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Blood-pressure and Pulse-rate Responses to Endotracheal Extubation with and without Prior Injection of Lidocaine

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Increases in heart rate and arterial blood pressure at the end of anesthesia and just after endotracheal extubation are common, and can produce dangerous increases in myocardial oxygen demand in patients who have coronary-artery disease.[1] Intravenous administration of lidocaine depresses cough reflexes during endotracheal extubation during "light" planes of general anesthesia; however, blood-pressure and pulse-rate responses during and after this therapeutic modality have not been carefully evaluated.[2,3] This study was undertaken to investigate and compare blood-pressure and pulse-rate responses to endotracheal extubation during light general anesthesia with and without prior iv administration of lidocaine.

METHODS

Eighty ASA class 1 or class 2 patients who were undergoing a variety of elective orthopedic, general surgical and gynecologic operations made up the experimental subjects. Informed written consent was obtained from each patient at the preoperative visit. The study was approved by the review committee for research with human subjects.

Every patient was premedicated with meperidine (50–75 mg, im), hydroxyzine hydrochloride (75–100 mg, im) and atropine (0.3–0.5 mg, im) 60–90 min before the scheduled operation. Anesthesia was induced with thiopental (4 mg/kg, iv), the trachea was intubated after administration of succinylcholine (1.5 mg/kg, iv) and anesthesia was maintained with halothane (0.3–1.5 per cent) and nitrous oxide (50 per cent) in oxygen. Muscle paralysis was achieved with an intermittent infusion of 0.1 per cent solution of succinylcholine in 5 per cent dextrose in water.

Patients were randomly divided into two groups of 40 each with regard to management of endotracheal extubation at the end of operation.¶ Patients in Group I received an iv injection of 2 per cent lidocaine (1 mg/kg) 2 min prior to extubation, when the halothane concentration (0.2–0.3 per cent) was judged just adequate to prevent coughing on the endotracheal tube. One minute prior to endotracheal extubation, nitrous oxide and halothane were completely discontinued. Just prior to extubation, oropharyngeal suction was accomplished and the endotracheal cuff was completely deflated and then slowly withdrawn. Following endotracheal extubation 100 per cent oxygen was given by face mask.

Single measurements of systolic and diastolic blood pressures and heart rate were obtained every 1–2 min during the study and were recorded prior to and 1 min following lidocaine administration (1 min before endotracheal extubation), at extubation (2 min after lidocaine), 1 min after extubation, and upon entrance to the postanesthetic recovery room (6–8 min after lidocaine administration). Patients in Group II received 3 ml saline solution, iv, 2 min prior to endotracheal extubation, but were otherwise treated similarly and had data recorded at the same times as those in Group I.

Blood pressures and pulse rates recorded for this study were obtained by an individual (not the anesthesiologist) who was unaware of the medication administered before endotracheal extubation. Neither the anesthesiologist nor the recorder knew whether saline solution or lidocaine had been administered. Data were evaluated for statistical significance using the Student *t* test for paired data (for comparison of changes from control in each group) and the Student *t* test for unpaired data (for comparison of Group I with Group II).

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¶ Random assignment was accomplished before induction of anesthesia by an individual not involved in administration of the anesthetic or measurements of blood pressure and pulse rate during the study.

TABLE 1. Responses to Extubation with and without Prior Intravenous Administration of Lidocaine (Mean ± SD)

	Blood Pressure (torr)		Heart Rate (Beats/Min)
	Systolic	Diastolic	
Two minutes before extubation (prior to injection of lidocaine or saline solution)			
Group I	112 ± 11	70 ± 8	76 ± 9
Group II	108 ± 9	68 ± 8	78 ± 10
One minute after injection of lidocaine or saline solution			
Group I	104 ± 9	70 ± 7	72 ± 9
Group II	114 ± 10	70 ± 8	80 ± 9
At extubation			
Group I	114 ± 12	70 ± 9	76 ± 8
Group II	124 ± 11*	80 ± 10*	92 ± 10*
One minute after extubation			
Group I	120 ± 11	76 ± 8	84 ± 11
Group II	132 ± 13†‡	90 ± 12†‡	96 ± 11†‡
Upon entrance into the recovery room			
Group I	118 ± 12	78 ± 9	84 ± 10
Group II	136 ± 14†‡	94 ± 10†‡	101 ± 12†‡

* $P < 0.05$, † $P < 0.01$, Student t test for paired data, compared with values obtained 2 min before extubation.

‡ $P < 0.025$, Student t test for unpaired data, compared with Group I values obtained at the same time.

RESULTS

Patients in the two groups had similar blood pressures and pulse rates 2 min before endotracheal extubation and prior to lidocaine administration (table 1). Patients in Group I did not sustain a significant elevation in systolic or diastolic blood pressure or pulse rate at or after extubation or in the recovery room. Patients in Group II experienced ($P < 0.05$) increases in both pressures and pulse rate during endotracheal extubation which persisted for 1 min after extubation and were still present upon arrival in the recovery room. Changes in systolic and diastolic arterial blood pressures and pulse rates in patients of Group II became significantly greater than those of patients in Group I 1 min after endotracheal extubation, and remained so upon entrance into the recovery room. Twenty-eight of 40 patients in Group II coughed at or shortly after extubation. No patients in Group I coughed at any time during the study.

