British Journal of Anaesthesia 1993; **70**: 163–166

TRACHEAL INTUBATION AFTER INDUCTION OF ANAESTHESIA WITH PROPOFOL, ALFENTANIL AND I.V. LIGNOCAINE

J. A. H. DAVIDSON AND J. A. GILLESPIE

SUMMARY

We have assessed tracheal intubating conditions in 60 ASA I or II patients after induction of anaesthesia with propofol 2.5 mg kg^{-1} and alfentanil 10 or 20 μg kg^{-1} with or without i.v. lignocaine 1 mg kg^{-1}. No neuromuscular blocking agents were administered. Patients were allocated randomly to four groups: group 1 = propofol–alfentanil 10 μg kg^{-1}; group 2 = propofol–alfentanil 10 μg kg^{-1}–lignocaine 1 mg kg^{-1}; group 3 = propofol–alfentanil 20 μg kg^{-1}; group 4 = propofol–alfentanil 20 μg kg^{-1}–lignocaine 1 mg kg^{-1}. Intubating conditions were assessed as acceptable or unacceptable on the basis of a scoring system dependent on ease of laryngoscopy, vocal cord position and coughing on insertion of the tracheal tube. Intubating conditions were acceptable in 20%, 73%, 73% and 93% of patients in groups 1–4, respectively. Intubating conditions were better and there was less coughing in the lignocaine group. (Br. J. Anaesth. 1993; **70**: 163–166)

KEY WORDS

Anaesthesia: intravenous. Intubation tracheal: technique.

Recent work has suggested that in premedicated patients an induction dose of propofol 2.5 mg kg^{-1} may provide adequate conditions for laryngoscopy and tracheal intubation without the need for neuromuscular blocking agents [1]. The efficacy of this single agent technique, however, has been debated [2], but in several individuals it is associated with sub-optimal conditions: undesirable haemodynamic responses, difficulty in seeing the larynx and intolerance of the tracheal tube.

It has been demonstrated that adjuvant agents, such as opioids, adrenergic blockers, and local anaesthetic agents, may suppress the cardiovascular response to laryngoscopy and tracheal intubation [3, 4] and improve tolerance of the tracheal tube. Although alfentanil and lignocaine have been shown to attenuate the cardiovascular response to intubation [5, 6], the effect of a combination of these drugs on intubating conditions remains poorly documented.

In this double-blind, randomized study, intubating conditions and haemodynamic response to intubation (in the absence of neuromuscular block) were assessed in patients induced with propofol 2.5 mg kg^{-1} and alfentanil 10 or 20 μg kg^{-1}. The effect of supplementing this regimen with i.v. lignocaine 1 mg kg^{-1} was evaluated.

PATIENTS AND METHODS

We studied 60 ASA I or II patients undergoing elective gynaecological surgery. Ethics Committee approval was obtained and each patient gave written informed consent. Patients whose physical characteristics suggested difficulties in intubation (Mallampati Class III) [7] and those who had a previously documented failed intubation were excluded from the study. All patients were premedicated with temazepam 20–30 mg approximately 1 h before anaesthesia. Patients were allocated randomly to one of four groups: group 1 = propofol 2.5 mg kg^{-1} and alfentanil 10 μg kg^{-1}; group 2 = propofol 2.5 mg kg^{-1}, alfentanil 10 μg kg^{-1} and i.v. lignocaine 1 mg kg^{-1}; group 3 = propofol 2.5 mg kg^{-1} and alfentanil 20 μg kg^{-1}; group 4 = propofol 2.5 mg kg^{-1}, alfentanil 20 μg kg^{-1} and i.v. lignocaine 1 mg kg^{-1}. Lignocaine and alfentanil were placed in 5-ml syringes and 0.9% saline was added to make a volume of 5 ml. Patients in groups 1 and 3 received saline 5 ml in place of lignocaine. Saline or lignocaine was administered 2 min and alfentanil 1 min before induction of anaesthesia with propofol. All injections were given over a period of 20 s. Laryngoscopy and intubation were attempted after loss of eyelash reflex (No. 3 Macintosh laryngoscope blade, 8.0-mm Portex tracheal tube). The study was performed in a double-blind fashion, the anaesthetist performing the intubation (J.A.H.D. or J.A.G.) being unaware of the group to which the patient had been allocated. Ease of intubation was assessed on the basis of the scoring system devised by Helbo-Hansen, Ravlo and Trap-Anderson [8]. Degree of coughing, position and movement of the vocal cords, and ease of laryngoscopy were estimated on a scale of 1–4 (table

