PREVENTION OF INTRACRANIAL HYPERTENSION DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

Use of a second dose of thiopentone

V. K. N. Unni, R. A. Johnston, H. S. A. Young and R. J. McBride

SUMMARY

In nine patients, with preoperative ICP monitoring, anaesthesia was induced with thiopentone 5 mg kg\(^{-1}\) given over 1 min, followed by pancuronium 0.1 mg kg\(^{-1}\). After manual hyperventilation with nitrous oxide and oxygen for 3 min they were given thiopentone 2.5 mg kg\(^{-1}\) over 30 s (phase 1); 30 s later laryngoscopy was performed and topical analgesia administered to the larynx. Endotracheal intubation was performed 1 min after spraying the cords (phase 2). The measurements continued for a further 5 min during which the patients were mechanically ventilated (phase 3). ICP and intra-arterial pressure were recorded. Although there was a significant decrease \((P < 0.05)\) in MAP at the end of the second dose of thiopentone, there were no other significant changes in ICP, MAP or PacO\(_2\) throughout the study. In two patients there were transient decreases in cerebral perfusion pressure to less than 60 mm Hg. Although MAP increased in five of the patients during laryngoscopy and intubation, there was no increase in ICP, showing that the MAP was still within the autoregulatory limits.

Acute increases in intracranial pressure (ICP) and mean arterial pressure (MAP) during laryngoscopy and endotracheal intubation have been well documented. These increases, which are more marked in neurosurgical patients with intracranial hypertension (Shapiro, Galindo et al., 1972), might lead to brain shift (Fitch and McDowall, 1971) and tissue compression with concomitant decreases in total or regional cerebral blood flow, or both (Zwetnow, 1970). Techniques recommended by different authors to prevent these changes in ICP and MAP during laryngoscopy and endotracheal intubation include the administration of thiopentone or trimetaphan immediately before the procedures (Shapiro and Aidinis, 1975), pretreatment with \(\beta\)-adrenoceptor blocking agents (Greenbaum, 1976), and pretreatment with lignocaine (Bedford et al., 1980). The present study was conducted to evaluate the efficacy of a second dose of thiopentone in preventing the changes in systemic arterial, intracranial and cerebral perfusion pressures during these procedures.

PATIENTS AND METHODS

Nine patients undergoing preoperative ICP monitoring, who were scheduled for craniotomy, were studied. Their ages, sex and pathological diagnoses are given in table I. All were free from major cardiac and respiratory disease. They were premedicated with diazepam 10 mg by mouth approximately 2 h before the induction of anaesthesia. Control measurements of arterial pressure and ICP were recorded for 10 min before the induction of anaesthesia. The subsequent management was divided into three phases as follows:

**Phase 1**—atropine 0.6 mg i.v. One minute later thiopentone 5 mg kg\(^{-1}\) was given over 1 min, followed by pancuronium 0.1 mg kg\(^{-1}\). Hyperventilation with nitrous oxide 10 litre in oxygen 5 litre for 3 min using a non-rebreathing system. Thiopentone 2.5 mg kg\(^{-1}\) given over 30 s.

**Phase 2**—Laryngoscopy and topical analgesia to the larynx, 30 s after the second dose of thiopentone. Hyperventilation for 1 min followed by endotracheal intubation.

**Phase 3**—Hyperventilation for 5 min during which all measurements were continued.

ICP was measured using a subarachnoid Rich mond screw. Arterial pressure was measured using an intra-arterial cannula in the radial or the dorsalis pedis artery. The intracranial and arterial pressures were recorded simultaneously on FM magnetic tape and on an Elcomatic paper recorder. The magnetic tape was subjected to off-line computer analysis to produce values for mean ICP and arterial pressure. Cerebral perfusion pressure (CPP) was calculated as the difference between ICP and MAP. ECG was
### TABLE I. Mean values for ICP, MAP and $P_{acO_2}$ during control period (C) and phases 1, 2, and 3 (P1, P2, P3). SAH = Subarachnoid haemorrhage; n.s. = Not significant

