Haemodynamic responses to extubation after cardiac surgery with and without continued sedation

J. CONTI, D. SMITH

Summary
We studied the haemodynamic response to cessation of mechanical ventilation and removal of the tracheal tube in 84 patients after coronary artery bypass grafting. Patients were sedated on the ICU with propofol 1–3 mg kg\(^{-1}\) h\(^{-1}\), and randomly allocated to extubation while awake or while still sedated. Systolic and diastolic blood pressures and heart rate increased significantly faster in the awake group as mechanical ventilation was stopped; systolic blood pressure 6.1 (3.0) vs 0.7 (1.8) mm Hg min\(^{-1}\), diastolic blood pressure 2.1 (1.6) vs 0.2 (0.9) mm Hg min\(^{-1}\), heart rate 2.1 (1.7) vs 0.2 (0.5) beats min\(^{-2}\); \(P < 0.01\) in each case. Treatment was required for systolic hypertension during discontinuation of mechanical ventilation in 20 patients (53%) in the awake group and in three patients (7.5%) in the sedated group (\(P < 0.001\)). No patient in the sedated group had any new ischaemic ECG changes. Significant new ST segment changes did not occur in the sedated group but were present in five patients in the awake group (\(P = 0.013\)), one of whom suffered a perioperative myocardial infarction. Removal of the tracheal tube while patients are still sedated after coronary artery bypass grafting is safe, and reduces the incidences of haemodynamic disturbance and myocardial ischaemia during extubation. (Br. J. Anaesth. 1998; 80: 834–836)

Keywords: intubation tracheal; extubation; cardiovascular responses; surgery cardiovascular

Avoidance of tachycardia and hypertension after coronary artery bypass graft (CABG) surgery is important for reducing postoperative morbidity and mortality.\(^1\) After cardiac surgery, mechanical ventilation is usually discontinued and the tracheal tube removed when the patient is awake,\(^2\)\(^3\) producing significant tachycardia and hypertension.\(^4\)\(^5\) Esmolol may ablate these haemodynamic changes,\(^6\) but at the dose required may have serious cardiovascular side effects. We investigated if removing the tracheal tube while patients are still sedated after CABG could safely reduce the haemodynamic response to extubation.

Methods and results
After obtaining from the Southampton Joint Ethics Committee and informed written consent, we studied 84 consecutive patients under-going CABG over a 2-month period. All regular medications, except angiotensin-converting-enzyme inhibitors, diuretics and digoxin, were continued until the morning of surgery. Premedication was with oral lorazepam 1–3 mg 2 h before induction of anaesthesia, then morphine 5–10 mg and hyoscine 0.1–0.4 mg i.m. 1 h later.

Anaesthesia was induced with fentanyl 10 \(\mu\)g kg\(^{-1}\) midazolam 2–5 mg or thiopentone 1–1.5 mg kg\(^{-1}\), and pancuronium, and maintained with fentanyl to a total of 30–35 \(\mu\)g kg\(^{-1}\) and an infusion of propofol 1–3 mg kg\(^{-1}\) h\(^{-1}\). Midazolam 4 mg was given at the start of cardiopulmonary bypass. A hypothermic bypass technique, with membrane oxygenator (Dideco D703) and arterial line filter, was used in all patients. Neuromuscular block was not antagonized at the end of surgery, and glyceryl trinitrate (GTN) 0.5 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) was infused as a coronary vasodilator in most patients.

After surgery, patients were transferred to the cardiothoracic ICU where their lungs were ventilated, using a synchronized intermittent mandatory ventilation mode (Engstrom Erica) with 10 cm H\(_2\)O inspiratory assist. Systolic, diastolic and mean arterial blood pressures, central venous pressure, heart rate and nasopharyngeal temperature data were stored in the ICU monitors (PPG Hellige SMU612 or Mennen Horizon XL). A standard 5-lead ECG was used, with continuous computerized ST segment analysis in leads II and V5. We defined greater than 0.2 mV of new ST segment depression or greater than 0.3 mV of new ST segment elevation 60 ms after the J-point as a significant change.

On admission to the ICU patients were randomly allocated by a computer-generated sequence to removal of the tracheal tube while awake or while still sedated. During mechanical ventilation sedation was maintained with propofol 1–3 mg kg\(^{-1}\) h\(^{-1}\). Adequate pain control was ensured with bolus doses of morphine 1–2 mg i.v. if required. Unless contraindicated by poor renal function or a history of gastrointestinal bleeding, patients received rectal didocifenc 100 mg and domperidone 30 mg at least 1 h before extubation. Systolic blood pressure was maintained at 90–130 mm Hg with infusions of GTN or inotropic agents as appropriate.

Correspondence to D. S.
Before discontinuing mechanical ventilation, the $P_{1.5}$ was adjusted to 4.5–5.5 kPa. The tracheal tube was removed when patients were warm (nasopharyngeal temperature $>36^\circ$C), haemodynamically stable, with minimal blood loss and a $P_{1.5}$ of $>9.0$ kPa on an $R_{1.5} < 0.4$. The propofol infusion was then adjusted to just obtrude the eyelash reflex and each patient was allowed to stabilize at this level for a minimum of 10 min.

For patients in the awake group, the propofol infusion was stopped and patients allowed to wake up. During this time they were allowed to breathe via a Water’s circuit giving manual assistance as necessary. The tracheal tube was removed when spontaneous respiratory efforts and gas exchange were satisfactory and patients were awake.

For those in the sedated group, the propofol infusion was continued while mechanical ventilation was stopped and the patients allowed to breathe via a Water’s circuit giving manual assistance as necessary. When spontaneous respiratory efforts and gas exchange were satisfactory, the tracheal tube was removed and the propofol infusion stopped.