J. A. H. DAVIDSON*, M.B., CH.B., F.R.C.ANAES., Clinical Shock Study Group, Department of Anaesthesia, Western Infirmary, Dumbarton Road, Glasgow G11 6NT. J. A. GILLESPIE, M.B., CH.B., F.R.C.ANAES., Department of Anaesthesia, Glasgow Royal Infirmary, Castle Street, Glasgow G4 0SF. Accepted for Publication: July 28, 1992.

* Present address, for correspondence: University Department of Anaesthesia, Glasgow Royal Infirmary, Castle Street, Glasgow G4 0SF.

TABLE I. *Intubating condition score*

	Score			
	1	2	3	4
Laryngoscopy	Easy	Fair	Difficult	Impossible
Vocal cords	Open	Moving	Closing	Closed
Coughing	None	Slight	Moderate	Severe

TABLE II. *Patient data (mean (range or SD))*

	Group 1	Group 2	Group 3	Group 4
Age (yr)	32.4 (24–46)	29.7 (24–38)	30.9 (24–40)	31.87 (25–38)
Weight (kg)	60.6 (11.4)	67.9 (12.7)	61.8 (14.7)	61.1 (11.8)

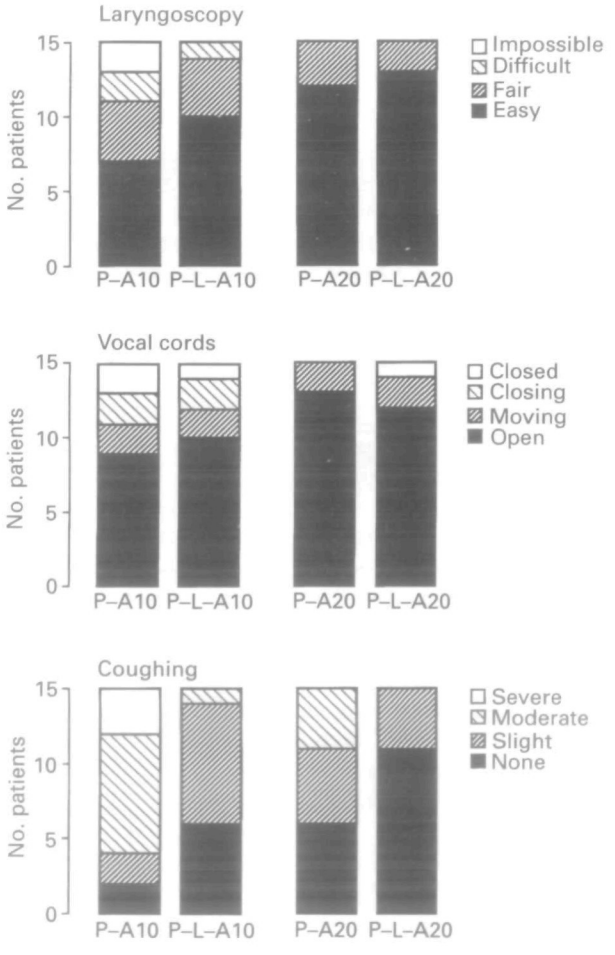

FIG. 1. Intubating condition score for laryngoscopy, vocal cords and coughing in patients from each group. P = Propofol; A10 = alfentanil 10 μg kg^{-1}; A20 = alfentanil 20 μg kg^{-1}; L = lignocaine.

I). Intubating conditions were judged acceptable when all scores were 2 or less. If any of the scores were 3 or 4, intubating conditions were judged unacceptable. Patients in whom intubation was impossible were given suxamethonium to optimize conditions.

