TITLE : Cardiovascular Responses to Extubation

AUTHORS : R. Richard Edde, M.D.

AFFILIATION: Department of Anesthesiology, University of Oklahoma Health Sciences Center, Oklahoma City, and the Veterans Administration Medical Center, Oklahoma City, Oklahoma 73104.

Introduction. Cardiovascular responses to laryngoscopy and tracheal intubation are well-documented. Techniques in mitigating these responses have also been well-studied.[1] The present study was undertaken to determine what cardiovascular responses were involved during extubation and whether these responses could be attenuated in a manner similar to those employed during laryngoscopy.

Methods. After informed consent and institutional approval for the study were obtained, forty healthy (ASA Class I) patients who underwent extra-abdominal urologic or orthopedic surgical procedures were studied. They were divided into four groups of ten patients each and randomly assigned to Group A: control; Group B: saline control; Group C: tracheal anesthesia with 40 mg. lidocaine per endotracheal tube; or Group D: lidocaine 50 mg. intravenously. All patients received a preoperative medication of diazepam, 0.2 mg/kg and atropine 0.4 mg. Induction of anesthesia was accomplished with thiopental, 4 mg/kg. Anesthesia was maintained with halothane and nitrous oxide/oxygen to yield an FI02 of 0.4. During emergence, the halothane concentration was reduced to the minimum amount judged necessary to prevent coughing.

Five minutes prior to extubation, either saline, one ml. (Group B), or lidocaine 40 mg, one ml., (Group C) was injected down the endotracheal tube. Group D received lidocaine, 50 mg. intravenously, five minutes before extubation.

Blood pressure and pulse rates were measured, and mean arterial pressure and rate-pressure products calculated before and immediately after extubation.

Results. Results reveal that the control group and saline control group had a significant increase in mean arterial pressure, pulse rate and rate-pressure product immediately after extubation. Pretreatment with lidocaine per endotracheal tube resulted in no significnat differences in these parameters.

Lidocaine given intravenously (Group D) was associated with significant increases in pulse, and rate-pressure product whereas mean arterial pressure remained unchanged.

Although no attempt was made to quantitate the amount of lidocaine actually in contact with tracheal mucosa or how these amounts compare with measured blood levels of lidocaine, these preliminary results seem to indicate that lidocaine administered through an endotracheal tube may attenuate increases in blood pressure, heart rate, and rate-pressure products. We feel that a beneficial effect on cardiovascular hemodynamics may be derived from tracheal anesthesia prior to extubation.

References.

1. Stoelting RK: Circulatory changes during direct laryngoscopy and tracheal intubation. Anesthesiology 47: 381-384, 1977.

TABLE 1. CIRCULATORY CHANGES FOLLOWING EXTUBATION

	BEFORE EXTUBATION	IMMEDIATELY AFTER EXTUBATION
GROUP A (Control)		
Mean arterial pressure	91± 2	98 ±2**
Pulse	80± 4	92 ±3**
Rate-Pressure product	9231± 605	11666 ±546*
GROUP B (Saline Control)		
Mean arterial pressure	92± 2	99± 2**
Pulse	81± 3	95± 2**
Rate-Pressure product	9242± 432	12010± 522*
GROUP C (Lidocaine ETT)		
Mean arterial pressure	91± 3	92± 2 (NS)
Pulse	84± 3	85± 3 (NS)
Rate-Pressure product	9725± 584	9729± 465 (NS)
GROUP D (Lidocaine IV)		
Mean arterial pressure	92± 3	94± 3 (NS)
Pulse	76± 3	81±4*
Rate-Pressure product	8850± 570	9543± 584*

**p< 0.005 compared to before extubation
*p< 0.05 compared to before extubation
NS not statistically significant
ETT=Endotracheal Tube