

## SHORT COMMUNICATIONS

### Myocardial ischaemia during tracheal intubation and extubation

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#### Summary

The incidence of myocardial ischaemia during tracheal intubation and extubation was compared using ambulatory ECG monitoring in 60 patients undergoing a variety of different surgical operations. Seven patients had myocardial ischaemia after tracheal intubation and seven patients during tracheal extubation. The patients who developed myocardial ischaemia during tracheal extubation had significantly greater rate–pressure products immediately before tracheal extubation ( $P < 0.05$ ) and 1 min after tracheal extubation ( $P < 0.01$ ) compared with those patients who did not develop myocardial ischaemia during extubation. (*Br. J. Anaesth.* 1994; 73: 537–539)

#### Key words

Heart, ischaemia Intubation tracheal Intubation tracheal, extubation.

Myocardial ischaemia occurring during surgery has been shown to be associated with adverse post-operative outcome [1]. In 1979, Roy, Edelist and Gilbert showed that tracheal intubation could precipitate myocardial ischaemia [2]. It has been shown that the haemodynamic consequences of extubation are similar to those after intubation [3], but there has been little work investigating the incidence of myocardial ischaemia during extubation. The aims of this study were to compare the incidence of myocardial ischaemia during tracheal intubation and extubation in patients undergoing non-cardiac surgery and to investigate the role of haemodynamic changes during these periods.

#### Methods and results

We studied 60 patients undergoing a variety of surgical procedures. The study was approved by the local Ethics Committee and written informed consent was obtained from all patients.

All patients received a standard anaesthetic, including the methods of tracheal intubation and extubation. After premedication with temazepam, anaesthesia was induced with alfentanil  $10 \mu\text{g kg}^{-1}$  and a dose of etomidate sufficient to obtund the eyelash reflex. Neuromuscular block was produced with atracurium  $0.5 \text{ mg kg}^{-1}$  and tracheal intubation was performed 3 min after induction. Patients' lungs were ventilated with enflurane and nitrous oxide in

oxygen and bolus doses of morphine i.v.; doses were adjusted as clinically appropriate in individual patients. At the end of operation enflurane was discontinued; laryngoscopy and suction of the oropharynx were performed 3 min later. Two minutes later neuromuscular block was antagonized with neostigmine 2.5 mg and glycopyrronium 0.5 mg and the nitrous oxide was discontinued. The trachea was then extubated when clinically appropriate. Recordings of the patient's heart rate, arterial pressure and peripheral oxygen saturation were obtained every 1 min throughout the periods covering tracheal intubation and extubation.

Myocardial ischaemia was detected using the Compas computerized ambulatory ECG surveillance system, a microprocessor-based solid state system programmed with algorithms for accurate analysis of the ECG [4]. We recorded leads V2 and V5 covering the period from 2 h before operation until 10 min after tracheal intubation. Monitoring was discontinued at this point until the surgeon no longer required the use of diathermy, approximately 10 min before the end of surgery. The monitor was then reattached and the patient monitored for a further period until 30 min after extubation. Significant ischaemia was defined as ST segment depression of 2 mm or more below, or elevation of 3 mm or more above baseline, 60 ms after the J point, persisting for 1 min or more. All ambulatory ECG recordings were analysed after the patient had completed the study.

Haemodynamic variables were compared within each group using repeat measures ANOVA to identify statistical changes, and Student's paired  $t$  tests were then used to identify where changes occurred. Intubation and extubation values were then compared together to identify the relative changes during the two time periods. Haemodynamic variables of the patients who developed myocardial ischaemia were compared with those who did not develop myocardial ischaemia using the Mann–Whitney  $U$  test.

We studied 60 patients (37 males and 23 females) with a mean age of 66 (range 38–84) yr undergoing a variety of surgical procedures. Twenty-one patients had known ischaemic heart disease (IHD) (angina or previous myocardial infarction) and 16 patients without known IHD were regarded as having strong

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**Table 1** Haemodynamic data of all patients during tracheal intubation and extubation (mean (SD)). Intubation: significant differences compared with before induction, \* $P < 0.05$ , \*\* $P < 0.01$ , \*\*\* $P < 0.001$ . Extubation: significant differences compared with preinduction values, † $P < 0.05$ , †† $P < 0.01$ , ††† $P < 0.001$ ; compared with 1 min after tracheal intubation, ‡† $P < 0.01$ , ‡†† $P < 0.001$ .