After tracheal intubation, the lungs were ventilated with 67% nitrous oxide in oxygen 70 ml kg^{-1} min^{-1} (fresh gas flow) via a Mapleson D

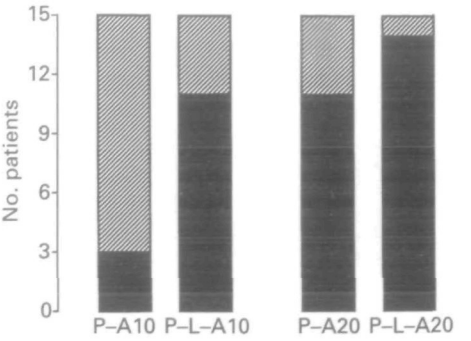

FIG. 2. Overall intubating conditions in patients from the four groups. All scores ⩽ 2 = acceptable intubating conditions (■); any score > 2 = unacceptable intubating conditions (▨). P = Propofol; A10 = alfentanil 10 μg kg^{-1}; A20 = alfentanil 20 μg kg^{-1}; L = lignocaine.

system, and after completion of the study period (2 min after intubation), inhalation agents were added to maintain anaesthesia.

Patients were monitored continuously by ECG and pulse oximetry. A baseline arterial pressure (Dinamap, Critikon) was measured before induction and was repeated immediately after both induction and intubation and thereafter at 1 and 2 min.

Statistics

The chi-square test and the Mann–Whitney U test were used for non-parametric data and the paired Student's t test and ANOVA for parametric data. The Bonferroni correction was used for inter- and intragroup comparisons. $P < 0.05$ was regarded as significant.

RESULTS

The four groups were comparable in age and weight (table II). There were no significant intergroup differences for baseline mean arterial pressure and heart rate. One patient commented spontaneously on tinnitus after injection of lignocaine. There was no other evidence of lignocaine toxicity.

Intubation was completed successfully in 56 of the 60 patients. In two patients in group 1, visualization of the larynx was impossible on laryngoscopy and in one patient from group 1 and one patient from group 2, the vocal cords were tightly closed, preventing intubation. After suxamethonium, intubation was achieved successfully in these four patients. Assessment of laryngoscopy, vocal cords and coughing are shown in figure 1. Vocal cord assessment was not greatly different between the four groups. Although ease of laryngoscopy improved with both the addition of i.v. lignocaine and the larger dose of alfentanil, the differences were not statistically significant. The incidence of coughing on insertion of the tracheal tube was reduced with lignocaine added to both the propofol–alfentanil 10 μg kg^{-1} ($P < 0.01$) and the propofol–alfentanil 20 μg kg^{-1} regimens.

The overall assessment of intubating conditions is shown in figure 2. Only three of 15 patients (20%) in group 1 were deemed to have satisfactory intubating conditions, compared with 11 of 15 patients (73%) in groups 2 and 3 and 14 of 15 patients (93%) in group 4. There was a statistically significant dif-

