RESPIRATION I

A971

TITLE: INTRAVENOUS LIDOCAINE INHIBITS INDUCED LARYNGOSPASM IN DOGS DURING HALOTHANE ANESTHESIA

AUTHORS: P-O. Jarnberg, M.D., Ph.D.; P. Andersen, M.D.; J. Cohen, M.D., Ph.D.; M. Jamond, M.D.; J. Smith, M.D.

AFFILIATION: Depts. of Anesthesiology and Otolaryngology, Head and Neck Surgery, Oregon Health Sciences University, Portland OR 97201

Intravenous administration of lidocaine has been proposed as a method to prevent the occurrence of laryngospasm.[1] Clinical studies regarding its effectiveness are contradictory[2,3,4]. Part of this controversy may stem from the difficulty in correctly identifying and quantifying laryngospasm in the clinical setting. A recently described canine model allows visualization of the larynx and measurement of the duration and the strength of laryngospasms.[5] Using a modification of this model we designed a study to test the hypothesis that i.v. lidocaine is effective in preventing laryngospasm.

Material and Methods: 10 mongrel dogs were anesthetized with i.v. thiopental and maintained with halothane in O_2 administered via a tracheostomy tube. End tidal halothane and CO_2 concentrations were measured with a mass spectrometer. The larynx was visualized with the aid of a large speculum inserted into the dog's mouth. Intraglottic pressure (IGP) was measured from a balloon catheter connected to a pressure transducer and positioned between the vocal processes. A femoral arterial catheter was used for blood pressure measurements and blood sampling. Laryngospasm, visually observed, was induced by spraying the cords with 2.5 cc of 0.1 M, NH_4OH at an anesthetic level defined by the appearance of a brisk corneal reflex, with the animal remaining immobile. Dogs 1-2 were used to verify that repeated, reproducible laryngospasms could be induced. In dogs 3-10 a control laryngospasm was first induced. After one hour, 1 to 4 mg/kg of i.v. lidocaine was administered, and 2 minutes later an attempt to induce laryngospasm was made. A simultaneous blood sample for determination of serum lidocaine level was drawn. Attempts to induce laryngospasm were repeated hourly as above until 2 consecutive successful attempts were obtained. Data analysis was conducted by dividing the trials by whether or not laryngospasm occurred. Student's t-test was used to compare end tidal CO_2 and halothane levels between the two groups. Chi-square analysis was used to compare frequencies of laryngospasm with and without treatment with lidocaine. $p < 0.05$ was regarded as significant.

Results: 53 attempts to induce laryngospasm were made. Irrespective of serum lidocaine level, 51% produced laryngospasm, while 49% resulted in no laryngospasm. There were no significant differences in end tidal CO_2 or halothane concentrations between the two groups. The mean increase in IGP during laryngospasm was 45 $\pm$ 15 mmHg and the mean duration was 61 $\pm$ 19 sec. Treatment with i.v. lidocaine produced a significantly decreased rate of laryngospasm at serum lidocaine levels equal to or above 0.4 μg/ml ($p < 0.01$).

Conclusion: Intravenously administered lidocaine significantly lowers the incidence of induced laryngospasm in a canine model. A serum lidocaine level of 0.4 μg/ml appears to be the lower limit of the therapeutic range, although further study is necessary to construct a dose-response curve. This model seems to be a viable method for evaluation of the influence of drugs on the development of laryngospasm.

References

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A972

TITLE: LACK OF BRONCHODILATOR EFFECT OF SUBANESTHETIC CONCENTRATION OF ISOFLURANE IN ASTHMA

AUTHORS: F. Mercier(§), M.D., D. Benhamou (§), M.D., A. Denjean (¶), M.D.

AFFILIATION : Département d'Anesthésie (§) et Laboratoire d'Explorations Fonctionnelles (¶), Hopital Antoine Béclère, 157, rue de la Porte de Trivaux, 92141, CLAMART-FRANCE

Bronchodilating effect of halogenated anesthetics has been demonstrated in both experimental (1) and clinical conditions (2). However using subanesthetic concentrations of halothane and isoflurane, we failed to demonstrate any bronchodilation in severe chronic asthma (3). The aim of this study was to investigate the effect of subanesthetic concentration of isoflurane to reverse methacholine induced bronchoconstriction in patients with mild asthma.

6 patients with mild asthma (2 men, 4 women, mean age : 24.8 $\pm$ 4.4 y.o.) gave informed consent for the study. Each subject was studied twice (cross over) after a six hours fast, at the same time of the day one week apart, any medication being stopped 24h prior to each investigation. Baseline flow-volume curves (body plethysmograph: PULMED 3303-IMF Marseille-France, pneumotachograph FLEISCH #3) were obtained in triplicate and a cumulative dose-response curve to aerosolized methacholine (Mefar Dosimeter, Brescia-Italy) was performed. After the completion of the dose-response curve, the subjects were submitted to inhalation via a face mask of either isoflurane (0.75% in oxygen) or oxygen in a randomized order, during 8 min, in a supine position. Level of consciousness, arterial blood pressure, heart rate and arterial oxygenation were monitored as for routine general anesthesia. Then flow-volume curves were repeated at 14, 17, 20, 25 and 30 min after the end of methacholine inhalation. Data (mean±sd) were analyzed by a two-way analysis of variance and t test when necessary.

Baseline FEV_1 were near normal values on each experimental day (89±5% vs 90±5% of predicted values). Methacholine inhalation induced a similar bronchoconstriction on both days (figure 1) : FEV_1 decreased to 64.5±4 % of baseline value before oxygen inhalation (O_2) and to 62.5±3 % before isoflurane. During isoflurane inhalation, patients were sleepy and rapidly arousable. A progressive improvement of FEV_1 with time was observed, but there was no statistical difference between experimental days (figure 1).

In humans with mild asthma, subanesthetic concentration of isoflurane does not produce any significant improvement as compared with spontaneous recovery after methacholine-induced bronchoconstriction. Thus, isoflurane is not a powerful bronchodilator at low concentration.

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