	Heart rate (beat min <sup>-1</sup> )	Systolic AP (mm Hg)	Diastolic AP (mm Hg)	Rate–pressure product
<b>Intubation</b>				
Before induction	75.9 (12.4)	150.8 (26.0)	82.8 (14.9)	11 413 (2538)
End of induction	66.6 (12.8)	107.9 (25.2)	61.6 (16.5)	7 260 (2479)
After intubation				
1 min	88.0 (16.8)***	145.4 (39.1)	90.6 (27.8)*	13 147 (5122)**
3 min	78.1 (14.5)	144.5 (35.8)	82.9 (18.6)	11 462 (3762)
5 min	71.8 (16.3)	130.3 (28.8)	72.5 (17.0)	9 551 (3644)
<b>Extubation</b>				
End of surgery	68.1 (13.8)	133.0 (19.7)	77.9 (11.0)	9 008 (2021)
After laryngoscopy (1 min)	79.6 (16.5)	148.0 (24.4)	89.8 (13.6)†††	11 744 (2905)
After suction (2 min)	81.2 (17.7)††	152.2 (22.8)	91.7 (13.9)†††	12 327 (3086)†
Immediately before extubation	86.4 (18.2)†††	157.1 (28.3)	92.7 (15.5)†††	13 654 (4117)†††
After extubation				
1 min	89.3 (19.9)†††	161.2 (28.9)†††‡	94.8 (19.1)†††	14 515 (4517)†††‡
3 min	84.3 (19.9)†††	164.3 (26.5)†††‡‡	91.3 (17.7)†††	13 847 (4489)†††
5 min	83.2 (20.7)††	160.5 (25.1)†††‡	86.6 (16.7)	13 440 (4351)††

risk factors for its development (treated hypertension, diabetes mellitus, peripheral vascular disease or a past history of cardiac failure).

Twelve patients developed myocardial ischaemia at some time during monitoring, two of whom had evidence of myocardial ischaemia during all monitoring periods. This included seven of 21 patients with known IHD, two of 16 patients without known IHD but with risk factors, and three of 23 patients with neither known IHD nor risk factors ( $P = 0.17$ , chi-square).

Seven patients had episodes of silent myocardial ischaemia in the 2 h before surgery. Three of these patients had episodes of myocardial ischaemia in the anaesthetic room before induction and these patients continued to have myocardial ischaemia throughout the peri-intubation period. Two other patients developed myocardial ischaemia after induction of anaesthesia and another two patients developed myocardial ischaemia after tracheal intubation. Thus a total of seven patients had myocardial ischaemia in the period immediately after tracheal intubation, and of these, five had myocardial ischaemia in the 2 h before anaesthesia. One patient had myocardial ischaemia after tracheal intubation but at no other time.

At the end of surgery, before extubation was commenced, two patients had episodes of myocardial ischaemia and these continued to have ischaemia throughout the peri-extubation period. During emergence from anaesthesia (the period from laryngoscopy and orotracheal suction until 5 min after tracheal extubation), another five patients developed myocardial ischaemia. These patients first developed myocardial ischaemia 3 min before extubation (two patients), 1 min before extubation (one patient) and at the same time as extubation (two patients). Thus a total of seven patients had myocardial ischaemia during extubation. Four of these patients had myocardial ischaemia before operation, but two patients developed myocardial ischaemia during this period only. Four patients had myocardial ischaemia during both intubation and extubation. Five of the

seven patients who had myocardial ischaemia during extubation continued to show myocardial ischaemia in the recovery room and one other patient developed myocardial ischaemia 6 min after extubation.

Mean haemodynamic variables during induction and emergence from anaesthesia are shown in table 1. In addition, the rate–pressure product (RPP) of patients who developed myocardial ischaemia was compared with those patients who did not. There were no significant differences after induction and intubation. However, at extubation, RPP values for the seven patients who developed ischaemia were significantly greater than for the 53 patients who did not, immediately before extubation ( $P < 0.05$ ) and 1 min after extubation ( $P < 0.01$ ).

### Comment

We have shown that both tracheal intubation and extubation provoked myocardial ischaemia, as detected by ambulatory ECG monitoring. In addition, RPP immediately before extubation and 1 min after extubation was significantly greater in those patients who had myocardial ischaemia during extubation compared with those who did not.

In 1979, Roy, Edelist and Gilbert [2] first showed that tracheal intubation could precipitate myocardial ischaemia. We believe that this is the first study to have documented a similar relationship between tracheal extubation and myocardial ischaemia. However, the uncontrolled nature of the study suggests that our results, in relation to the role of RPP in the development of myocardial ischaemia during extubation, should be interpreted with care. The study was intended as a preliminary investigation into the incidence of myocardial ischaemia during tracheal intubation and extubation. As a consequence, we investigated patients from several surgical groups with a variety of different preoperative risk factors. The relationship between RPP and myocardial ischaemia may be causal or may be the result of an association with other factors which may lead to the development of myocardial ischaemia and it is not

possible to make firm conclusions from these data. However, this study does suggest the possibility that reducing the haemodynamic response to extubation in at risk patients may reduce the risk of developing myocardial ischaemia.

While there have been numerous studies assessing different methods of reducing the haemodynamic response to intubation, there are only a few studies examining ways of reducing this response during extubation [5, 6]. We feel that further studies in this area with regard to the development of myocardial ischaemia would be of value.

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