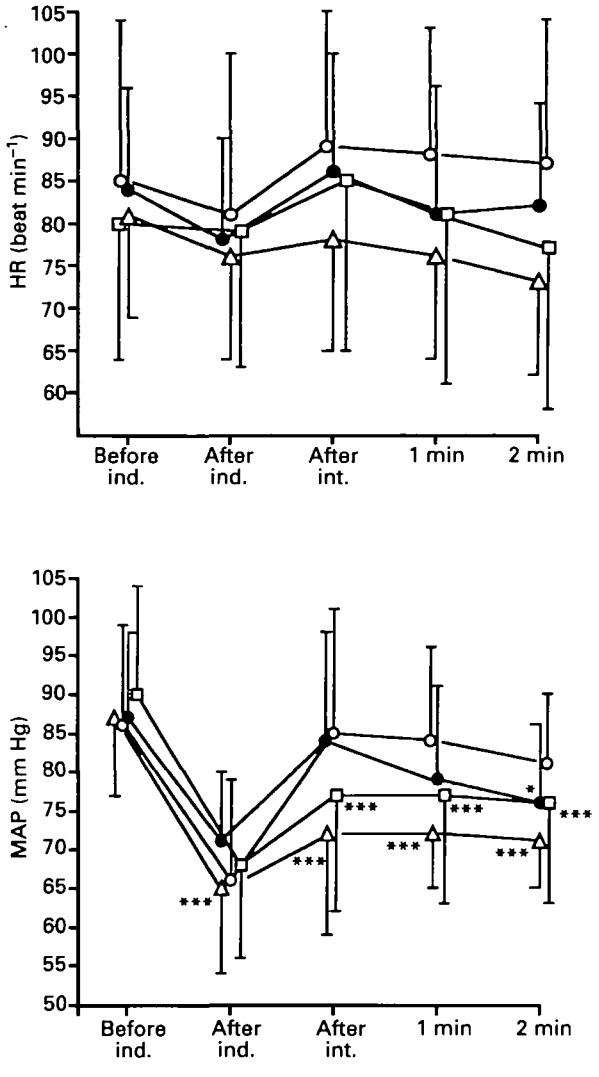

FIG. 3. Cardiovascular (heart rate (HR) and arterial pressure (MAP)) responses to laryngoscopy and intubation in the propofol–alfentanil 10 μg kg^{-1} (○), propofol–alfentanil 10 μg kg^{-1} with lignocaine (●), propofol–alfentanil 20 μg kg^{-1} (□) and propofol–alfentanil 20 μg kg^{-1} with lignocaine (△) groups. Within group statistically significant changes compared with the value before induction: *$P < 0.05$; ***$P < 0.001$. ind. = induction; int. = intubation.

ference ($P < 0.05$) between overall intubating conditions in group 1 (propofol–alfentanil 10 μg kg^{-1}) and group 3 (propofol–alfentanil 20 μg kg^{-1}). Addition of lignocaine to propofol–alfentanil 10 μg kg^{-1} resulted in an improvement in intubating conditions ($P < 0.05$). Similarly, supplementing propofol–alfentanil 20 μg kg^{-1} with lignocaine improved intubating conditions, although this did not achieve statistical significance.

Cardiovascular responses to induction and intubation are shown in figure 3. Heart rate decreased after induction and increased after intubation in all groups. However, none of the changes was statistically significant compared with baseline values. Similarly, mean arterial pressure decreased in all groups after induction ($P < 0.001$). However, there was an increase in all groups after intubation, but the mean arterial pressure remained depressed in groups 3 and 4 compared with pre-induction values ($P < 0.005$ and $P < 0.001$, respectively).

DISCUSSION

Several studies have concluded that intubation is possible without the use of neuromuscular blocking agents. Lewis demonstrated in 1948 that adequate conditions for intubation could be achieved using thiopentone alone [9]. McKeating and colleagues [10] found that, when no neuromuscular blocking drugs were given, laryngoscopy was possible significantly more often after propofol than after thiopentone. Conditions, however, are not always optimal. Keavney [1], using only propofol 2.5 mg kg^{-1}, reported satisfactory intubating conditions in 12 of 20 patients (60%), whereas Saarnivaara and Klemola [2], using a similar regimen, reported that only five of 13 patients (38%) had intubating conditions that were adequate. Even after administration of alfentanil 20 μg kg^{-1} 30 s before induction, in only 10 of 15 patients (66%) were intubating conditions deemed to have been adequate [2].

In this study, we have demonstrated that the use of i.v. lignocaine to supplement propofol–alfentanil anaesthesia improved intubating conditions. Lignocaine 1 mg kg^{-1} i.v., when administered 2 min before induction with propofol–alfentanil 10 μg kg^{-1}, increased the number of patients with acceptable intubating conditions from three of 15 (20%) to 11 of 15 (79%) ($P < 0.05$). The improvement in intubating conditions was largely caused by a reduction in the incidence and severity of coughing after insertion of the tracheal tube, although an improvement in the ease of laryngoscopy was also apparent.

