E.C.G. CHANGES DURING HALOTHANE AND ENFLURANE ANAESTHESIA FOR E.N.T. SURGERY IN CHILDREN

L. LINDGREN

SUMMARY
E.c.g. changes were compared in 152 children undergoing adenoidectomy or adenotonsillectomy (T + A) under halothane or enflurane anaesthesia. Junctional rhythm occurred in 4-16% of the children in adenoidectomy groups and in 11-33% in T + A groups. Bundle branch block occurred in 4% of the children anaesthetized with halothane, but not with enflurane and was particularly common in association with thiopentone and T + A operations; one patient had bifocal ventricular tachycardia. QT interval was prolonged compared with control after thiopentone \( (P<0.001) \) and thiopentone and suxamethonium \( (P<0.02) \). QT interval was not changed after Althesin with or without suxamethonium. Mean preanaesthetic QT interval \( (\pm\) SEM) was significantly prolonged \( (492 \pm 22\, \text{ms}; \text{normal} \, 440\, \text{ms}) \) in children showing aberrant conduction with chaotic rhythm, but normal \( (438 \pm 5\, \text{ms}) \) when bundle branch block or junctional rhythm was present during halothane anaesthesia. QT interval was prolonged significantly in enflurane but not in halothane anaesthesia.

Oral surgery under general anaesthesia may provoke serious cardiac arrhythmia (Kaufman, 1965; Tuohy, 1968), often initiated as a reflex caused by the stimulation of the pharynx and trachea (Katz and Bigger, 1970). Halothane is associated with a high frequency of arrhythmia during oral surgery (Tuohy, 1968; Fisch et al., 1969; Saarnivaara et al., 1974; Gotta et al., 1976). Atlee and Rusy (1972, 1977) have shown that halothane prolongs A-V nodal, His–Purkinje and ventricular conduction, whereas enflurane only prolongs A-V nodal conduction in dogs. They concluded that conduction changes such as occur with halothane are necessary for ventricular arrhythmias caused by re-entry of excitation and that enflurane is less likely to cause these arrhythmias.

Thiopentone appears to increase aberrant ventricular conduction in patients with Wolf–Parkinson–White syndrome (Kadis and Gianelly, 1973). Cundy (1973) has reported a case in which the administration of Althesin effectively abolished ventricular ectopic beats. Alexander (1974) and Saarnivaara and Kentala (1977) found that Althesin protected against ventricular arrhythmia during oral surgery.

Prolongation of the QT interval in e.c.g. is a sign of imbalance in cardiac sympathetic activity (Schwartz, Stone and Brown, 1976). This is associated with ventricular arrhythmia in acute myocardial infarction (Ahnve, Lundman and Shoaheh-var, 1978) and is a predictor of sudden death in this disease (Schwartz and Wolf, 1978). Wig and others (1979) have described sudden cardiac arrest in a patient with a prolonged QT interval during halothane anaesthesia. Kentala and Repo (1979) found a prolonged QT interval during exercise testing to be of prognostic value in relation to survival after acute myocardial infarction.

The present study was designed to compare the frequency of cardiac arrhythmia during halothane or enflurane anaesthesia, after induction with thiopentone or Althesin, in children undergoing e.n.t. surgery. The effect of anaesthetics on QT intervals was studied and the changes in QT intervals in association with cardiac arrhythmia were evaluated.

PATIENTS AND METHODS
One hundred and fifty-two unselected children, with no cardiac disease, undergoing adenoidectomy or adenotonsillectomy (T+A) were studied (table I).

Anaesthesia
Premedication was triclofos 70 mg kg\(^{-1}\) and atropine 0.03 mg kg\(^{-1}\) given orally about 90 min before the start of anaesthesia.