DISCUSSION

Patients who have coronary-artery disease are at risk during cardiovascular stimulation. Increases in arterial blood pressure and heart rate increase myocardial oxygen requirements and can produce myocardial ischemia or infarction in these patients.[1] Recent reports have demonstrated that topically or intravenously administered lidocaine attenuates circulatory responses to endotracheal intubation.[3–6] However, little attention has been focused on blockade of circulatory stimulation secondary to light planes of anesthesia at the end of operation and just after extubation. The results of this study demonstrate that intravenous injection of lidocaine (1 mg/kg) 2 min before endotracheal extubation prevents coughing and increases in arterial blood pressure and pulse rate during and after extubation.

Recently, Stoelting showed that oropharyngeal viscous lidocaine attenuated but did not totally eliminate increases in arterial blood pressure and heart rate during endotracheal intubation following thiamylal and succinylcholine.[5] Serum lidocaine concentrations did not exceed 0.5 μg/ml during intubation in that study. It was not possible to measure serum lidocaine concentrations in this investigation; however, Schartz and Iwamoto** found that a plasma lidocaine concentration of 3.26 μg/ml 5 min after administration of 2 per cent lidocaine (2 mg/kg) prevented coughing upon extubation in 14 patients lightly anesthetized with enflurane and nitrous oxide. In contrast, the same investigators showed that ten of 15 patients similarly anesthetized coughed at endotracheal extubation 5 min later, when plasma lidocaine concentrations averaged 2.01 μg/ml.

Our data do not reveal whether the beneficial effects of lidocaine in this study were due to tracheal absorption of the compound from blood or greater general anesthesia from central nervous system effects of lidocaine. Either of these mechanisms could explain why patients in Group I experienced no significant change in arterial blood pressure and heart rate with endotracheal extubation and those in Group II sustained significant elevations in these variables. In a recent report,[7] we demonstrated that similar protection from coughing and cardiovascular stimulation with endotracheal extubation occurs when lidocaine is sprayed down the endotracheal tube.

Our findings also do not indicate the optimal time for endotracheal extubation after intravenous injection of lidocaine. However, since two thirds of the patients of Schwartz and Iwamoto coughed during endotracheal extubation 10 min after intravenous administration of twice as much lidocaine as was given in this study, it is likely that the ideal time for extubation

** Schwartz H, Iwamoto K: A quantitative study of intravenous lidocaine for suppression of the cough reflex. Abstracts, Annual Meeting of the American Society of Anesthesiologists, October 1977, pp 773–774.

is not far from 2 min following administration of lidocaine as in this study.

In conclusion, the results of this study demonstrate that intravenous injection of lidocaine (1 mg/kg) administered 2 min before endotracheal extubation prevents coughing and increases in blood pressure and pulse rate during and after extubation. Our findings suggest that intravenous administration of lidocaine prior to endotracheal extubation should be advantageous to the patient who has coronary-artery disease and cannot tolerate increases in cardiovascular dynamics.

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Exacerbation of Iatrogenic Hypercarbia by PEEP

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The efficacy of positive end-expiratory pressure (PEEP) in the treatment of adult acute respiratory failure, and improvement of functional residual capacity (FRC), is well recognized.[1,2] A PEEP device commercially available for intraoperative use† has been reported to be effective and safe.[3]

The following case report demonstrates a detrimental effect of this device upon carbon dioxide elimination in a cardiac patient, when combined with a malfunctioning anesthesia circle system. The summary of this case report will focus on the respiratory system.

Report of a Case

Fourteen days after mitral valve replacement and two-vessel coronary-artery-bypass grafting, a 64-year-old woman weighing 62 kg was scheduled for removal of an intra-aortic counterpulsation balloon. Past surgical and medical history included numerous myocardial infarctions, triple coronary-artery-bypass grafting two years previously, severe mitral-valve regurgitation secondary to papillary muscle infarction, and chronic congestive heart failure. Preoperative respiratory support was provided, via a tracheostomy, with an MA-1® ventilator, utilizing an intermittent mandatory ventilation (IMV) mode and PEEP. Arterial blood-gas analysis showed satisfactory ventilation (table 1). The patient was awake and oriented.

During transport to the operating room, ventilation was assisted by use of a Jackson-Rees modification of the Ayre's T-piece, and a 10-l/min flow of oxygen. In the operating room, ventilation was provided either manually or with an Air Shields® ventilator, utilizing the previously checked Ohio® anesthesia machine. Anesthetic medications included nitrous oxide, fentanyl, diazepam, and pancuronium. Axillary temperature was 37 C throughout the procedure. Intraoperatively, 5 cm H_2O PEEP was placed in the exhalation limb of the anesthesia circuit. After the application of PEEP, the patient appeared progressively agitated, began to resist mechanical ventilation, and the inspiratory peak pressures on the absorber canister pressure gauge increased to 45 cm H_2O. The oxygen analyzer in the inspired gas circuit confirmed the F_{IO_2} expected from the flowmeter settings. Adequate oxygenation was confirmed by serial arterial blood-gas analyses, but progressive hypercarbia became evident (table 1). Breath sounds, easily auscultated over the chest, were equal bilaterally. Ventilation coordinated well with visual impressions of inflation of the chest. No abnormality was discovered with the tracheostomy tube or cuff. Carbon dioxide was not being administered, and the CO_2-absorption system and soda-lime appeared normal. The operation was completed, and the patient was transported to the intensive care unit, where ventilation was provided as preoperatively. Arterial blood-gas analyses twice during the first hour in the intensive care unit showed normocarbia, and were consistent with corresponding preoperative values (table 1). There were no sequelae, and the patient made an uneventful recovery.

Postoperative evaluation of the anesthesia circuit demonstrated (beneath the fogged valve cover) that

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