NON-INVASIVE MEASUREMENT OF CARDIAC OUTPUT DURING INDUCTION OF ANAESTHESIA AND TRACHEAL INTUBATION: THIOPENTONE AND PROPOFOL COMPARED

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SUMMARY

We have investigated the haemodynamic changes in response to induction of anaesthesia and tracheal intubation in patients who received either thiopentone 5 mg kg⁻¹ or propofol 3 mg kg⁻¹ followed by atracurium 0.5 mg kg⁻¹ and fentanyl 1.5 μg kg⁻¹. Anaesthesia was maintained with 0.6% enflurane and 50% nitrous oxide in oxygen with assisted ventilation. Cardiac output and heart rate (HR) were monitored continuously with a transthoracic impedance monitor. Mean HR did not change after induction in each group, but increased after tracheal intubation in both groups (P < 0.01). Mean cardiac index (CI) decreased after induction (P < 0.05) and decreased further after tracheal intubation in both groups (P < 0.05). There was no difference between the two groups with respect to changes in CI and HR. Mean arterial pressure (MAP) and systemic vascular resistance (SVR) did not change significantly after induction in the thiopentone group. Both variables increased from preinduction values 1 min after tracheal intubation (P < 0.001). In contrast, both MAP and SVR decreased after induction in the propofol group (P < 0.001) and did not differ from preinduction values 1 min after tracheal intubation. MAP and SVR were greater in the thiopentone group compared with the propofol group after induction and tracheal intubation (P < 0.01).

KEY WORDS

Since the first report by King and colleagues [1], the haemodynamic consequences of tracheal intubation have been investigated widely, and many methods of obtunding the pressor response have been evaluated. Because of difficulties in the measurement of cardiac output (CO), these studies have been confined mainly to the measurement of changes in heart rate (HR) and mean arterial pressure (MAP). The Bomed non-invasive continuous cardiac output monitor (BOMED) measures continuously changes in the thoracic electrical impedance to a 2.5-mA, 70-KHz alternating current during the cardiac cycle. These measured changes are used to derive a value for cardiac output using the Bernstein modification of the Sramek formula [2] and is updated continuously on a 12-heartbeat cycle. The measurements have been shown to correlate well with those derived using thermodilution techniques [3–9] and they follow CO trends as well as, if not better than, thermodilution techniques [4, 7, 10]. This study was designed to examine the effects of induction of anaesthesia and tracheal intubation in patients of ASA grade I or II receiving either propofol or thiopentone given as part of a balanced anaesthetic technique.

PATIENTS AND METHOD

After receiving local Ethics Committee approval, we obtained informed consent from 32 female patients of ASA grade I or II undergoing laparoscopy. Patients were premedicated with temazepam 20 mg given orally 1 h before operation. On arrival of the patient in the anaesthetic room...
CARDIAC OUTPUT AFTER THIOPENTONE OR PROPOFOL

### Table 1. Patient characteristics (mean (range or SEM)). Induction of anaesthesia was with propofol (group P) or thiopentone (group T). BSA = body surface area

<table>
<thead>
<tr>
<th></th>
<th>Age (yr)</th>
<th>BSA (m²)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group P</td>
<td>30.1 (25–39)</td>
<td>1.61 (0.04)</td>
<td>159 (1.8)</td>
<td>59.7 (2.6)</td>
</tr>
<tr>
<td>Group T</td>
<td>29.7 (19–44)</td>
<td>1.66 (0.03)</td>
<td>158 (1.6)</td>
<td>65.5 (3.2)</td>
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</tbody>
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**Fig. 1.** Changes in cardiac index (CI) and heart rate (HR) after induction of anaesthesia and tracheal intubation (mean, SEM) in patients given thiopentone (●) or propofol (○). Significant changes (P < 0.05): * from baseline (B); † from preintubation value (I).

There was no difference in height, weight, age or BSA between the patients given thiopentone (group T) or propofol (group P) (table I). One patient was excluded from the trial because of

room, an 18-gauge cannula was inserted into a vein in the antecubital fossa. Non-invasive arterial pressure was measured using an automated method (Dinamap). Following skin preparation, two thoracic sensing electrodes (Medicotest-VL-00-S) were placed at the level of the xiphoid in each mid axillary line. Two cervical sensing electrodes were placed laterally at the root of the neck. Two current sensing electrodes were positioned 5 cm above the cervical and 5 cm below the thoracic sensing electrodes and connected to the BOMED (BoMed NCCOM3, software version 6, BoMed Biomedical, Irvine, CA). The waveform derived by the BOMED was monitored. A period of 5 min was allowed for the patient's recordings to stabilize. The preinduction (baseline) measurements of MAP, CO and HR were recorded.

Patients were allocated randomly to one of two equal groups to receive either thiopentone 5 mg kg⁻¹ or propofol 3 mg kg⁻¹ given over 20 s. The induction agent was followed immediately by fentanyl 1.5 μg kg⁻¹ and atracurium 0.5 mg kg⁻¹. Tracheal intubation was performed 90 s after induction of anaesthesia by the same anaesthetist on every occasion. The view of the larynx during laryngoscopy was graded according to the classification described by Cormack and Lehane [11]. The patient was excluded from the trial if the vocal cords were not visualized (laryngoscopic grade 3 or 4). Anaesthesia was maintained subsequently with 0.6% enflurane and 50% nitrous oxide in oxygen. Ventilation was assisted using a Manley MP2 ventilator and normocapnia maintained and monitored with a capnograph. Further recordings of HR, MAP and CO were made 90 s after induction of anaesthesia (just before tracheal intubation) and at 1-min intervals for 8 min after tracheal intubation.