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Drugs</th>
<th>ICP (mm Hg)</th>
<th>MAP (mm Hg)</th>
<th>$P_{acO_2}$ (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>C</td>
<td>P1</td>
<td>P2</td>
</tr>
<tr>
<td>42</td>
<td>M</td>
<td>SAH</td>
<td>Tramexamic acid</td>
<td>21</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>36</td>
<td>M</td>
<td>Cerebellar astrocytoma</td>
<td>Dexamethasone</td>
<td>8</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>40</td>
<td>F</td>
<td>SAH</td>
<td>Tramexamic acid</td>
<td>20</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>42</td>
<td>M</td>
<td>Parietal astrocytoma</td>
<td>Dexamethasone</td>
<td>21</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>44</td>
<td>M</td>
<td>Arnold Chiari</td>
<td>Dexamethasone</td>
<td>10</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>35</td>
<td>M</td>
<td>Parietal secondary glioma</td>
<td>Dexamethasone</td>
<td>18</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>46</td>
<td>M</td>
<td>Temporal glioma</td>
<td>Dexamethasone</td>
<td>20</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>64</td>
<td>M</td>
<td>Parietal subdural</td>
<td>Dexamethasone</td>
<td>5</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>61</td>
<td>M</td>
<td>Occipital glioma</td>
<td>Dexamethasone</td>
<td>16</td>
<td>17</td>
<td>16</td>
</tr>
</tbody>
</table>

Mean SEM P

- **ICP (mm Hg)**
  - Mean: 15.4
  - SEM: 2.06
  - P: n.s.

- **MAP (mm Hg)**
  - Mean: 13.6
  - SEM: 2.26
  - P: n.s.

- **$P_{acO_2}$ (kPa)**
  - Mean: 11.9
  - SEM: 2.94
  - P: n.s.

### TABLE II. Mean values for ICP, MAP and CPP during control period (C) and at 3, 5, 6 and 8 min of induction of anaesthesia, and the peak changes at laryngoscopy and topical spray (L) and endotracheal intubation (I). n.s. = Not significant

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Sex</th>
<th>C</th>
<th>3 min</th>
<th>5 min</th>
<th>6 min</th>
<th>L</th>
<th>8 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>42</td>
<td>M</td>
<td>21</td>
<td>9</td>
<td>10</td>
<td>10</td>
<td>11</td>
<td>115</td>
</tr>
<tr>
<td>36</td>
<td>M</td>
<td>8</td>
<td>12</td>
<td>10</td>
<td>10</td>
<td>11</td>
<td>100</td>
</tr>
<tr>
<td>40</td>
<td>F</td>
<td>20</td>
<td>23</td>
<td>22</td>
<td>20</td>
<td>23</td>
<td>97</td>
</tr>
<tr>
<td>42</td>
<td>M</td>
<td>21</td>
<td>11</td>
<td>13</td>
<td>8</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>44</td>
<td>M</td>
<td>10</td>
<td>11</td>
<td>10</td>
<td>13</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>35</td>
<td>M</td>
<td>18</td>
<td>11</td>
<td>10</td>
<td>21</td>
<td>30</td>
<td>18</td>
</tr>
<tr>
<td>46</td>
<td>M</td>
<td>20</td>
<td>11</td>
<td>10</td>
<td>8</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>64</td>
<td>M</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>61</td>
<td>M</td>
<td>16</td>
<td>12</td>
<td>18</td>
<td>15</td>
<td>18</td>
<td>14</td>
</tr>
</tbody>
</table>

Mean SEM P

- **ICP (mm Hg)**
  - Mean: 11.2
  - SEM: 1.86
  - P: n.s.

- **MAP (mm Hg)**
  - Mean: 10.1
  - SEM: 2.47
  - P: n.s.

- **CPP (mm Hg)**
  - Mean: 12.1
  - SEM: 1.67
  - P: n.s.
monitored continuously using standard limb leads (Lead II). Arterial blood was collected every minute from a second arterial cannula. Each sample collected over a 30-s period was analysed for pH, P\textsubscript{CO}_2 and P\textsubscript{O}_2 using an IL 613 blood-gas analyser.