Just before extubation, the nasogastric tube was suctioned and removed. The mouth was cleared of secretions and a suction catheter passed down the tracheal tube, which was removed with the suction catheter in situ. Arterial blood gas analysis was performed before cessation of mechanical ventilation and 20 min after extubation.

Hypertension was treated by increasing the infusion rate of GTN. If the systolic blood pressure was $>150$ mm Hg a bolus dose of GTN 1 mg was given, and the infusion rate of GTN further increased. A second bolus of GTN was given if required 2 min later. We looked for changes from a baseline value taken 10 min before cessation of mechanical ventilation in the heart rate, systolic (SBP) and diastolic (DBP) blood pressures. Data were analysed by linear regression to measure the rate of increase of all haemodynamic variables (heart rate, SBP, DBP) from cessation of mechanical ventilation until removal of the tracheal tube, to take into consideration the different duration of recovery of spontaneous ventilation for individual patients. An unequal variance $t$-test was then performed using SPSS for Windows (version 6.1). Baseline corrected $t$-testing was performed at set time points as used previously, with statistical significance assumed at $P < 0.01$. Non-parametric data were analysed using the chi-square test, assuming statistical significance at $P < 0.05$.

Data were collected on 79 of the 84 patients who were enrolled in the study. Five patients were excluded because of major surgical or postoperative complications. One further patient in the awake group was excluded because the propofol was stopped several hours before extubation and the patient was extremely slow to waken. A total of 38 patients were managed according to the study procedure in the awake group and 40 patients in the sedated group.

There were no differences between the two groups in patient characteristics, including age, body mass index, history of hypertension, left ventricular ejection fraction, previous myocardial infarction, preoperative drug history and mean time to removal of the tracheal tube (441 min vs 448 min, overall range 175–1173 min). One patient in the awake group received morphine before removal of the tracheal tube, but no patient woke with severe pain.

Mean (range) infusion rates of propofol before weaning were $1.84$ (0–3.2) mg kg$^{-1}$ h$^{-1}$ for patients in the awake group and $1.64$ (0.5–3.0) mg kg$^{-1}$ h$^{-1}$ for patients in the sedated group. The mean (range) duration of weaning was $7.44$ (5–18) min for patients in the awake group and $7.25$ (5–24) min for patients in the sedated group. For patients in the awake group, the mean time from stopping the propofol infusion to extubation was $14.1$ (6–38) min. There were no significant differences between groups in blood gas analysis before or after extubation; the mean (range) arterial $P_{O_2}$ after removal of the tracheal tube was $5.3$ (4.1–6.5) kPa for patients extubated awake and $5.4$ (4.2–7.1) kPa for patients extubated while sedated.

Haemodynamic data during cessation of mechanical ventilation and extubation are illustrated in fig.1. There were no significant differences between the groups in baseline values for any haemodynamic variable. Systolic and diastolic blood pressures and heart rate increased significantly faster in the awake group during cessation of mechanical ventilation: systolic blood pressure $6.1$ (3.0) vs $0.7$ (1.8) mm Hg

---

Figure 1 Changes in systolic blood pressure (circles), diastolic blood pressure (squares) and heart rate (diamonds) during cessation of mechanical ventilation and removal of the tracheal tube while sedated or awake. Time (min) is from cessation of mechanical ventilation or removal of the tracheal tube; B = baseline measurement before cessation of mechanical ventilation or removal of the tracheal tube; E = extubation.
min⁻¹, diastolic blood pressure 2.1 (1.6) vs 0.2 (0.9) mm Hg min⁻¹, heart rate 2.1 (1.7) vs 0.2 (0.5) beats min⁻¹; P < 0.01 in each case. Not all patients in the awake group had haemodynamic changes associated with cessation of mechanical ventilation, but two patients developed ischaemic ECG changes with only modest increases in heart rate and systolic pressure. After removal of the tracheal tube, there was a further non-significant increase in all haemodynamic variables in both groups; the differences from baseline for all variables except diastolic blood pressure (P = 0.11) were still significant at 10 min after extubation.

No patients required treatment for systolic hypertension before mechanical ventilation was stopped. However, treatment was required during cessation of mechanical ventilation in 20 patients (53%) in the awake group and in three patients (7.5%) in the sedated group (P < 0.001). Long-term β-blocker therapy before operation had no influence on the need for antihypertensive treatment.

The ST segment was interpretable in 26 of the 38 patients in the awake group and 28 of the 40 patients in the sedated group. No patient in the sedated group had any new ischaemic ECG changes. Significant new ST segment changes occurred in five patients in the awake group (P = 0.013) and lasted for 1–5 min; in one of these patients there was already ECG evidence of ischaemia before mechanical ventilation was stopped. In all five patients, the ST segment changes were associated with significant increases in heart rate or systolic pressure or both in three cases to >40% above baseline. One patient with new ischaemic changes suffered a perioperative myocardial infarction.

Comment

The incidences of haemodynamic disturbance and new ischaemic ECG changes in patients in whom mechanical ventilation was stopped and the tracheal tube removed while they were awake are in keeping with those in other studies of patients undergoing cardiac surgery; both incidences are markedly reduced by removing the tracheal tube while patients are sedated. We encountered no problems with the airway or respiration in the sedated group, and no patients required reinsertion of the tracheal tube. The low use of opioids and incidence of pain on waking from propofol infusion in patients undergoing cardiac surgery has been commented on before. Several authorities state that patients should awake before extubation so that their neurological status can be assessed. However, we have not found this to be necessary; waking while intubated is stressful and has risks other than haemodynamic compromise.

Acknowledgements

We thank the nursing staff of the Wessex Cardiothoracic Unit, Professor M. J. Campbell and Mr M. Mullee of the Department of Medical Statistics and Computing, University of Southampton, and Viamed plc.

References