CARDIOVASCULAR AND PLASMA CATECHOLAMINE RESPONSES AT TRACHEAL EXTRUBATION

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SUMMARY

The haemodynamic responses to tracheal extubation at the end of surgery were compared with those occurring at tracheal intubation in 12 patients undergoing major elective surgery. Arterial cannulation was performed and heart rate (HR), systolic arterial pressure (SAP) and diastolic arterial pressure (DAP) were measured before induction of anaesthesia, before tracheal intubation, at the end of surgery and 1, 3 and 5 min after tracheal extubation. Laryngoscopy was avoided at the end of surgery. At all but the first of these stages, venous blood was obtained for measurement of plasma concentrations of adrenaline and noradrenaline. Rate-pressure product (RPP) was derived from SAP x HR. After tracheal intubation there were significant (P < 0.05) increases in HR, DAP, RPP and in plasma concentrations of both adrenaline and noradrenaline. After extubation, only HR and adrenaline concentration at 5 min after extubation increased significantly compared with measurements at the end of surgery.

KEY WORDS


It is well known that, after tracheal intubation, there are increases in arterial pressure and heart rate associated with an increase in plasma concentrations of both noradrenaline and adrenaline [1,2]. There is a correlation between the magnitude of the pressor response and the increase in the plasma concentrations of noradrenaline. The changes in catecholamine concentrations occur rapidly and last for only about 5 min [1]. An increase in heart rate and arterial pressure also occurs after tracheal extubation [3], but there are no studies to date on corresponding changes in plasma catecholamine concentrations.

PATIENTS AND METHODS

After obtaining Ethics Committee approval and informed patient consent, we studied 12 patients undergoing major elective surgery, for whom radial artery cannulation would be considered routine monitoring.

Patients with ischaemic heart disease, hypertension, heart failure or a history of cerebrovascular accident, atopy or allergy and those taking mono-amine oxidase inhibitors were excluded from the study.

Premedication comprised oral diazepam 10–20 mg, 2 h before surgery. In the anaesthetic room, a venous cannula and radial artery cannula were inserted under local anaesthesia. The ECG was displayed continuously using the CM5 lead and systemic arterial pressure was measured using a Gould P23XL transducer. Neuromuscular block was monitored using a Datex relaxograph. End-expired carbon dioxide was measured with a Datex infra-red carbon dioxide analyser.

After a 10-min stabilization period, baseline systolic arterial pressure (SAP), diastolic arterial pressure (DAP) and heart rate (HR) were recorded.

Anaesthesia was induced using thiopentone 4–5 mg kg⁻¹ given over 60 s and, after baseline calibration of the relaxograph, vecuronium 0.1 mg kg⁻¹ and morphine 0.15 mg kg⁻¹ were administered i.v. Ventilation was controlled manually with 33% oxygen in nitrous oxide using a Bain coaxial breathing system and a fresh gas flow sufficient to maintain an end-expired Pco₂ of 5 kPa.

One minute after induction of anaesthesia, HR, SAP and DAP were recorded and a 10-ml sample of arterial blood was obtained for measurement of plasma catecholamine concentrations. Two minutes after induction or when the first twitch (T1) of a train-of-four was less than or equal to 20% of baseline if this had not yet occurred, the patient's trachea was intubated. Further recordings of HR, SAP and DAP were made and a second blood sample obtained 1 min after tracheal extubation.

Following intubation, anaesthesia was maintained using 1 MAC enflurane and 66% nitrous oxide in oxygen. Supplements of vecuronium and morphine were given as necessary by the anaesthetist, who was not one of the investigators. End-expired Pco₂ was maintained at 4–5 kPa.

Enflurane was discontinued at the beginning of skin closure and, when surgery was complete (all
surgery being hip replacement surgery performed by the same surgeon), a further blood sample was taken and recordings of SAP and HR made. Residual neuromuscular block was antagonized with neostigmine 2.5 mg and glycopyrremonium 0.5 mg. Pharyngeal suction was carried out without laryngoscopy using a 10-French gauge catheter. End-expired carbon dioxide was maintained at 5 kPa with manual ventilation using 100% oxygen. The tracheal tube was removed smoothly when the train-of-four ratio was 70% and after spontaneous ventilation had returned.

After tracheal extubation, 100% oxygen was administered via a face mask. Blood samples were taken and recordings of HR and SAP were made at 1, 3 and 5 min after tracheal extubation. Arterial blood samples were obtained 1 min after extubation from eight of the 12 patients for gas analysis using an Instrumentation Laboratory 1302 blood-gas analyser.

Blood for measurement of the catecholamine concentrations was collected into "Vacutainer" tubes containing lithium heparin; the samples were centrifuged and the separated plasma stored at −70 °C until analysis was performed using high pressure liquid chromatography [1].

Statistical analysis of the results was performed using Student's paired t tests, two-way analysis of variance and Bonferroni P < 0.05 testing.

We studied 12 patients (five male). Most patients were undergoing revision hip replacement. Their ages were from 44 to 70 yr and weights 44–102 kg (table I).

