Recovery after halothane anaesthesia induced with thiopental, propofol–alfentanil or halothane for day-case adenoidectomy in small children

H. VIITANEN, P. ANNILLA, M. RORARIUS M. PALOHEIMO AND G. BAER

Summary
We studied recovery from halothane anaesthesia in 93 children, aged 1–3 yr, undergoing day-case adenoidectomy. Children were allocated randomly to receive thiopental 5 mg kg\(^{-1}\) (group TH), alfentanil 10 \(\mu g\) kg\(^{-1}\) and propofol 3 mg kg\(^{-1}\) (group PAH) or 5% halothane (group HH) for induction of anaesthesia. In group TH, tracheal intubation was facilitated with succinylcholine (suxamethonium) 1.5 mg kg\(^{-1}\). In groups PAH and HH, tracheal intubation was performed without neuromuscular block, and succinylcholine was used only if required. Anaesthesia was maintained with 1–3% halothane during spontaneous respiration. Times to achieving predetermined recovery end-points were recorded. Quality of recovery was assessed using a score of 1–9 (best to worst) for sedation, crying, restlessness and agitation. A postoperative questionnaire was used to determine the well-being of the child at home, 24 h after operation. Emergence from anaesthesia (response to non-painful stimuli) occurred earlier in group HH (mean 9 (SD 6) min) than in groups PAH (13 (6) min, \(P<0.01\)) and TH (18 (14) min, \(P<0.01\)). Sitting up, walking and home readiness were achieved earlier in groups PAH and HH than in group TH (\(P<0.05\) for each variable). Children in group TH were more sedated during the first 30 min after anaesthesia than those in the two other groups (\(P<0.05\)) while emergence-related delirium was more common in group HH than in group TH (\(P<0.01\)). Well-being at home was similar in all groups. We conclude that induction of halothane anaesthesia with propofol–alfentanil or halothane provided more rapid recovery and earlier discharge than that with thiopental. (Br. J. Anaesth. 1998; 81: 960–962)

Keywords: anaesthesia, paediatric; anaesthetics i.v., propofol; anaesthetics i.v., thiopental; anaesthetics volatile, halothane; recovery, postoperative; surgery, paediatric

Children aged 1–3 yr form a common group of patients undergoing short day-case procedures. The anaesthetic used should have minimal postoperative side effects to ensure rapid recovery from anaesthesia and return to a normal routine at home.

Propofol is a short-acting i.v. anaesthetic which, as an induction or maintenance agent, provides rapid recovery from anaesthesia.\(^1\) However, rapid recovery does not necessarily shorten the time to discharge.\(^1\) Further, duration of anaesthesia may override the benefit gained from propofol as an induction agent, making recovery more dependent on the maintenance agent.\(^2\)

Few studies have investigated recovery after propofol-induced anaesthesia in children less than 3 yr. We decided to compare the recovery profile, after halothane anaesthesia, of an induction dose of propofol with two conventional induction agents, thiopental and halothane, in children aged 1–3 yr undergoing day-case adenoidectomy.

Methods and results

After obtaining approval from the local Hospital Ethics Committee and written informed consent from parents, we studied 93 children, ASA I–II, aged 1–3 yr, undergoing day-case adenoidectomy. No premedication was used. Each child was allocated randomly to one of three groups using a computer-generated random number table: group TH (\(n=31\)) received thiopental 5 mg kg\(^{-1}\); group PAH (\(n=31\)) received propofol 3 mg kg\(^{-1}\) and alfentanil 10 \(\mu g\) kg\(^{-1}\) (60 s before propofol); and group HH (\(n=31\)) 5% inspired halothane for induction of anaesthesia. In group TH, tracheal intubation was facilitated with succinylcholine 1.5 mg kg\(^{-1}\). In groups PAH and HH, tracheal intubation was performed without neuromuscular block, and succinylcholine 1.5 mg kg\(^{-1}\) was given only if required. In all groups, anaesthesia was maintained with 1–3% halothane and 70% nitrous oxide in oxygen delivered via a Bain co-axial breathing system with the child breathing spontaneously. All children received diclofenac 12.5 mg rectally after tracheal intubation. Standard monitoring was used throughout anaesthesia.