Student's t test was used to test the significance of difference between results, a difference being considered significant when the P value was less than 0.05.

RESULTS

The results are presented in two different forms. Table I gives the mean values for ICP, MAP and P\textsubscript{ACO}_2 during the control period, and during phases 1, 2 and 3. Table II shows the values for ICP, MAP and CPP during the control period and at 3, 5, 6 and 8 min (1 min after intubation) as well as the peak changes during laryngoscopy and intubation.

Intracranial pressure. Although there were changes in ICP in individual patients, in the series as a whole the ICP remained below control values throughout the study (tables I, II). Except in one patient during laryngoscopy and endotracheal intubation, the ICP did not increase above preinduction values (table II). The decrease in ICP noted was not significant.

Mean arterial pressure. In the majority of patients, MAP decreased during phases 1 and 3, and increased during phase 2. However, these changes were not significant (table I). The maximum decrease in MAP was at 6 min, that is, 30 s after the second dose of thiopentone, this change being significant. All but two patients showed an increase in MAP during laryngoscopy and intubation. These were transient changes as the MAP returned to control values as soon as the procedures had been completed (table II). The CPP followed the pattern of MAP and the mean changes were not significant. In two patients the CPP decreased transiently below 60 mm Hg, but the CPP was never less than 50 mm Hg at any time (table II).

Arterial P\textsubscript{CO}_2. A small decrease in average P\textsubscript{CO}_2 values was noticed during the study.

DISCUSSION

During the induction of anaesthesia in the neurological patient, several factors can produce an increase in ICP. These include hypoxia, hypercapnia, straining, laryngoscopy, endotracheal intubation and drugs used during induction. A smooth induction should exclude the first three, while one could, to a certain extent, avoid the use of drugs that are known to increase the ICP. The present study demonstrated that the administration of thiopentone, just before laryngoscopy and endotracheal intubation, prevented an increase in ICP during these procedures.

The most interesting finding in the present study was that, although MAP during laryngoscopy and intubation increased above the control values, ICP remained near the control figures in most patients, thus showing that MAP was still below the upper limit of autoregulation. The hypotensive effect of the second dose of thiopentone was manifest at about 30 s after its administration, and this seems the optimum time for laryngoscopy.

The magnitude of the increase in ICP during endotracheal intubation has been reported to be between 50 mm Hg (Misfeldt, Jorgensen and Rishoj, 1974) and 100 mm Hg (Greenbaum et al., 1975). However, these findings are not comparable to those of the present study. Misfeldt and colleagues (1974) used suxamethonium, a drug known to increase ICP (Sondergard, 1961), while Greenbaum and colleagues (1975) used methohexitone as the induction agent. From the latter study it would seem that methohexitone does not produce the same decrease in ICP as thiopentone or Althesin, as in many of their patients there was a progressive increase in ICP even before laryngoscopy and endotracheal intubation were undertaken.

Shapiro and co-workers (Shapiro, Galindo et al., 1972; Shapiro, Wyte et al., 1972; Shapiro et al., 1973) recommended thiopentone as a means of reducing ICP in neurosurgical patients. Although they did not use a second dose, the increase in ICP was not as great as that recorded by Greenbaum and colleagues (1975) or Misfeldt, Jorgensen and Rishoj (1974).

One of the problems anticipated in the present study was arterial hypotension following large doses of thiopentone. However, the slow administration of the drug minimized the arterial hypotension as the rate of administration of thiopentone is an important determinant of its haemodynamic effects (Fieldman, Ridley and Wood, 1955). Nevertheless, there was a significant decrease in MAP after the second dose of thiopentone. Since there was a decrease in ICP in the majority of patients, the CPP was well maintained. In two patients (2 and 5 in table II), after the second dose of thiopentone, CPP
decreased below 60 mm Hg. As the MAP in these patients, just before the second dose of thiopentone, was 70 and 74 mm Hg, respectively, it would seem advisable not to use a second dose if the MAP decreases below 80 mm Hg as the decrease in CPP may be sufficient to impair cerebral autoregulation.