A needle was inserted to the left cubital vein. In the adenoidectomy group, 45 children were anaes-
thetized with halothane (induction with thiopentone 5 mg kg\(^{-1}\) in 20, Althesin 0.06 ml kg\(^{-1}\) in 25) and 53 with enflurane (25 and 28). In the T + A group, 27 children were anaesthetized with halothane and 27 with enflurane, in either case following thiopentone 5 mg kg\(^{-1}\) and suxamethonium 1.5 mg kg\(^{-1}\) i.v. The trachea was intubated. Mean delivered concentration of halothane was 1.1 (v/v) and of enflurane 2.0. Both anaesthetics were administered in 70% nitrous oxide in oxygen. A Rees system was used for children weighing less than 25 kg and a circle system with carbon dioxide absorption for the remainder. Ventilation of the lungs was usually controlled, but in a few patients it was assisted. End-tidal carbon dioxide concentration was kept at 5.5-6%. All the children were anaesthetized by the author.

The mean duration (± SD) of anaesthesia for adenoidectomy was 31 ± 7 min and that of the operation 20 ± 7 min. The corresponding figures for T + A were 35 ± 9 min and 24 ± 8 min, respectively.

**Assessment of e.c.g. changes and QT interval**

Heart rate and the e.c.g lead-AVR with three surface electrodes were displayed continuously on an oscilloscope (Mode 280, Kone Osakeyhtio, Espoo, Finland) and the e.c.g. was recorded. Monitoring was started 1 min before the insertion of an i.v. cannula and discontinued 2 min after extubation.

The e.c.g recordings were analysed with special reference to bundle branch block and aberrant conduction. Junctional rhythm was classified according to the following system. If the P wave preceded the QRS complex and PQ interval was shorter than 0.12 s, the junctional rhythm was considered to be upper junctional. If the P wave was hidden within the QRS complex or if it followed the QRS complex, then the junctional rhythm was considered to be middle or lower junctional, respectively (Saarnivaara and Kentala, 1977). E.c.g. changes occurring before the start of inhalation anaesthesia are excluded from this study.

QT intervals were measured before the induction of anaesthesia, after laryngoscopy, after intubation, 3 min after the start of the inhalation anaesthesia, after adenoidectomy or T + A and after extubation. From the e.c.g. (paper speed 25 mm s\(^{-1}\)) the QT interval was measured from the onset of QRS complex to the end of the T wave. The mean of four successive beats was determined. Rate correction was made according to the formula: QT\(_c\) = QT/\(\sqrt{R-R'}\) (Bazett, 1920). QT values exceeding 440 ms were defined as prolonged.

To analyse the effects of the induction agents on QT interval, QT intervals were measured immediately before and after venepuncture, 45 s after the i.v. anaesthetic, 30 s after suxamethonium, after laryngoscopy and after intubation. Mean age, weight and preanaesthetic QT interval (± SD) of the children in the thiopentone group were 5.3 ± 0.5 yr, 18.8 ± 1.3 kg and 432 ± 4 ms and in the Althesin group 4.6 ± 0.6 yr, 18.5 ± 1.4 kg and 423 ± 4 ms.

Student's \(t\) tests for paired and unpaired data were used for the statistical analysis of the results.

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**Table 1. Sex, age, weight and haemoglobin concentration in different treatment groups.**

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Haemoglobin (g litre(^{-1}))</th>
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</thead>
<tbody>
<tr>
<td><strong>Halothane</strong></td>
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<tr>
<td>Adenoidectomy</td>
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<tr>
<td>Thiopentone (20)</td>
<td>14</td>
<td>4.1 ± 2.6</td>
<td>17.7 ± 7.6</td>
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</tr>
<tr>
<td>Althesin (25)</td>
<td>14</td>
<td>4.7 ± 2.3</td>
<td>17.5 ± 4.7</td>
<td>136 ± 9.0</td>
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<tr>
<td>T + A</td>
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<tr>
<td>Thiopentone (27)</td>
<td>17</td>
<td>7.2 ± 2.8</td>
<td>25.6 ± 9.6</td>
<td>124 ± 8.0</td>
</tr>
<tr>
<td><strong>Enflurane</strong></td>
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<tr>
<td>Thiopentone (25)</td>
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<td>4.3 ± 2.7</td>
<td>18.2 ± 6.5</td>
<td>135 ± 9.4</td>
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<tr>
<td>Althesin (27)</td>
<td>14</td>
<td>4.9 ± 2.5</td>
<td>18.5 ± 5.8</td>
<td>132 ± 7.0</td>
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<tr>
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<tr>
<td>Thiopentone (27)</td>
<td>15</td>
<td>6.4 ± 2.7</td>
<td>22.7 ± 8.1</td>
<td>127 ± 8.2</td>
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RESULTS
The mean preanaesthetic QT intervals (± SD) ranged from 425 ± 39 to 447 ± 37 ms in different groups.