In the recovery room, the recovery and behaviour of the children were assessed by the same nurse, who was unaware of the induction method used. Sedation, crying, restlessness (motor activity) and agitation were evaluated using an open scale scoring from 1–9 (best to worst). Scores were recorded at 10-min intervals for the first hour and then every 30
Thiopental, propofol or halothane for induction of halothane anaesthesia in children

961

min until discharge. If the child at any point of evaluation scored six or more for crying, restlessless or agitation, recovery from anaesthesia was considered delirious. Repeated doses of pethidine 5 mg i.v. were given for postoperative pain relief at the discretion of the recovery nurse. Times to achieving the following predetermined recovery end-points were measured (from discontinuation of halothane): (1) time to responding to non-painful stimuli (emergence time); (2) time to first dose of pethidine; (3) time to sitting up; (4) time to drinking; (5) time to walking; and (6) time taken to achieve the criteria for discharge from the recovery room, which were stable vital signs for at least 30 min, able to walk according to age, tolerance of clear fluids, no nausea or vomiting, and no pain.

A postoperative questionnaire was given to parents to be completed at home. Parents were asked to record the well-being and behaviour of the child (tiredness, eating, drinking ability, sleeping, vomiting, pain) until 24 h after discharge.

Analyses were performed using one-way analysis of variance (ANOVA) with Bonferroni’s correction, Mann Whitney U test and chi-square test where appropriate. P < 0.05 was considered significant. Data are presented as mean (sd or number (%). In order to detect a 20-min difference in discharge time with a mean value of 100 min (sd 25 min), 25 patients were required in each group. This gave the study a power of 80% at α = 0.05.

The three groups were comparable in age, weight, duration of anaesthesia and surgery. Mean duration of anaesthesia was 22 (5), 24 (7) and 23 (6) min in groups TH, PAH and HH, respectively (ns). Tracheal intubation was successful in all patients in groups TH and HH. Seven patients in group PAH were given succinylcholine to facilitate tracheal intubation. The mean dose of rectal diclofenac was 0.95–0.98 mg kg⁻¹ in the different groups (ns). Time to achieve recovery end-points are shown in table 1. Children in group PAH were more sedated than those in group HH at each time. The incidence of emergence-related agitation scored six or more for crying, restlessness or agitation, was 7%, 23% and 29% in groups TH, PAH and HH respectively (ns). No children experienced delirium in the higher sedation scores at 60 and 90 min after anaesthesia. The incidence of emergence delirium and later administration of pethidine in the propofol–alfentanil group compared with the halothane group. Further, the longer redistribution half-life of thiopental (43 min) compared with propofol (26 min) resulted in higher scores for sedation for a longer period after anaesthesia in the thiopental group.

The use of alfentanil in the propofol–alfentanil group may have prolonged time to emergence. However, although emergence was fastest in the halothane group, children in the propofol–alfentanil group did not differ from those in the halothane group for any of the other recovery variables. This probably reflects the rapid clearance of propofol and alfentanil in children.

The short duration of anaesthesia in our study could also explain why the use of no i.v. induction agent provided the fastest emergence and rapid recovery from anaesthesia. Failure of several tissues to reach equilibration with the alveolar anaesthetic partial pressure during a short anaesthetic would result in more rapid elimination of halothane after discontinuation. Hanallah and colleagues found that early recovery (full Steward score) from anaesthesia induced with halothane did not differ from thiopental. Our results could be explained by the longer duration of anaesthesia (> 80 min) in their study.

More children experienced “delirious recoveries” (crying, restlessness and agitation) in the halothane group compared with the thiopental group. Time to peak blood concentration after rectal diclofenac was 30 min while for some children, would have occurred 10–15 min after anaesthesia. Rapid emergence from anaesthesia may have led to the delirious recoveries through early perception of pain and could account for the significantly earlier administration of analgesics in the halothane group. The reduced incidence of emergence delirium and later administration of pethidine in the propofol–alfentanil group could be a result of the analgesic effect of alfentanil extending into the immediate recovery period.