There are many reports on the antitussive effects of i.v. lignocaine [11–13]. Lignocaine has also been shown to attenuate the increase in heart rate [13], arterial pressure [14] and intracranial pressure [15] associated with laryngoscopy and intubation. The mechanism whereby i.v. lignocaine suppresses the cough reflex is still unclear, but the efficacy has been shown to increase in a dose-dependent manner, and correlates well with plasma concentrations [13]. Yukioka and colleagues demonstrated that administration of lignocaine 2 mg kg^{-1} was effective in blocking the cough reflex during intubation in patients in whom anaesthesia was induced and maintained with nitrous oxide and halothane in oxygen, coughing being effectively eliminated by plasma concentrations of lignocaine in excess of 3 μg ml^{-1} [13]. It was planned initially to use a 2-mg kg^{-1} dose of i.v. lignocaine in this study; however, after a small pilot study in which five of six patients complained spontaneously of tinnitus, circumoral paraesthesia and dizziness, the dose was reduced to 1 mg kg^{-1}. This dose significantly reduces the incidence of coughing [13] and, as we have demonstrated, was associated with minimal side effects.

It is likely that the antitussive effect of lignocaine is caused at least partially by an increase in the depth of general anaesthesia. Indeed, blood concentrations of 3–6 μg ml^{-1} are known to potentiate the effects of nitrous oxide anaesthesia with a 10–28% reduction in the minimum alveolar concentration (MAC) of halothane [16].

REFERENCES

1. Keaveny JP, Knell PJ. Intubation under induction doses of propofol. *Anaesthesia* 1988; 43S: 80–81.
2. Saarnivaara L, Klemola UM. Injection pain, intubating conditions and cardiovascular changes following induction of anaesthesia with propofol alone or in combination with alfentanil. *Acta Anaesthesiologica Scandinavica* 1991; 35: 19–23.
3. Bedford RF, Marshall WK. Cardiovascular responses to endotracheal intubation during four anaesthetic techniques. *Acta Anaesthesiologica Scandinavica* 1984; 28: 563–566.
4. Ebert JP, Pearson JD, Gelman S, Harris C, Bradley EL. Circulatory responses to laryngoscopy: the comparative effects of placebo, fentanyl and esmolol. *Canadian Journal of Anaesthesia* 1989; 36: 301–306.
5. Black TE, Kay B, Healy TEJ. Reducing the haemodynamic responses to laryngoscopy and intubation. *Anaesthesia* 1984; 39: 883–887.
6. Abou-Madi MN, Keszler H, Yacoub JM. Cardiovascular reactions to laryngoscopy and intubation following small and large intravenous doses of lidocaine. *Canadian Anaesthetists Society Journal* 1977; 24: 12–19.
7. Mallampatti SR, Gatt SP, Gugino LD, Desai SP, Waraksa B, Freiberger D, Liu PL. A clinical sign to predict difficult tracheal intubation: a prospective study. *Canadian Anaesthetists Society Journal* 1985; 32: 429–434.
8. Helbo-Hansen S, Ravlo O, Trap-Anderson S. The influence of alfentanil on the intubating conditions after priming with vecuronium. *Acta Anaesthesiologica Scandinavica* 1988; 32: 41–44.
9. Lewis CB. Endotracheal intubation under thiopentone. *Anaesthesia* 1948; 3: 113–115.
10. McKeating K, Bali IM, Dundee JW. The effects of thiopentone and propofol on upper airway integrity. *Anaesthesia* 1988; 43: 638–640.
11. Stenhause JE, Gaskin L. A study of intravenous lidocaine as a suppressant of cough reflex. *Anesthesiology* 1963; 24: 285–290.
12. Poulton TJ, James FM. Cough suppression by lidocaine. *Anesthesiology* 1979; 50: 470–472.
13. Yukioka H, Yoshimoto N, Nishimura K, Fujimori M. Intravenous lidocaine as a suppressant of coughing during tracheal intubation. *Anesthesia and Analgesia* 1985; 64: 1189–1192.
14. Abou-Madi MN, Keszler H, Yacoub JM. Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. *Canadian Anaesthetists Society Journal* 1977; 24: 2–19.
15. Hammill JF, Bedford RF, Weaver DC, Colohan AR. Lidocaine before endotracheal intubation: Intravenous or laryngotracheal? *Anesthesiology* 1981; 55: 578–581.
16. Himes RS, DiFazio CH, Burney RG. Effects of lidocaine on the anesthetic requirements for nitrous oxide and halothane. *Anesthesiology* 1977; 47: 437–440.