There was no disturbance in the e.c.g. 1 min before venepuncture. Table II shows that junctional rhythm occurred in 4–16% of the children in the adenoidectomy groups. In the T + A groups, the frequency of junctional rhythm ranged from 11% to 33%. The frequency of upper and middle junctional rhythms was similar in each group.

<table>
<thead>
<tr>
<th></th>
<th>Junctional rhythm</th>
<th>QRS complex changes</th>
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<tr>
<td></td>
<td>Upper</td>
<td>Middle</td>
<td>BBB</td>
<td>BBB + aberrant conduction</td>
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<tr>
<td>Halothane</td>
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<td>Adenoidectomy</td>
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<tr>
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<td>0</td>
<td>10</td>
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<tr>
<td>Althesin (25)</td>
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<td>16</td>
<td>4</td>
<td>4</td>
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<td>T + A</td>
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<tr>
<td>Thiopentone (27)</td>
<td>30</td>
<td>33</td>
<td>4</td>
<td>11</td>
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<td>12</td>
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<td>0</td>
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<tr>
<td>Althesin (27)</td>
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<td>7</td>
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<td>T + A</td>
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<tr>
<td>Thiopentone (27)</td>
<td>11</td>
<td>15</td>
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Neither bundle branch block (BBB) nor BBB plus aberrant conduction was seen during enflurane anaesthesia, whereas the frequency of BBB ranged from 0% to 4% and BBB plus aberrant conduction was seen in six children in the halothane groups. Aberrant conduction was always associated with the surgical manipulation. In addition to the results shown in table II, one child in the Althesin plus halothane group developed BBB with aberrant conduction before the start of spontaneous breathing when the end-tidal carbon dioxide was 9.5%, but this disappeared after breathing 100% oxygen. In three children BBB and aberrant conduction disappeared without treatment. In the remaining three children, e.g. became chaotic and this lasted more than 5 min. Administration of lignocaine 1 mg kg\(^{-1}\) i.v. promptly abolished the arrhythmia. The mean duration (± SEM) of QT interval during BBB with aberrant conduction was 477 ± 14 ms. In the children treated with lignocaine the mean pretreatment (± SEM) QT interval was 509 ± 24 ms and decreased significantly after lignocaine, followed by sinus rhythm (437 ± 9.7 ms) (P < 0.05).

The T wave became biphasic in 15–41% of the children anaesthetized with enflurane and in 0–8% anaesthetized with halothane. A typical example of T wave becoming biphasic is shown in figure 1. The arterial pressure was maintained during all types of e.c.g. changes.

Figure 2 shows that the mean preanaesthetic QT interval was significantly longer (492 ± 22 ms) in the children showing BBB plus aberrant conduction than in the children without e.g. changes (426 ± 5.2 ms) (P < 0.01) and in the children with junctional rhythm or BBB (438 ± 5.3 ms) (P < 0.05). Figure 3 shows the e.g. changes of a healthy 9-yr-old boy undergoing T + A with halothane anaesthesia. Preanaesthetic QT interval was 594 ms. During the dissection of the left tonsil there was aberrant conduction with chaotic rhythm which resulted in bifocal ventricular tachycardia. A regular supraventricular rhythm was restored after injection of lignocaine 1 mg kg\(^{-1}\) i.v. and sinus rhythm followed. This boy’s QT interval was checked 1 year later after a normal school day and it was 421 ms. Figure 4A shows the e.g. changes of a healthy 4-yr-old girl undergoing adenoidectomy with Althesin plus halothane anaesthesia. Preanaesthetic QT interval...
Fig. 1. E.c.g. of a healthy 3-yr-old girl undergoing adenoidectomy under thiopentone and enflurane. Typical change in T wave during enflurane is seen. 1 = Preanaesthetic e.c.g. with QT 393 ms. 2 = 3 min after enflurane; QT 439 ms and T wave notched. 3 = After adenoidectomy; QT 445 ms and T biphasic. 4 = After extubation; QT 432 ms and T wave notched. Speed of paper 25 mm s⁻¹.

