

firmed that the balance of autonomic control is more important than the absolute level of sympathetic or parasympathetic activity.

Our patient may have had either psychologically or mechanically induced autonomic nervous system activity that predisposed the ventricle to tachycardia. Pain and anxiety could have caused increased sympathetic activity, which transiently increased vulnerability to cardiac arrhythmia. Extubation of the trachea may have been provided the stimulation necessary for an immediate shift in autonomic balance in control of the heart and terminated the arrhythmia. Removal of the endotracheal tube could have stimulated tracheal, laryngeal, and pharyngeal receptors, producing a burst of parasympathetic afferent activity.¹³ Reflex efferent parasympathetic discharge¹⁴ then might have counteracted the relative sympathetic overactivity and restored normal cardiac rhythm. Besides the burst of parasympathetic activity caused by the removal of the endotracheal tube, the psychological relief obtained may have reduced the level of sympathetic activity. The ventricular tachycardia was terminated immediately with extubation of the trachea, and normal cardiac rhythm was maintained without the aid of the usual exogenous pharmacologic agents.

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End-tidal Enflurane Concentration for Endotracheal Intubation

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Inhalational induction followed by endotracheal intubation is a technique frequently employed in pediatric anesthesia. In an earlier study¹ we introduced the term MAC_{EI}, defined as the end-tidal concentration of a gas or vapor needed by 50 per cent of the population to prevent all movement both during and immediately after laryngoscopy and endotracheal intubation. MAC_{EI} for halothane was calculated to be 1.33 per cent at sea level. In the present report,

similar methods were employed to determine MAC_{EI} for enflurane in pediatric patients.

METHODS

Twenty-four studies were performed in 22 ASA I surgical patients, aged 2 to 6 years. Informed consent regarding the nature and risks of the study was obtained from the parent or guardian of each participant. Premedication consisted of atropine, 0.015 mg/kg. A precordial stethoscope was used to monitor heart and breath sounds. Blood pressure was measured indirectly, and lead II of the electrocardiogram was continuously displayed. Body temperature was monitored with a rectal thermistor. Induction of anesthesia was accomplished with enflurane, 4-5 per cent, and oxygen (5 l/min) delivered from an Enfluromatic vaporizer through a Jackson-Rees modification of an Ayres

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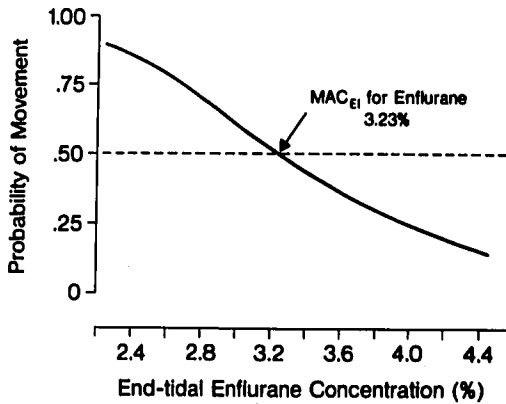


FIG. 1. Dose-response curve for enflurane plotted from logit analyses of individual end-tidal concentrations and the respective reactions to intubation. Corrected to barometric conditions at sea level, MAC_{EI} for enflurane is 2.93 per cent.

T-piece. The accuracy of gas concentrations produced by the vaporizer had been previously verified by calibration with a Varian 1400 gas chromatograph. Inspired and expired enflurane and carbon dioxide concentrations were continuously monitored with a Beckman LB II infrared gas analyzer, sampling at a rate of 200 ml/min. The infrared analyzer was calibrated with the vaporizer before each trial.

Enflurane concentrations at three sites were recorded. First, the inspired enflurane concentration was measured at the fresh-gas outflow port of the vaporizer. Next, an estimate of the end-tidal concentration was obtained by sampling at a point in the delivery system 2 cm proximal to the inflow of fresh gas. Last, in the patient whose trachea was successfully intubated, the end-tidal enflurane concentration was corroborated by gas analysis through a catheter inserted to 2 cm from the distal end of the endotracheal tube. There was no significant difference between samples obtained from the latter two sites. Therefore, when conditions were such that an endotracheal tube could not be inserted, the externally measured value for end-tidal enflurane concentration was used to determine MAC_{EI} .

TABLE 1. Enflurane Concentrations and Percentages of Patients Moving upon Intubation

| Number of trials | Enflurane Concentration (Per Cent) | | Patients Moving (Per Cent) |
|------------------|------------------------------------|-----------------------------|----------------------------|
| | Inspired* Mean \pm SD | End-tidal† Mean \pm SD | |
| 2 | 2.06 \pm .01 | 2.03 \pm .03 | 100 |
| 5 | 2.55 \pm .09 | 2.52 \pm .07 | 100 |
| 9 | 3.01 \pm .03 | 2.95 \pm .03 | 56 |
| 8 | 3.57 \pm .04 | 3.53 \pm .05 | 38 |

* Measured in nonbreathing apparatus.

† Measured through catheter in endotracheal tube.

