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Cardiovascular changes with the laryngeal mask airway in cardiac anaesthesia

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Background. The laryngeal mask airway (LMA) causes fewer haemodynamic changes, particularly in mean arterial pressure (MAP) and heart rate (HR), than tracheal intubation using either laryngoscopy or the intubating LMA. There are no data for patients with coronary artery disease.

Method. We studied 27 patients having coronary artery bypass grafting, prospectively randomized to be managed with either the LMA or tracheal intubation using either laryngoscopy or the ILMA. We used invasive monitoring to compare the haemodynamic effects in each group during induction and emergence from anaesthesia.

Results. Both methods of intubation caused an increase in MAP compared with the LMA ($P<0.05$). Mixed venous oxygen saturation increased in the intubated patients but not with the LMA ($P<0.05$). HR did not change at induction in the LMA group. Changes at extubation were similar in all groups but cardiac index was lower in the LMA group ($P<0.05$).

Conclusion. The LMA allows airway management without hypertension and tachycardia and should be considered when anaesthetizing patients with coronary disease.

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The laryngeal mask airway (LMA†) and intubating ILMA (Intavent, Berkshire, UK) provide a hands-free airway.[1] Insertion of the LMA, but not the ILMA (Intavent), has fewer haemodynamic effects than tracheal intubation.[2 3]

There are no reports of the effects in patients with coronary artery disease. In a pilot study, we used the LMA in 22 patients undergoing coronary artery bypass grafting (CABG). One patient required intubation after cardiopulmonary bypass because mechanical ventilation was difficult. We found that giving more muscle relaxant before restarting ventilation seemed to solve this difficulty. Sevoflurane was used for induction of anaesthesia after a previous study of this method for CABG.[4]

Methods and results

The study had ethical committee approval. After obtaining written consent, we used sealed envelopes to allocate patients randomly to groups to be managed by laryngoscopy and tracheal intubation (TT group n=9), tracheal intubation via the ILMA (ILMA group n=9) or the LMA (LMA group n=9). We excluded patients who were obese or who had chronic obstructive pulmonary disease, gastric reflux or if transoesophageal echocardiography was planned. In one LMA patient the planned surgery was altered and this patient was omitted from the study.

Medications were continued until the morning of surgery, except angiotensin-converting enzyme inhibitors. Premedication was with temazepam 10–20 mg, plus papaveretum and hyoscine 0.1 ml 10 kg^{-1} i.m. Monitoring was started with the patient awake, using an oximetric pulmonary artery catheter (Abbott Opticath 7.5F, Chicago, USA), arterial cannula, pulse oximeter and five-lead ECG. We recorded heart rate (HR), mean arterial pressure (MAP), mixed venous oxygen saturation and cardiac index, and we calculated the systemic vascular resistance index and pulmonary vascular resistance index, using an Abbott Oximetrix 3, Cardiac Output Computer (Chicago, USA). We used ST-segment analysis (leads II and V) using a

†LMA® is the property of Intavent Limited.

Table 1 Haemodynamic data. *Within-group difference, $P<0.05$; †between-group difference, $P<0.05$. Values are mean (SD). TT, tracheal tube; LMA, laryngeal mask airway; ILMA, intubating LMA; panc, pancuronium

		TT group	ILMA group	LMA group
Heart rate (beats min^{-1})	Awake	63 (19)	56 (11)	61 (12)
	Panc + 3 min	59 (15)	53 (11)	60 (11)
	After intubation	68 (17)	61 (12)	60 (10)
	At recovery	80 (20)	76 (14)	76 (12)
	After extubation	85 (20)	77 (15)	78 (16)
Mean arterial pressure (mm Hg)	Awake	100 (16)	96 (12)	98 (13)
	Panc + 3 min	65 (13)*	70 (13)*	78 (20)
	After intubation	89 (26)*	91 (15)*	75 (18)
	At recovery	90 (9)	104 (20)	93 (18)
	After extubation	92 (6)	102 (23)	90 (11)
Mixed venous oxygen saturation	Awake	72 (6)	70 (7)	73 (4)
	Panc + 3 min	79 (6)*	79 (9)*	81 (5)
	After intubation	85 (5)*	88 (6)*	83 (4)
	At recovery	70 (7)	70 (9)	69 (9)
	After extubation	71 (7)	67 (5)	66 (80
Cardiac index (litre min^{-1} m^{-2})	Awake	2.7 (0.6)	2.4 (0.4)	2.6 (0.6)
	Panc + 3 min	1.7 (0.4)	1.9 (0.3)*	2.1 (0.5)
	After intubation	2.2 (0.5)	2.3 (0.3)*	2.2 (0.5)
	At recovery	3.1 (0.8)†	2.9 (0.9)	2.1 (0.4)†
	After extubation	3.1 (0.8)†	3.0 (0.7)	2.3 (0.3)†