Fig. 2. Preanaesthetic QT intervals in groups anaesthetized with halothane: I = all the children; II = children with no arrhythmias; III = children with junctional rhythm or bundle branch block (BBB); IV = children having BBB with aberrant conduction. Number in parentheses. Bars indicate SEM. * = P < 0.01 from group II. ● = P < 0.05 from group III.

Fig. 3. E.c.g. changes of a healthy 9-yr-old boy undergoing T + A under thiopentone and halothane. 1 = Preanaesthetic e.c.g. with QT 594 ms. 2 = Dissection of the left tonsil. QT 560 ms. Aberrant conduction with chaotic rhythm. 3 = Twenty seconds later; bifocal ventricular tachycardia. Arrow indicates lignocaine 1 mg kg⁻¹. Arterial pressure 120/80 mmHg. QT 557 ms. 4 = Ten seconds after administration of lignocaine; supraventricular regular rhythm with QT 456 ms. 5 = Sinus rhythm after extubation. QT 461 ms. Speed of paper 25 mm s⁻¹.

was 402 ms. After insertion of the mouth gag there was BBB, which disappeared when the concentration of halothane was reduced. Figure 4B shows the e.c.g. changes in a healthy 5-yr-old girl undergoing adenoidectomy with thiopentone plus halothane anaesthesia. Preanaesthetic QT interval was 465 ms. Pressure on the adenoidectomy bed caused BBB plus aberrant conduction which dis-
appeared after cessation of the surgical manipulation.

QT interval was significantly longer 45 s after thiopentone (449 ± 4 ms) than after venepuncture (430 ± 4 ms); (P< 0.001) (fig. 5) and was further prolonged 30 s after suxamethonium (P<0.02). It remained unchanged 45 s after Althesin compared with after venepuncture. Suxamethonium after Althesin did not prolong QT. In the thiopentone and Althesin groups, the QT intervals during laryngoscopy and intubation remained the same as after suxamethonium.

QT interval after inhalation of halothane for 3 min, after tonsillectomy and after extubation did not differ significantly from the value after intubation (459 ± 8.4 ms), whereas after inhalation of enflurane for 3 min QT was significantly prolonged (471 ± 4 ms) (P<0.05) and remained so until extubation (fig. 6). QT intervals during enflurane anaesthesia were significantly longer than the corresponding values during halothane anaesthesia.

QT interval after inhalation of halothane for 3 min did not differ significantly from the values after intubation in either the thiopentone (473 ± 15 ms) or the Althesin group (429 ± 7 ms).

After inhalation of halothane for 3 min QT was significantly longer in the thiopentone (457 ± 9 ms) compared with the Althesin group (427 ± 5.6 ms).
Junctional rhythm occurred in association with both inhalation anaesthetics, but BBB and BBB plus aberrant conduction only with halothane. The preanaesthetic QT interval was significantly prolonged in the children developing BBB plus aberrant conduction. QT was significantly longer after thiopentone plus suxamethonium than after Althesin plus suxamethonium. QT was prolonged significantly during enflurane anaesthesia, but not during halothane anaesthesia.