Pancuronium was preferred to tubocurarine in the present study because of the hypotensive effect of the latter (Thomas, 1957). Seven of nine patients in whom anaesthesia was induced with thiopentone and tubocurarine by Moss and co-workers (1978) showed a decrease in CPP to less than 50 mm Hg, and in three the CPP remained at this value for at least 5 min after the induction of anaesthesia. None of our patients had a CPP of less than 50 mm Hg. Pancuronium has a systemic hypertensive effect (Kelman and Kennedy, 1971) which may have counteracted some of the hypotensive action of thiopentone.

Table II shows that the second dose of thiopentone did not attenuate the increase in MAP during laryngoscopy and intubation. In patients with a recent subarachnoid haemorrhage, a sudden increase in MAP might precipitate fresh bleeding from an aneurysm. Conversely, a decrease in MAP might impair the blood flow to areas where autoregulation is defective. Therefore, in these patients it may be advisable to use some other agent such as a β-adrenergic blocking agent to attenuate the increase in MAP during laryngoscopy and intubation.

It was anticipated that the early introduction of nitrous oxide might cause an increase in ICP as this agent is known to increase the CBF (Thye and Michenfelder, 1968) and ICP (Phirman and Shapiro, 1977) in man. Misfeldt, Jorgensen and Rishoj (1974) have shown that prior hyperventilation minimized this effect of nitrous oxide. The decrease in PACO₂ in the present study was not significant and the patients were not hypocapnic. However, the prevention of hypercapnia may be one of the reasons why nitrous oxide did not produce any increase in ICP. In the study by Moss and colleagues (1978) the early introduction of nitrous oxide did not seem to have influenced the ICP.

The changes in PACO₂ in the present study were small, although early hyperventilation was attempted in every patient. During phase 1, passive hyperventilation was possible only in the latter part, once the effect of pancuronium was established. During phase 2 there were brief periods of apnoea during laryngoscopy to spray the cords and for endotracheal intubation. Again, during phase 3, it was necessary to disconnect the patient from the anaesthetic system for brief periods to fix the endotracheal tube.

The present work was not undertaken to study the effects of a particular drug on ICP. However, using the technique described, it was possible to abolish the increase in ICP during intubation of the trachea in the majority of patients. Nevertheless, in patients with intracranial aneurysms it may be better to use some other technique to attenuate the increase in ICP during laryngoscopy and intubation.

ACKNOWLEDGEMENTS

We are grateful to Messrs D. S. Gordon, I. C. Bailey and D. P. Byrnes for permitting us to carry out this study on patients under their care. We also thank Miss P. Johnston, Sister-in-charge of the neurosurgical theatres and Mr T. Ryan, medical physics technician, for their help during this study.

REFERENCES


PREVENTION OF INTRACRANIAL HYPERTENSION

**SUMMARY**

En nueve pacientes con vigilancia ICP preoperatoria, se indujo la anestesia con 5 mg kg⁻¹ de tiopental administrada durante 1 min, seguida por 0,1 mg kg⁻¹ de pancuronio. Después de una hiperventilación manual con oxígeno y oxígeno durante 3 min, se les administró 2.5 mg kg⁻¹ de tiopental en un período de 30 s (Fase 1); 30 s más tarde, se llevó a cabo la laringoscopia y se administró una analgesia tópica a la larínge. Se llevó a cabo la intubación endotracheal 1 min después de rociar las cuerdas (Fase 2). Siguió las mediciones durante unos 5 min adicionales en que se ventilaron mecánicamente a los pacientes (Fase 3). Se registraron el ICP y la presión intracraneal. Aunque hubo un descenso significativo (P<0,05) del MAP al fin de la segunda dosis de tiopental, no hubo ningún cambio adicional en el ICP, el MAP o el PaCO₂ durante todo el estudio. En dos pacientes, se registró un descenso transitorio de la presión de perfusión cerebrovascular hasta menos de 60 mm Hg. Aunque el MAP aumentó en cinco de los pacientes durante la laringoscopia y la intubación, no hubo incremento del ICP, lo que demuestra que el MAP se hallaba todavía dentro de los límites autoregulatorios.