There was a significant increase in HR 1 min after intubation (P < 0.05) and significant increases in DAP (P < 0.05), rate–pressure product (P < 0.05) and plasma concentrations of noradrenaline (P < 0.05) and adrenaline (P < 0.05) (table II).

HR increased significantly at 1 min and 3 min after tracheal extubation compared with recordings made at the end of surgery (P < 0.05). There were no changes in SAP or DAP after tracheal extubation compared with the end of surgery (P < 0.05).

There were no significant changes in noradrenaline concentrations after tracheal extubation, but adrenaline concentrations increased with time and were significantly so 5 min after the end of surgery (P < 0.05).

Analysis of arterial blood samples obtained from eight of the patients revealed PaO2 values in the range 4.77–6.86 kPa and PaCO2 values of 19.5–39.3 kPa.

RESULTS

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DISCUSSION

Measures are often taken to obtund the haemodynamic responses to tracheal intubation [4–8]. Changes occurring at tracheal extubation are less well documented but, nonetheless, some authors have advocated methods to attenuate them [3].

Elia and colleagues [9] observed minimal systemic or coronary haemodynamic responses after tracheal extubation without laryngoscopy. However, in the seven patients studied, the trachea was extubated under carefully controlled conditions in an intensive care unit after coronary artery by-pass surgery. Wallin and co-workers [10] observed that an i.v. infusion of lignocaine during and after surgery suppressed extubation-induced hypertension and tachycardia. Bidwai and associates [3] also noted that the increases in heart rate and arterial pressure observed after tracheal extubation could be attenuated by prior administration of i.v. lignocaine.

In the present study, there were increases in heart rate, diastolic arterial pressure and plasma concentrations of noradrenaline after tracheal intubation. After tracheal extubation at the end of anaesthesia, there were increases in heart rate and plasma concentrations of adrenaline.

The increase in adrenaline concentration during and after surgery may represent a physiological response to trauma and surgery. The absence of a

| TABLE I. Patient characteristics, intraoperative morphine administered and PaCO2 (1 min after extubation) (mean (sd) [range]) |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Age (yr)        | 56.5 (10.4) [44-70] |
| Weight (kg)     | 65.8 (13.4) [47-102] |
| Dose of morphine (mg kg⁻¹) | 0.18 (0.03) [0.14-0.24] |
| PaCO2 (kPa)     | 5.54 (0.8) [4.8-6.9] |

| TABLE II. Mean (sd) for heart rate, systolic arterial pressure (SAP), diastolic arterial pressure (DAP), rate–pressure product and catecholamine concentrations. ind. = Induction; int. = intubation. †P < 0.05 after intubation compared with before induction or after extubation compared with end of surgery (two-way ANOVA and Bonferroni t test). ‡P < 0.05, after intubation compared with before induction (paired t test) |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Before ind.     | After ind.      | After int.      | End of surgery  | 1 min           | 3 min           | 5 min           |
| Heart rate      | 73.4 (1.4)      | 84.3 (1.4)      | 93.1*           | 66.6            | 88.2*           | 81.9*           | 77.7            |
| (beat min⁻¹)    | (4.1)           | (3.8)           | (4.1)           | (2.4)           | (4.8)           | (4.7)           | (4.1)           |
| SAP (mm Hg)     | 149.6           | 144.9           | 133.1           | 140.5           | 148.6           | 143.8           |
| (5.0)           | (6.8)           | (7.9)           | (4.1)           | (6.2)           | (6.4)           | (7.3)           |
| DAP (mm Hg)     | 69.6            | 69.6            | 80.0*           | 70.9            | 78.0           | 77.4            | 74.9            |
| (1.4)           | (2.6)           | (3.3)           | (2.1)           | (3.3)           | (3.2)           | (3.9)           |
| Rate-pressure product | 11046 (859) | 11232 (701) | 13778* (866) | 8831 (402) | 12297* (758) | 12209* (826) | 11274 (923) |
| Noradrenaline (pmol ml⁻¹) | 2.6 (0.3) | 3.2* (0.3) | 3.3 (0.4) | 2.8 (0.4) | 3.0 (0.3) | 3.1 (0.4) |
| Adrenaline (pmol ml⁻¹) | 0.4 (0.6) | 0.9 (0.2) | 1.2 (0.2) | 1.4 (0.2) | 1.4* (0.2) | 1.4* (0.2) |
noradrenaline response may indicate that the stimulus of extubation had less effect than tracheal intubation as it was performed without laryngoscopy.

The haemodynamic changes may have been influenced also by administration of neostigmine and glycopyrronium. It is difficult to separate the effects of extubation from those of antagonism of neuromuscular block. An increase in heart rate was evident at 1 and 3 min after extubation and this may have been caused by the anticholinergic. Glycopyrronium causes less tachycardia than atropine when given with neostigmine [11].

It is not clear in some of the previous publications on tracheal extubation if laryngoscopy was used or not. Laryngoscopy alone produces a significant pressor response [12], presumably because of stimulation of the supraglottic region. We avoided this by omitting laryngoscopy at the end of surgery and used only a soft catheter for clearance of secretions, in order to examine only the changes associated with emergence and extubation.

REFERENCES