**Functional rhythm**

Junctional rhythm occurred in 4–33% in the different anaesthetic groups. These results with halothane are in agreement with those of Alexander (1971), Alexander, Bekheit and Fletcher (1972), Alexander (1974) and Saarnivaara and Kentala (1977), who found junctional rhythm in 33–51% of adults during oral surgery. Halothane has a tendency to cause junctional rhythm (Johnstone, 1956) because it reduces the firing rate of the sino-atrial pacemaker (Flacke and Alper, 1962; Hauswirth and Schaer, 1967; Gersh and Prys-Roberts, 1972). Junctional rhythm occurred during enflurane anaesthesia as often as during halothane anaesthesia. The frequency of junctional rhythm during enflurane anaesthesia in the present study was in the range found by Söderberg and Grattidge (1975) in children. The mechanism by which enflurane causes junctional rhythm cannot be explained.

**QRS complex**

BBB was not seen during enflurane anaesthesia, but occurred in two children anaesthetized with halothane. In six children BBB was associated with aberrant conduction. In addition to junctional rhythm, the other typical e.g. changes caused by halothane are ventricular arrhythmia, especially in
the presence of respiratory acidosis, hypoxia or other causes of sympathetic stimulation such as nervousness before operation (Apivor, 1960) or surgical manipulation (Alexander, 1971; Alexander, Bekheit and Fletcher, 1972). In the present study, the concentration of oxygen was 30% and end-tidal carbon dioxide was 5.5–6%. Surgical stimulation may be a factor. This is supported by Alexander (1971) who found that 23% of his patients developed ectopic rhythms in response to oral surgery under halothane anaesthesia. Alexander, Bekheit and Fletcher (1972) concluded that afferent impulses caused by oral surgery may be mediated by trigeminal nerve endings and may stimulate nerve centres in the medulla where sympathetic impulses reaching the heart are initiated.

The reason for the occurrence of BBB or BBB with aberrant conduction during halothane, but not enflurane, anaesthesia may be explained by the study of Atlee and Rusy (1977) who found that enflurane, like halothane (Atlee and Rusy, 1972), prolongs A–V nodal but not His–Purkinje and ventricular conduction. Atlee and Rusy (1977) suggested that re-entry of excitation requires slowing of conduction, unidirectional block and a pathway for an entrant impulse to reach the conduction system before arrival of the next normal impulse from above. This may explain why, in our study, and in the study of Alexander and Murtagh (1979), all the patients who developed aberrant conduction also had BBB.

Alexander (1971) suggested that the origin of aberration is supraventricular because lignocaine was relatively ineffective in abolishing this arrhythmia. In the present study, however, lignocaine abolished aberrant conduction with chaotic rhythm in three instances. Since the study reported here, lignocaine has proved to be ineffective in two of 10 children with aberrant conduction during halothane anaesthesia. In the present study, aberrant conduction led to bifocal ventricular tachycardia in a 9-yr-old boy. This arrhythmia might have been fatal without treatment; Gotta and others (1976) have found that aberrant conduction is a precursor of serious cardiac arrhythmia during halothane anaesthesia for oral surgery.

The frequency of BBB plus aberrant conduction after thiopentone induction was twice that after Althesin. The antiarrhythmic effect of Althesin was noted by Cundy (1973), Alexander (1974) and Saarnivaara and Kentala (1977).

**QT interval in association with i.v. anaesthetics and suxamethonium**

Prolongation of QT interval may be congenital (Jervell and Lange-Nielsen, 1957; Romano, Genre and Pongiglione, 1963; Ward, 1964) or acquired by administration of quinidine, tricyclic antidepressants (Schwartz and Wolf, 1978) or noradrenaline (Abildskov, 1976). Tricyclic antidepressants cause accumulation of noradrenaline at extracellular sites (Carlsson et al., 1969). Prolongation of QT during a hypertensive period has been found in patients with phaeochromocytoma (Cheng and Bashour, 1976). These results give further evidence that, in the presence of increased amounts of catecholamines, QT is prolonged. Hypocalcaemia may prolong QT (Surawicz, 1964). The children in the present study were healthy and in electrolyte balance.

