the patient was areflexic. After 2 h bronchospasm persisted despite salbutamol dose increase to 1.2 mg/Kg and a bolus dose of prednisolone (400 mg i.v.). Sedation with propofol was attempted: after a bolus dose of 70 mg was administered, peak inspiratory pressure decreased to 70 cmH_2O. The effect lasted for 10 min, and a subsequent slow increase to the preceeding values of peak impiratory pressure was observed. A second dose of 70 mg was administered, producing again a dramatic decrease in peak inspiratory pressure from 90–63 cmH_2O. Also PEEPi and total resistances of the respiratory system showed a decrease, from 14–10.5 H_2O and from 25.5–15.5 $cmH_2O\,l^{-1}\,s^{-2}$, respectively. In view of this good result, sedation with propofol was continued for 10 h, observing a constant decrease of peak inspiratory pressure and autoPEEP values. The following day the patient was rapidly weaned and extubated and, after 5 days, he was discharged in good clinical condition. In this case report propofol induced bronchodilation has been proved not only by measuring peak inspiratory pressure and autoPEEP values but also with the direct measurement of the total resistances of the respiratory system.

Non-specific bronchodilation effect has been observed in status asthmaticus with sedation; however we administered propofol in an already anesthetized patient, and the relationship between injection and bronchodilatation was observed several times (Fig. 1). The use of propofol as a sedative agent could be an interesting alternative in view of the rapid metabolism and of the intrinsic bronchodilation activity. Prospective controlled trials are needed to confirm this aspect.

Yours faithfully

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