


EFFECT OF NALOXONE ON LOSS OF CONSCIOUSNESS INDUCED BY I.V. KETAMINE

Sir,—It has been shown that ketamine interacts with opiate receptors, and that its agonistic action at these receptors is partially responsible for its analgesic effect (Smith et al., 1980). However, studies concerning the antagonism of ketamine-induced narcosis by naloxone have shown conflicting results. According to Kraynack and Gintautas (1982), the intraperitoneal injection of naloxone in rats did not alter ketamine sleeping time, whereas they observed a dose-related antagonism of ketamine narcosis by intra-cerebroventricular injection of naloxone. The data reported by Stella, Crescenti and Torri (1984) showing that ketamine anaesthesia is strongly antagonized by i.v. naloxone in man are thus very interesting.

We have tried to reproduce the results of Stella, Crescenti and Torri in 100 healthy young adult women. All were to undergo voluntary abortion, were unpremedicated and received an injection of naloxone 6 μg kg⁻¹ followed, 5 min later, by an i.v. injection of 0.404 mg kg⁻¹.

Our results differ completely from Stella's as 52% of our patients lost consciousness; these results prove that i.v. low doses of naloxone do not prevent ketamine-induced narcosis, since ketamine 0.404 mg kg⁻¹ is its ED₅₀.

These data seem to confirm that the hypnotic activity of ketamine is not exclusively mediated through opiate receptors, and that much higher doses of naloxone i.v. would be required to antagonize ketamine-induced narcosis.

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REFERENCES


Sir,—It is very difficult to say whether the results reported by Amiot and coworkers differ from ours (Stella, Crescenti and Torri, 1984), because of statistical differences between the two groups of patients.

First, we evaluated ED₅₀ for ketamine in a random sample of patients of both sexes, whose ages ranged from 20 to 50 yr. Amiot's patients were pregnant, premenopausal women.

Most likely, the ED₅₀ for ketamine may vary remarkably in these patients, compared with the values obtained in our studies. The dose of 0.404 mg kg⁻¹ cannot be regarded as ED₅₀ for his patients. In other words, it cannot be expected to cause loss of consciousness in nearly 50% of patients.

In summary, we have two dissimilar groups of patients coupled with two non-equipotent doses. Therefore, the two consciousness loss percentages are not comparable.

It would have been interesting to perform a comparison between placebo-treated patients of the same kind, in order to validate the percentage obtained in the naloxone-treated group. Despite to this, pregnancy-induced pharmacokinetic and pharmacodynamic interference between naloxone and ketamine cannot be ruled out.

It is difficult to state that ketamine induced loss of consciousness, since this drug provokes a peculiar state of dissociation. For this reason, during placebo comparisons, it is crucial to follow a double-blind experimental design, in order to minimize loss of consciousness evaluation errors, resulting from the subjectiveness of the clinician.

In our experience, other factors like injection speed and dilution volume also influence the results.

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REFERENCE


"ESOPHAGEAL GASTRIC TUBE AIRWAY" — A POTENTIAL HAZARD

Sir,—We were most interested to read the report of the use of the "Esophageal Gastric Tube Airway" (EGTA) in a case of failed intubation during obstetric anaesthesia (Tunstall and Geddes, 1984). We feel that this apparatus presents particular advantages in some cases of difficult intubation. Conversion of unsatisfactory extradural anaesthesia to general anaesthesia after the abdomen is opened is occasionally required. Where tracheal intubation proves impossible in this situation, rapid movement of the patient to the left lateral, head-down position as part of the "Failed Intubation Drill" (Tunstall, 1980) may not be possible.

We should, however, like to draw attention to a potential hazard associated with the use of the esophageal tube. There is a one-way valve situated within the lumen of the tube at the upper end which will prevent passage of gastric contents out of the tube. In use this valve may prevent spontaneous decompression of gastric pressure through the tube and predispose to esophageal regurgitation (since the esophagus is a distensible structure and the inflated esophageal balloon cannot guarantee occlusion). A second problem may arise should the esophageal tube happen to be passed into the trachea. Should this situation occur and be recognized, then it might be wished to ventilate the patient using the "Esophageal tube". When the valve becomes wet it will obstruct expiratory flow.

The EGTA was designed for use by paramedical personnel when ventilation is supplied by blowing into the mask (Gordon, 1977). This brings the face of the resuscitator close to the outlet