The patient's body surface area (BSA) was derived from a standard nomogram for height and weight [12]. Cardiac index (CI) and systemic vascular resistance (SVR) were calculated using the standard formulae. All data were analysed using paired and unpaired t tests as appropriate and multivariate analysis of variance (MANOVA).

**RESULTS**

There was no difference in height, weight, age or BSA between the patients given thiopentone (group T) or propofol (group P) (table I). One patient was excluded from the trial because of
difficulty with tracheal intubation (laryngoscopic grade 3). There were thus 16 patients in group P and 15 in group T. None of the patients coughed at tracheal intubation.

Cardiac index

There was no difference in the mean baseline CI or in response to induction and tracheal intubation (fig. 1) in the two groups. Induction of anaesthesia was associated with a decrease in CI in both groups \( (P < 0.05) \) and a further decrease 1 min after tracheal intubation \( (P < 0.05) \).

Heart rate

There was no difference between the two groups in the mean baseline HR or in the response to induction and tracheal intubation (fig. 1). Induction of anaesthesia did not change HR in each group and there was a significant increase in HR in both groups 1 min after tracheal intubation \( (P < 0.01) \).

Mean arterial pressure

The mean baseline MAP was not significantly different between the two groups (fig. 2). Induction with propofol was associated with a significant decrease in MAP \( (P < 0.001) \) which did not occur in group T. MAP increased in both groups 1 min after tracheal intubation. The MAP was significantly greater in group T after induction \( (\text{MANOVA} \ P < 0.01) \). Individual t tests identified these differences to be before and 6 min after tracheal intubation \( (P < 0.01) \). The 1-min post-intubation MAP readings were compared with preintubation MAP. There was no difference between the two groups in the degree of change in MAP in response to tracheal intubation.

Systemic vascular resistance

Mean SVR (fig. 2) decreased after induction with propofol \( (P < 0.001) \), but not after thiopentone. SVR was greater in group T after induction compared with group P \( (P < 0.01) \). One minute after tracheal intubation, SVR increased from baseline in the thiopentone group \( (P < 0.001) \), whereas after propofol it returned to baseline. There was no difference between the two groups in the degree of change in SVR in response to tracheal intubation.

DISCUSSION

In this study cardiac index decreased after induction of anaesthesia in both groups. However, we found that there was no difference between the effects of thiopentone and propofol on CI. The changes in HR and MAP are comparable to those observed in other studies. The vasodilator effect of propofol [13] was confirmed by the greater reduction in SVR after induction of anaesthesia in group P compared with group T. The changes in MAP were a reflection of these changes in SVR in both groups. This may explain why, in comparison with baseline values, hypertension occurs in response to tracheal intubation after induction of anaesthesia with thiopentone but not after propofol [14].

The bioimpedence method \( (Q_{Bi}) \) offers a simple, non-invasive method for measurement of CI in ASA I or II patients. It has been compared extensively with the thermodilution method \( (Q_{TD}) \)
mainly, but not exclusively [7, 9, 10], in the critical care setting. The reported correlation coefficients vary between 0.62 and 0.97 [3–10]. When assessing reliability, the coefficient of variation has been found to vary between 5.1% and 8.9% with Q_{BI} compared with 8.1–18.6% with Q_{TD} [7, 8, 10]. It has been calculated that a 5–10% change in CO may be detected reliably with 25–100 measurements [15]. The work by Sullivan and colleagues [9] is more relevant to this study because they compared Q_{BI} with Q_{TD} in patients undergoing anaesthesia for aortic aneurysm surgery. The measurements were made awake, following induction, following tracheal intubation, before skin incision and at stages during the surgical procedure. All these studies show that Q_{BI} consistently neither underestimates nor overestimates CO in comparison with Q_{TD} and that it is reliable.

The similarity in haemodynamic changes in response to tracheal intubation in the two groups may suggest that there is no difference between the two agents, thiopentone and propofol, in their effects on the mechanisms underlying the pressor response to tracheal intubation. The dose equivalence ratio of thiopentone:propofol (1:1.6) is based on work by Grounds, Moore and Morgan [16]. They studied unpremedicated patients and did not administer any other anaesthetic agent. The doses we used are extrapolated from those used in their study. We cannot assume, therefore, that the ratio ensures equipotency and some may consider the dose of propofol to be large. In addition, enflurane and IPPV may have affected our findings and the small dose of fentanyl given at induction is known to obtund [17], although not abolish, the pressor response to tracheal intubation.

The continued decrease in CI after tracheal intubation (when concomitant increases in HR and MAP occurred) was an unexpected finding. We might have expected to see an increase in CI in response to tracheal intubation because of release of catecholamines at this time [18–20]. Although Moffit and colleagues [21] found such an increase in CI after tracheal intubation, Patrick and co-workers [22] found a lower CI after tracheal intubation in patients undergoing coronary artery surgery. Barash, Kopriva and Giles [23], using a radionuclide technique, found a decrease in stroke volume in response to tracheal intubation in patients without cardiovascular disease. Possible explanations for this unexpected decrease in CI include the effects of IPPV [24] in addition to those of thiopentone, propofol and enflurane on myocardial contractility. A small preload or a large afterload may also lead to a decrease in CO. These changes might result from the effects of the instantaneous release of catecholamines on the pulmonary and systemic circulations. The balance between the positive and negative inotropic effects at the time of tracheal intubation is clearly important, and the decrease in CI may be simply a result of a persisting negative inotropic effect of the anaesthetic agents which is greater than any positive inotropic effect of the catecholamines released in response to tracheal intubation. This requires further investigation.

REFERENCES