QT interval was prolonged significantly after thiopentone induction and further after suxamethonium given after thiopentone, whereas it remained unchanged after Althesin. Suxamethonium given after Althesin did not prolong QT. Thiopentone depresses activity in all excitable tissues and increases the plasma concentration of catecholamines (Goodman and Gilman, 1975). Takki and others (1972) could not find an increase in the catecholamines in association with intubation after thiopentone plus suxamethonium. In the present study, the reason for prolongation of QT after thiopentone is unexplained. Stimulation of sympathetic ganglia may be caused by suxamethonium (Gallindo and Davis, 1962; Stoner and Urbach, 1968). Kadis and Gianelly (1973) found that thiopentone caused further aberration in patients with Wolf–Parkinson–White syndrome and Suppan (1979) has anaesthetized a patient with this disease using Althesin. Ahnve, Lundman and Shoaleh-var (1978) found that the prolongation of QT was a predictor of ventricular arrhythmia in acute myocardial infarction. Althesin may prevent ventricular arrhythmia or aberration by stabilizing the QT interval.

**QT interval in association with QRS complex changes**

The children with BBB without aberration had a normal preanaesthetic QT interval whereas, in those showing BBB with aberration, QT was significantly prolonged before anaesthesia and during the arrhythmia. One child with a preanaesthetic QT of 594 ms developed bifocal ventricular
tachycardia during halothane anaesthesia. One year later QT was normal. The cause may have been preoperative anxiety. Halothane sensitizes the heart to the arrhythmogenic effect of catecholamines (Raventos, 1956; Katz and Katz, 1966). The prolongation of preanaesthetic QT may reflect high plasma concentrations of catecholamines and has a prognostic value in relation to arrhythmia occurring during oral surgery under halothane anaesthesia. Wig and others (1979) described sudden cardiac arrest during halothane anaesthesia in a patient with a preanaesthetic QT interval of 700 ms.

Olley and Fowler (1970) found that propranolol appeared to be effective in patients with congenital prolongation of QT in preventing syncopal ventricular fibrillation. Administration of a beta-blocker before induction of anaesthesia may be of value in patients with a prolonged preanaesthetic QT interval (Wig et al., 1979).

Lignocaine abolished aberrant conduction and normalized the prolonged QT interval. Atlee and Rusy (1977) state that, in addition to slowed conduction and unidirectional block, a potentially re-entrant impulse is required for aberration. The present results indicate that the prolongation of QT interval may activate this impulse.

QT interval remained the same, or shortened, during halothane anaesthesia, but was prolonged significantly during the first 3 min of enflurane anaesthesia and remained at this value throughout the anaesthesia. In spite of the prolongation of QT, neither aberration nor BBB was seen during enflurane anaesthesia, indicating that enflurane has antiarrhythmic properties. This result is supported by the findings of Reisner and Lippmann (1975) and Williams and Sone (1979), who found significantly fewer ventricular arrhythmias during enflurane than during halothane anaesthesia.

ACKNOWLEDGEMENTS

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VARIATIONS DE L’ELECTROCARDIOGRAMME PENDANT UNE ANESTHESIE A L’HALOTHANE ET L’ENFLURANE AVANT INTERVENTION CHIRURGICALE SUR DES ENFANTS

RESUME

On a comparé les variations de l’electrocardiogramme de 152 enfants subissant une excision des végétations adénoides ou une adéno-amygdalectomie (T + A) sous anesthésie à l’halothane ou à l’enflurane. Un rythme de jonction s’est produit dans 4–16% des enfants des groupes à excision des végétations adénoides et dans 11–33% des cas pour les groupes T + A. Le bloc de branche s’est produit dans 4% des enfants anesthésiés à l’halothane, mais aucun cas n’a été enregistré avec l’enflurane et il a été particulièrement courant en association avec le thio-pentone et les opérations de T + A. L’un des patients a souffert d’une tachycardie ventriculaire bifocale. L’intervalle TQ a été plus long par comparaison avec le témoin après le thio-pentone (P<0.001) et après le thio-pentone et le suxaméthonium (P<0.02). L’intervalle TQ n’a pas changé après l’Athésine, avec ou sans suxaméthonium. L’intervalle TQ moyen avant l’anesthésie (± erreur type des moyennes) a été prolongé d’une manière significative (492 ± 22 ms; normale 440 ms) chez les enfants souffrant d’une conduction anormale avec rythme chaotique, mais il était normal (438 ±5 ms) lorsque le bloc de branche ou le rythme de jonction était présent pendant l’anesthésie à l’halothane. L’intervalle TQ a été prolongé d’une manière significative avec l’anesthésie à l’enflurane, mais pas avec l’anesthésie à l’halothane.