Dynascope (Fukuda, Tokyo, Japan) and used standard criteria for myocardial ischaemia.[5]

Measurements were made at the following times: before the induction of anaesthesia; 3 min after giving pancuronium; 1 min after insertion of the airway; during recovery, breathing spontaneously via the airway; 1 min after removal of the airway.

If MAP was less than 50 mm Hg or greater than 110 mm Hg, or ECG signs of myocardial ischaemia were noted then drug treatment would be used.

Anaesthesia was induced with midazolam 1–2 mg and fentanyl 3 μg kg^{-1} i.v., followed by sevoflurane 4% in oxygen. Pancuronium 8 mg was given followed by manual ventilation for 3 min with sevoflurane 2% before the second set of measurements was recorded. Each group then had their airway inserted. In the LMA group, a wet pack was inserted around the mask without using a laryngoscope. The third readings were taken 1 min after airway insertion. Anaesthesia was continued with air/oxygen with sevoflurane, and further fentanyl, 7 μg kg^{-1} and midazolam, 1 mg. Atracurium was given if required. At chest closure, frusemide 10 mg, neostigmine 2.5 mg and glycopyrrolate 0.5 mg were given i.v., and the sevoflurane discontinued.

Patients were allowed to recover undisturbed and unsedated. When spontaneous respiratory effort started all patients were allowed to breathe oxygen. In the ILMA group, the tracheal tube was removed. The fourth set of readings were taken. If the patient was cooperative, the Pa_{O_2} was greater than 8.0 kPa with an FI_{O_2} of 0.4, the core temperature above 36.0 °C and blood loss less than 50 ml h^{-1}, then the tracheal tube/ILMA/LMA was removed, and a final set of readings was taken.

Statistical analysis was by repeated-measures ANOVA. Differences were considered significant if $P<0.05$. Considering MAP and HR (where differences of 15 mm Hg and 10 beats min^{-1} had been seen in our pilot study), a group size of 27 patients would have had an 80% power to detect a difference between the groups at a 5% significance level.

Patient characteristics in the three groups were comparable. The haemodynamic values are shown in Table 1. MAP increased at intubation in the TT and ILMA groups but not in the LMA group ($P<0.05$). HR increased in the TT and ILMA groups only, but these changes were not significant. No myocardial ischaemia was seen during induction or recovery.

Use of additional medication was similar in all groups, except that more atracurium was given to patients in the LMA group to facilitate restarting ventilation after bypass. Length of operation, time to eye opening (average TT=27 min, ILMA=38 min, LMA=31 min) and removal of airway (average TT=94 min, ILMA=66 min, LMA=79 min), were similar between groups. One TT patient required sedation for bleeding. Two ILMA patients required sedation, one for bleeding and one for insertion of a chest drain. These patients were excluded from the final set of readings. All patients made a full recovery.

Comment

With the LMA, HR and MAP were stable whereas the ILMA caused increases similar to those seen with laryngoscopy and intubation.

Circulatory disturbances can cause myocardial ischaemia and postoperative myocardial infarction.[6] Sevoflurane is a safe primary anaesthetic agent, with early recovery when used with low-dose opiates in CABG.[7]

Recovery was prompt and removal of the tracheal tube/ILMA/LMA was not associated with haemodynamic changes, suggesting that this is less stressful than airway insertion. On the second day after operation, none of the patients could recall discomfort with any type of airway.

Although we do not necessarily recommend use of the LMA for cardiac surgery, we have shown that cardiovascular stability can be achieved when the LMA is used, without need for pharmacological techniques to control the circulation in patients with ischaemic heart disease.

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