CARDIOVASCULAR RESPONSES TO TRACHEAL INTUBATION IN SMALL CHILDREN

Effects of the Induction of Anaesthesia with Halothane

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The i.v. induction of anaesthesia is followed by circulatory responses to tracheal intubation such as increases in arterial pressure, heart rate and the rate-pressureproduct (Kautto, 1981), prolongation of the Q-T interval on the electrocardiogram and cardiac arrhythmias, in both children and adults (Saarnivaara and Lindgren, 1983). Therefore, from a cardiovascular point of view, the i.v. induction of anaesthesia may be less than ideal. A previous study noted that the circulatory responses to tracheal intubation after an inhalation induction (enflurane followed by suxamethonium) were considerably attenuated when compared with those associated with an i.v. induction with thiopentone and suxamethonium (Lindgren and Saarnivaara, 1983).

Although halothane is used commonly for the induction of anaesthesia in small children, and provides ideal intubating conditions without the use of suxamethonium (Yakaitis, Blitt and Angiulo, 1977), there are no studies on the cardiovascular responses to tracheal intubation when halothane has been used to induce anaesthesia in children.

The present work was designed to study inhalation induction of anaesthesia with halothane in small children, with special reference to cardiovascular responses to tracheal intubation.

PATIENTS AND METHODS

Twenty-seven children were studied (table I). They were premedicated with triclofos 70 mg kg⁻¹ and atropine 0.03 mg kg⁻¹ by mouth, approximately 90 min before the induction of anaesthesia.

<table>
<thead>
<tr>
<th>Male/Female</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Haemoglobin concn (g litre⁻¹)</th>
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<tbody>
<tr>
<td>17/10</td>
<td>1.5 ± 0.6</td>
<td>11.2 ± 1.9</td>
<td>125 ± 7.6</td>
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Halothane 2.5% was administered with 70% nitrous oxide in oxygen, a Rees modification of the Ayre’s T-piece with a face mask being used. Twice the average value for minute ventilation was used as listed in Documenta Geigy Scientific Tables (Ciba–Geigy Ltd, 1975) as fresh gas flow. The times from the start of anaesthesia to the disappearance of eyelash reflex and to tracheal intubation were recorded with a stop-watch. The clinical signs of adequate depth of anaesthesia for
tracheal intubation were: fixed and small pupils, relaxation of the jaw and regular, free diaphragmatic breathing. Intubating conditions were classified as *good* if the vocal cords were open and motionless, *acceptable* if slightly moving and *unsatisfactory* if the child was coughing after intubation (table II). Systolic and diastolic arterial pressures were measured indirectly (Riva-Rocci method) before induction, and immediately before and after tracheal intubation as well as 5 min after intubation. Heart rate and the ECG (lead AVR) with three surface electrodes were displayed continuously on an oscilloscope and recorded at a paper speed of 25 mm s⁻¹. Monitoring was started 1 min before induction. The Q–T interval was measured before induction, immediately before and after, and 5 min after, tracheal intubation. The Q–T interval was measured from the onset of the QRS-complex to the end of the T-wave. On each occasion the mean Q–T interval from four successive beats was determined. Heart rate correction was made according to the formula: \( Q-T_c = Q-T/\sqrt{RR} \). Values less than 440 ms were considered to be normal (Bazett, 1920). At the beginning of the operation, the otolaryngologist classified the antisialagogue effect as *satisfactory* if the oropharynx was dry or moist and as *unsatisfactory* if it was wet or very wet (Boyd and Manford, 1973). All the children were anaesthetized by the same author (L.S.)

Student’s *t* test was used for the statistical analysis of the results. All data are given as mean ± SEM.

**RESULTS**

The eyelash reflex disappeared in 62 ± 3.6 s and tracheal intubation was possible 4.9 ± 0.2 min after the start of anaesthesia. Intubating conditions were good in 74%, acceptable in 11% and unsatisfactory in 15% of the children (table II). Immediately before intubation, the mean decreases in systolic and diastolic arterial pressures from the control values were 22 and 11 mm Hg, respectively. After tracheal intubation, the mean increases in systolic and diastolic arterial pressures from the values measured immediately before intubation were 11 and 6 mm Hg, respectively. The values measured 5 min after intubation did not differ

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**Table II. Intubating conditions after halothane induction.** Percentage numbers for 27 children. *Good = vocal cords open and motionless; acceptable = small movements in vocal cords; unsatisfactory = coughing after intubation.*

<table>
<thead>
<tr>
<th></th>
<th>Good</th>
<th>Acceptable</th>
<th>Unsatisfactory</th>
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<tbody>
<tr>
<td></td>
<td>74</td>
<td>11</td>
<td>15</td>
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</table>

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![Graph](https://example.com/graph.png)

**Fig. 1.** Systolic (■) and diastolic (□) arterial pressure during induction of anaesthesia with 2.5% halothane plus 70% nitrous oxide in oxygen. Mean values for 27 children. Bars indicate SEM. *P < 0.001: significant difference from the corresponding preceding arterial pressure.*
HALOTHANE INDUCTION IN CHILDREN

**FIG. 2.** Q-T intervals (ms) (□) and heart rate (beat min⁻¹) (□) during induction of anaesthesia. Mean values for 27 children: initial value; 4.9 ± 0.9 (SD) min after inhalation of 2.5% halothane with 70% nitrous oxide in oxygen (Halothane); after intubation (Intubation); 5 min after intubation. Significant differences from the preceding heart rate: ***P < 0.001; **P < 0.01; *P < 0.02.

significantly from the values obtained immediately after intubation (fig. 1). Values for arterial pressure from all the children were included in the results.