EKG-VERÄNDERUNGEN BEI KINDERN WÄHREND HALOTHAN- UND ENFLURANNARKOSE BEI POLYPEN-UND MANDELRESEKTIONEN

ZUSAMMENFASSUNG

EKG-Veränderungen wurden bei 152 Kindern verglichen, die unter Halothan- oder Enflurannarkose eine Adenoidektomie oder Adenotonsillektomie (T + A) ausgesetzt wurden. Synaptischer Rhythmus ergab sich bei 4–16% der Adenoidektomie-Gruppen und bei 11–33% der T + A-Gruppen. Bündelblockierungen traten bei 4% der mit Halothan narkotisierten Kinder auf, nicht aber bei Enfluran, und war besonders häufig bei Thiopentone und T + A-Operationen; ein Kind entwickelte bifokale Ventrikeltachykardie. Das QT-Intervall war der Kontrolle gegenüber verlängert bei Thiopentone (P<0,001) und bei Thiopentone und Suxamethonium (P<0,02). Es blieb unverändert bei Althesin, mit oder ohne Suxamethonium. Das mittlere vorntarkotische QT-Intervall (± SEM) war wesentlich verlängert (492 ± 22 ms; normal 440 ms); bei den Kindern, die Leitunregelmäßigkeiten mit chaotischem Rhythmus zeigten; es war aber normal (438 ±5 ms), wenn Bündelblockierungen oder synaptischer Rhythmus während der Halothannarkose zu beobachten war. Das QT-Intervall war deutlich verlängert bei Enfluran-, nicht aber bei Halothan- narkose.

CAMBIOS EN EL ELECTROENCEFALOGRAMA DE NIÑOS SOMETIDOS A OPERACIONES QUIRÚRGICAS DE ADENOIDECTOMIA O DE ADENOTONSILECTOMIA BAJO ANESTESIA CON HALOTANO Y ENFLURANO

SUMARIO

Se compararon los cambios en el electroencefalograma de 152 niños sometidos a operaciones quirúrgicas de adenoidectomía o de adenotonsillectomía (T + A) bajo anestesia con halotano y enfurano. El ritmo de unión tuvo lugar en un 4–16% de los niños de los grupos de adenoidectomía y en un 11–33% de los
grupos de T + A. El bloqueo del conjunto de la rama tuvo lugar en un 4% de los niños anestesiados con halotano, pero no con enflurano, y fue bastante común en las operaciones de T + A y en lo relativo al uso de tiopentona; uno de los pacientes sufrió taquicardia bifocal del ventrículo. El intervalo QT fue bastante prolongado en comparación con el grupo de control, después de administrar tiopentona ($P < 0.001$), y tiopentona y suxametonio ($P < 0.02$). El intervalo QT no cambió después de la administración de altesin, con o sin suxametonio. El intervalo medio QT preanestésico ($\pm$ SEM) fue significativamente largo (492 ± 22 miliseg.; el normal fue de 440 miliseg.) en niños que mostraban una conducción aberrante con ritmo caótico pero fue normal (438 ± 5 miliseg.) cuando estuvo presente el bloqueo del conjunto de la rama o el ritmo de unión durante la anestesia con halotano. El intervalo QT se prolongó significativamente con la anestesia de enflurano pero no con la de halotano.