Four end-tidal enflurane concentrations were studied: 2.07, 2.48, 2.98, and 3.55 per cent. Two patients were tested with more than one concentration. However, no patient was exposed to more than two test concentrations during the same procedure, and none of the test concentrations was administered more than once to the same patient. After induction, the randomly-selected end-tidal concentration was approached by slowly decreasing the inspired concentration. Spontaneous respirations were manually assisted. The estimated end-tidal concentration was established at the desired value (± 0.05 per cent), and maintained for 10 min to allow equilibration of cerebral and arterial blood gas tensions. Endotracheal intubation was then attempted. The process of intubation was evaluated according to the adequacy of conditions for laryngoscopy (easy visualization of the glottis, relaxation of the vocal cords, and absence of extremity movement) and the incidence of coughing or "bucking" immediately after an otherwise successful intubation. Logit analyses of responses to intubation were performed. In this manner the MAC_{EI} for enflurane was derived.

RESULTS

Table 1 shows mean values, at each sampling site, for the end-tidal concentrations tested. The response curve constructed on the basis of logit analyses of data in this patient population (fig. 1) revealed that MAC_{EI} is 3.23 per cent. Based on the slope equation, MAC_{EI} for 95 per cent of this population equals 4.50 per cent. These investigations were performed at an altitude of approximately 760 m (2,500 ft), where the barometric pressure is about 700 torr. Traditional MAC values have been determined at essentially sea level. After appropriate barometric corrections, MAC_{EI} at sea level is calculated to be 2.93 per cent.

DISCUSSION

Attempting endotracheal intubation without muscle relaxant and in the presence of an insufficient depth of anesthesia is hazardous. Inhalational induction with subsequent intubation is frequently performed in pediatric anesthesia. Enflurane has been recommended for this surgical population due to the rapidity of induction and emergence.² In children, MAC for enflurane has been estimated in the range of 2.0 to 2.2 per cent.^{3,†} These estimates are based on the assumption that an age-MAC relationship for en-

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flurane exists and is similar to that for halothane. At sea level MAC_{EI} values for enflurane and halothane are 2.93 and 1.33 per cent, respectively. In both instances, MAC_{EI} values appear to be about 30 per cent greater than MACs. It is not known whether similar relationships obtain with other volatile anesthetics. Of additional interest is that a plotting of log dose-response curves for enflurane and halothane (fig. 2) suggests a striking parallelism. This would indicate that, in regard to anesthetizing the upper airway, the potency differential between the two agents is fairly uniform over a wide range of possible responses.

Contrary to the experience of others,² inhalational induction to end-tidal enflurane concentrations of 2.9 per cent or more required a considerable period (15 to 20 min). This may be explained by the fact that, relative to MAC_{EI} , the inspired concentration of enflurane administered during induction (4–5 per cent) in this study was proportionately less than that delivered for induction with halothane (2–3 per cent) in the previous study. If the relationship between inspired concentration and MAC_{EI} for enflurane had been of the same magnitude as that which existed with halothane, enflurane administration would probably have resulted in a more rapid rate of anesthetic induction. Other possible explanations for prolonged induction are that we avoided hyperventilation, which would hasten establishment of a prescribed end-tidal concentration, and we strove to achieve an F_A/F_I ratio near unity.

Enflurane is known to produce central nervous system excitation beginning at an alveolar concentration of 2.5 per cent.⁴ This cerebral activity may be manifested by muscular movements. We observed such movements (primarily tonic-clonic twitching of hands and feet) in 15 of the 17 patients studied with end-tidal enflurane concentrations of 2.9 per cent or more. In six of these 15 patients, obvious muscular movement was accompanied by an apparent decrease in chest-wall compliance with concomitant difficulty in assisting respirations. Conceivably, this resistance may have been due to increased tonus in chest or upper airway musculature. The condition was effectively treated by lowering the inspired enflurane concentration. Such reactions at higher enflurane con-

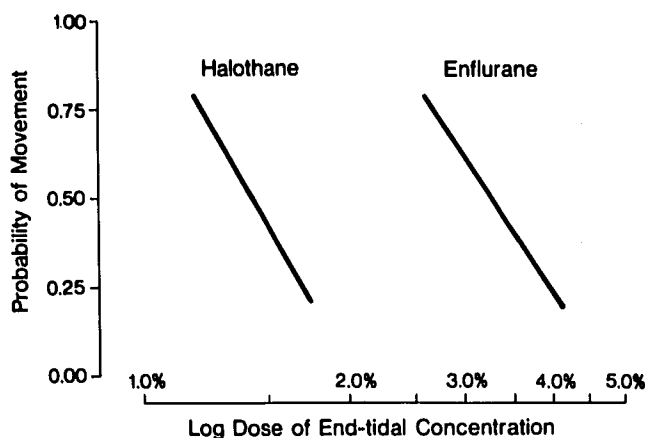


FIG. 2. Comparative plots of log dose-response curves for halothane and enflurane. Corrected to barometric conditions at sea level, MAC_{EI} values for halothane and enflurane are 1.33 and 2.93 per cent, respectively.

centrations dissuaded us from attempting to test an end-tidal concentration at which 100 per cent of the population would be sufficiently anesthetized for intubation.

Theoretically, anesthetic induction with a combination of enflurane and nitrous oxide would obviate the need for the high concentrations of enflurane employed in this study. In comparison, halothane may be used for the same purpose accompanied by oxygen alone. Also, hyperventilation may be used with halothane to hasten induction, and the potential hazard of central nervous system excitation is eliminated. For these reasons, halothane appears to be more suitable for children when intubation without the use of muscle relaxants is planned.

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