The antispasmodic effect was satisfactory in 87% and unsatisfactory in 13% of the children at the beginning of the operation. The mean preanaesthetic Q-T interval was 428 ± 5.3 ms. There were no significant changes in Q-T interval during the induction of anaesthesia. The mean control heart rate was 155 beat min⁻¹ before the start of anaesthesia and decreased significantly (P < 0.001) by a mean value of 26 beat min⁻¹ during the 4.9 ± 0.2 min of anaesthesia before tracheal intubation (fig. 2). Cardiac arrhythmias were not seen.

**DISCUSSION**

Inhalation induction of anaesthesia with halothane and 70% nitrous oxide in oxygen resulted in minimal cardiovascular responses to tracheal intubation. No cardiac arrhythmias were recorded at any time.

**Intubating conditions**

Tracheal intubation could be performed satisfactorily in 85% of the children. The MAC of halothane for endotracheal intubation in children aged 4 yr is 1.33% in oxygen (Yakaitis, Blitt and Angiulo, 1977). Since in the present study, halothane, was administered in 70% nitrous oxide in oxygen, the MAC for tracheal intubation was 0.42% (Saidman and Eger, 1964). Although the end-tidal concentration of halothane was not measured in the present study, end-tidal concentrations of 0.8% have been reported with inspired concentrations of 2% after 3 min administration of halothane to children (Lindgren, 1981a). In the present study, inspired 2.5% halothane with 70% nitrous oxide in oxygen was adequate for tracheal intubation within about 5 min.

**Cardiovascular responses**

The increase in systolic arterial pressure after intubation was within the same range as that seen with enflurane (Lindgren and Saarnivaara, 1983) and was less marked in the present study than noted previously by Saarnivaara (1977) and Lindgren, Saarnivaara and Himberg (1980) with thiopentone followed by suxamethonium. Laryngeal responses are not inhibited by barbiturates (Goodman and Gilman, 1980) and therefore halothane, like enflurane (Lindgren and Saarnivaara, 1983), seems to be preferable to thiopentone for laryngeal and tracheal manipulation.

The Q-T interval was not prolonged significantly during the induction of anaesthesia or the intubation of the trachea during halothane anaesthesia. Usually, the Q-T interval is shorter during the administration of halothane in children (Lindgren, 1981b). However, should the Q-T interval before anaesthesia be prolonged, serious ventricular arrhythmias may be associated with
the use of halothane during oral (Lindgren, 1981b) or general surgery (Wig et al., 1979). Since such prolongation of the Q-T interval can be reversed when the ventricular arrhythmia is treated with lignocaine (Lindgren, 1981b), we believe that prolongation of the Q-T interval, in association with the use of halothane, is a predictor of ventricular arrhythmia.

There is evidence that the Q-T interval is prolonged in the presence of high serum concentrations of catecholamines. Prolongation of the Q-T interval during a hypertensive period has been found in patients with phaeochromocytoma (Cheng and Bashour, 1976); increased serum noradrenaline concentrations have been demonstrated during manipulation of this tumour (Takki et al., 1972). Prolongation of the Q-T interval has been found after the injection of noradrenaline to dogs (Abildskov, 1976). Tracheal intubation also causes a sympathoadrenal stimulus (Tomori and Widdicombe, 1969), and increased arterial noradrenaline concentrations and increases in arterial pressure have been observed after intubation in adults (Russel et al., 1981). Therefore, tracheal intubation may cause prolongation of the Q-T interval associated with high serum catecholamine concentrations. Cardiac arrhythmias occur frequently after tracheal intubation in children (Saarnivaara and Lindgren, 1983). Halothane sensitizes the heart to the arrhythmogenic effect of catecholamines (Katz and Katz, 1966). Therefore, arrhythmias might be expected to develop after tracheal intubation during halothane anaesthesia.

In the present study, however, both the increase in arterial pressure and changes in Q-T interval after tracheal intubation were minimal and no cardiac arrhythmias were noted. This result may reflect the fact that halothane itself inhibits the burst of efferent sympathetic activity (Deutsch et al., 1962) caused by tracheal intubation. Therefore, halothane here may prevent the prolongation of the Q-T interval, the increase in arterial pressure and cardiac arrhythmia. On the other hand, after manipulations like dissection of the tonsils, sympathetic activity may increase by a different mechanism, resulting in prolongation of the Q-T interval and cardiac arrhythmia in the presence of halothane (Lindgren, 1981b). Consequently, tracheal intubation may not be comparable to tonsillectomy in regard to stimulation of sympathetic activity.

The present results confirm the earlier findings that halothane markedly inhibits laryngeal reflexes and facilitates tracheal intubation (Price, 1975; Yakaitis, Blitt and Angiulo, 1977). The heart rate decreased significantly during the induction of anaesthesia in our study. This may have been the result of the inhibition of the firing rate of the sinoatrial node caused by halothane (Flacke and Alper, 1962; Hauswirth and Schaer, 1967).

Thus, the induction of anaesthesia with halothane in small children provided adequate anaesthesia for tracheal intubation with minimal cardiovascular response, stabilization of Q-T interval and normal cardiac rhythm.

ACKNOWLEDGEMENT

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REFERENCES


