Effect of a remifentanil bolus dose on the cardiovascular response to emergence from anaesthesia and tracheal extubation†

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We have examined the effect of remifentanil on the haemodynamic response to emergence from anaesthesia and tracheal extubation in 40 ASA I–II female patients undergoing diagnostic laparoscopy, in a randomized, double-blind study. All patients received a standard general anaesthetic comprising propofol, vecuronium and 1% isoflurane with 66% nitrous oxide in oxygen. At the end of surgery, a bolus dose of remifentanil 1 µg kg⁻¹ (n = 20) or saline placebo (n = 20) was given and tracheal extubation was performed when standard criteria were achieved. Arterial pressure and heart rate were recorded non-invasively at 1-min intervals from the end of surgery. Remifentanil attenuated the increase in both mean arterial pressure (P < 0.001) and heart rate (P < 0.05) at extubation. Mean time to extubation was 7.2 (SEM 0.6) min and 4.0 (0.5) min in the remifentanil and saline groups, respectively (P < 0.001). There was no difference in the incidence of coughing at extubation, time to recovery from anaesthesia or time to fitness for discharge from the recovery room.

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Emergence from anaesthesia and tracheal extubation may be associated with increases in arterial pressure, heart rate, plasma concentrations of catecholamine, intracranial pressure and development of myocardial ischaemia in susceptible individuals.¹⁻³ These responses may be attenuated by i.v. opioids, vasodilators, β-blockers, local anaesthetics or by extubation under deep anaesthesia.³ The action of agents used at extubation should be brief to allow rapid return of protective reflexes and avoid residual hypotension, respiratory depression or sedation. The pharmacokinetic properties of remifentanil result in a rapid onset and offset of action,⁴ and it has been shown to attenuate cardiovascular responses to otracheal intubation.⁵ In this study, we have assessed the effect of remifentanil on the cardiovascular responses to, and timing of, emergence from anaesthesia and tracheal extubation.

Methods and results

After obtaining approval from the Local Research Ethics Committee and written informed consent, we studied 40 ASA I–II female patients, aged 18–50 yr, presenting for elective diagnostic gynaecological laparoscopy. Exclusion criteria included a history of hypertension, asthma, hypersensitivity to NSAID and predicted difficulty in intubation or airway maintenance. Patients were allocated randomly to one of two groups in a double-blind manner.

All patients received a standard general anaesthetic comprising propofol 2–3 mg kg⁻¹ (to loss of verbal contact) and fentanyl 1.5 µg kg⁻¹. Vecuronium 0.1 mg kg⁻¹ was given to facilitate tracheal intubation. After induction of anaesthesia, all patients received diclofenac 100 mg rectally. Anaesthesia was maintained with isoflurane and 66% nitrous oxide in oxygen using mechanical ventilation with a Datex MCM 980 ventilator (tidal volume 10 ml kg⁻¹). End-tidal carbon dioxide partial pressure was maintained at 4.5 kPa (Datex Capnomac) by adjustment of ventilatory frequency. Arterial pressure was measured non-invasively by an automatic oscillometric device (Datex Cardiocap), and heart rate was recorded from the ECG trace. Baseline mean arterial pressure (MAP) was obtained from the mean of three resting values in the anaesthetic room before any instrumentation. MAP during surgery was controlled to within 10% of resting preoperative baseline by titration of inspired isoflurane concentration.

At the end of surgery, wounds were infiltrated with 0.5% bupivacaine 10 ml. The time of the last suture was designated ‘time zero’. At this point the patient received remifentanil 1 µg kg⁻¹ or an equivalent volume of saline over 30 s. This was followed by neostigmine 2.5 mg with glycopyrrolate.

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0.5 mg to antagonize the residual effects of the non-depolarizing neuromuscular blocking agent. Mechanical ventilation was continued with 100% oxygen, and after 2 min, oropharyngeal suction was performed using a Yankauer sucker. Extubation was performed in a standard manner when patients were able to open their eyes and squeeze a hand on command. Extubation time was defined as time between drug administration (time zero) and extubation. Heart rate (HR), systolic (SAP) and diastolic (DAP) arterial pressures, and MAP were recorded every 1 min from time zero to 5 min after extubation. The incidence of coughing or gagging at extubation was noted. Sedation score (1=alert and responsive, 2=drowsy but responsive to verbal command and 3=unresponsive to verbal command) and ventilatory frequency were assessed by nurses on arrival in the recovery area and at 15-min intervals thereafter. The incidence of nausea and fitness for ward transfer were also determined by recovery nurses.

Statistical analysis was performed using multivariate analysis of variance for repeated measures (MANOVA with treatment group and time as the between- and within-group factors), paired and unpaired t tests and Mann–Whitney tests as appropriate, using SPSS for Windows computer software (release 6.0, 1993).

Patient characteristics were similar in the two groups. Mean age and weight were 32.7 (range 23–43) yr and 60.6 (SEM 2.1) kg in the remifentanil group and 32.0 (18–50) yr and 63.6 (2.8) kg in the placebo group, respectively. There were no significant differences in baseline MAP and HR between groups before or at the end of surgery (time zero) (Table 1). Within-group changes in MAP and HR in the remifentanil group at extubation were not significant. MAP in the saline group increased significantly at extubation and for 3 min after extubation (P<0.005 within-group); this was attenuated in the remifentanil group (P<0.001 between groups). HR increased significantly (P<0.05) for 4 min after extubation and was significantly greater in the placebo group compared with the remifentanil group (P<0.05). SAP and DAP changed in parallel with MAP and have not been reported separately.

Mean time to extubation was 7.2 min in the remifentanil group and 4.0 min in the saline group (P<0.001, Mann–Whitney test). Coughing or gagging at tracheal extubation was rated as ‘none or minimal’ in 11 patients in the remifentanil group and in nine patients in the saline group.

The incidence of drowsiness on arrival in the recovery room was 10 of 20 patients in the saline group compared with three of 20 in the remifentanil group (P=0.056), although times to discharge were similar (mean times 35.5 (SD 14.5) min and 33.7 (13.7) min in the remifentanil and saline groups, respectively). Two patients in the remifentanil group and one in the saline group reported nausea, but there was no vomiting or respiratory depression in any patient.

Discussion

Remifentanil 1 µg kg⁻¹, when given as a slow i.v. bolus at the end of surgery, attenuated the increase in MAP and HR associated with emergence from anaesthesia and tracheal extubation. The time at which extubation was possible was delayed after remifentanil, although this delay was not considered clinically significant. The greater tendency towards sedation in the saline group may reflect the shorter time between extubation and arrival in the recovery room, but there was no difference between groups in nausea and vomiting or time to discharge from the recovery room. The increase in MAP and HR at tracheal extubation in the saline group is consistent with previous data. There was no bradycardia or hypotension associated with remifentanil, although this has been reported previously at induction of anaesthesia and intubation. This may be a result of co-administration of glycopyrrolate with neostigmine, and also the effect of diminishing anaesthetic concentration at emergence from anaesthesia.

Remifentanil may be a useful agent to suppress the cardiovascular responses to extubation without compromising recovery from anaesthesia. It may also be suitable for attenuation of responses to other brief but noxious stimuli in patients undergoing short procedures under anaesthesia, for example manipulation of fractures or electroconvulsive therapy. Other applications may include use in patients in intensive care at risk of neurological or cardiovascular disease, before tracheal extubation, tracheal toilet or physiotherapy.

References