Table 1 Recovery times (min) from the end of anaesthesia in the three groups (mean (sd) and 95% confidence intervals (CI))

<table>
<thead>
<tr>
<th>Group</th>
<th>TH (n=31)</th>
<th>PAH (n=31)</th>
<th>HH (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emergence</td>
<td>18 (14)</td>
<td>13 (6)</td>
<td>9 (6)*</td>
</tr>
<tr>
<td>Sitting</td>
<td>13–23</td>
<td>10–14</td>
<td>7–11</td>
</tr>
<tr>
<td>Drinking</td>
<td>63 (24)*</td>
<td>50 (25)</td>
<td>43 (22)</td>
</tr>
<tr>
<td>Walking</td>
<td>52–71</td>
<td>40–60</td>
<td>35–51</td>
</tr>
<tr>
<td>Discharge</td>
<td>71–91</td>
<td>59–79</td>
<td>61–80</td>
</tr>
<tr>
<td></td>
<td>132 (29)*</td>
<td>112 (31)</td>
<td>108 (22)</td>
</tr>
<tr>
<td></td>
<td>121–142</td>
<td>100–123</td>
<td>100–116</td>
</tr>
</tbody>
</table>

Comment

Our findings differed from two previous studies investigating recovery after halothane anaesthesia induced with propofol, thiopental or halothane in children older than 3 yr. Aun and colleagues found no differences in early recovery characteristics (open eyes on command, full Steward score) between induction agents. Our results support the suggestion that as the duration of anaesthesia increases, recovery becomes more dependent on the maintenance agent rather than on the induction agent. The short duration of anaesthesia in our study (< 25 min) allowed both i.v. induction agents to have a residual effect in the early recovery period, thus explaining the slower awakening (emergence) in the thiopental and propofol–alfentanil groups compared with the halothane group. Further, the longer redistribution half-life of thiopental compared with propofol resulted in higher scores for sedation for a longer period after anaesthesia in the thiopental group.

Postoperative vomiting in the thiopental group may have prolonged time to emergence. The short duration of anaesthesia in our study could also explain why the use of no i.v. induction agent provided the fastest emergence and rapid recovery from anaesthesia. Failure of several tissues to reach equilibration with the alveolar anaesthetic partial pressure during a short anaesthetic would result in more rapid elimination of halothane after discontinuation. Hanallah and colleagues found that early recovery (full Steward score) from anaesthesia induced with halothane did not differ from thiopental. Our results could be explained by the longer duration of anaesthesia (> 80 min) in their study.

More children experienced “delirious recoveries” (crying, restlessness and agitation) in the halothane group compared with the thiopental group. Time to peak blood concentration after rectal diclofenac was 30 min while for some children, would have occurred 10–15 min after anaesthesia. Rapid emergence from anaesthesia may have led to the delirious recoveries through early perception of pain and could account for the significantly earlier administration of analgesics in the halothane group. The reduced incidence of emergence delirium and later administration of pethidine in the propofol–alfentanil group could be a result of the analgesic effect of alfentanil extending into the immediate recovery period.

Time to home readiness was similar in both the propofol–alfentanil and halothane groups, being significantly shorter than in the thiopental group. Later administration of pethidine in the thiopental group may have caused residual sedation, which is reflected in the higher sedation scores at 60 and 90 min after
anaesthesia. Also, more children vomited at a later stage in the thiopental group, which prolonged discharge time in some children.

In summary, we conclude that i.v. induction with propofol-alfentanil and inhalation induction with halothane provided similar rapid recovery characteristics after maintenance of anaesthesia with halothane. The only difference between the two groups was shorter emergence time and lower sedation scores during the first 10 min after the end of anaesthesia in the halothane group. Induction with thiopental resulted in significantly slower recovery after anaesthesia compared with the two other groups, but recovery at home was not affected by the induction agent.

References