Lidocaine in Arterial Blood after Laryngotracheal Administration

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Systemic lidocaine absorption occurs following topical administration of the local anesthetic. Pelton *et al.* reported lidocaine concentration elevations to about 2.7 μg/ml plasma following 3 mg/kg lidocaine delivered into the trachea with an aerosol dispenser.[1] This study reports arterial-blood lidocaine concentrations following laryngeal and tracheal administration of lidocaine (2 mg/kg) with a commercially available disposable cannula and syringe (Laryngotracheal Anesthesia Kit‡).

METHODS

Twelve adult patients, without known heart disease, were studied before elective operations. No patient was receiving any drug known to induce hepatic microsomal enzymes, and all received preanesthetic medication with morphine and scopolamine. A radial-artery catheter and one to two intravenous catheters were placed after local anesthesia with 0.5–2 ml of 1 per cent lidocaine. Intravenous administration of *d*-tubocurarine, 3 mg, was followed 3 minutes later by thiamylal, 4 mg/kg, and succinylcholine, 2 mg/kg. One minute after thiamylal–succinylcholine administration, direct laryngoscopy was performed. Before placement of a cuffed orotracheal tube, lidocaine (2 mg/kg, 4 per cent solution) was sprayed into the larynges and tracheas of ten patients using a Laryngotracheal Anesthesia Kit. The cannula was placed at least 10 cm through the glottic opening and the calculated lidocaine dose delivered by rapid depression of the syringe plunger. The distal end of the tracheal tube was lubricated with 2–3 ml of 2 per cent viscous lidocaine in four patients, whereas no lubricant was used in six patients. Two patients did not receive laryngotracheal lidocaine but were given lidocaine, 2 mg/kg, iv, 1 minute after administration of thiamylal and succinylcholine, after which a nonlubricated cuffed orotracheal tube was inserted. Ventilation was controlled and anesthesia maintained with 60 per cent nitrous oxide and 0.5 to 1.0 per cent halothane in oxygen.

Arterial-blood samples for lidocaine analysis[2] were obtained just before thiamylal administration (control), and 1, 4, 9, and 15 minutes after laryngotracheal or intravenous administration of lidocaine.

RESULTS

The concentration of lidocaine in arterial blood increased within 1 minute following laryngotracheal administration. Maximum measured lidocaine concentrations in arterial blood of patients whose tracheas were intubated with nonlubricated tracheal tubes were 1.7 ± 0.2 μg/ml (mean ± SE) 9 to 15 minutes after laryngotracheal administration (table 1). Maximum measured arterial-blood lidocaine concentrations in patients whose tracheas were intubated with viscous lidocaine-lubricated tracheal tubes were 2.4 ± 0.3 μg/ml 4 to 15 minutes after laryngotracheal administration (table 1).

After intravenous administration, peak arterial-blood lidocaine concentrations were greater, and occurred sooner, than after laryngotracheal administration, and concentrations were decreasing by 4 minutes (table 2).

DISCUSSION

Arterial-blood lidocaine concentrations between 2 and 5 μg/ml are necessary if protection against ventricular dysrhythmias is needed.[5] This therapeutic range is not associated with hemodynamic effects.[4] Maximum measured arterial-blood lidocaine concentrations after laryngotracheal administration of lidocaine (2 mg/kg) and endotracheal intubation with a nonlubricated orotracheal tube were usually less than 2 μg/ml. Therefore, protection against ventricular dysrhythmias or myocardial depression after laryngotracheal administration of lidocaine, 2 mg/kg, seems unlikely. Indeed, the only effect of laryngotracheal lidocaine just before intubation seems to be a

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Received from Indiana University School of Medicine, Indianapolis, Indiana 46202. Accepted for publication April 27, 1975.
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TABLE 1. Lidocaine Concentrations in Arterial Blood (μg Lidocaine Base/ml) after Laryngotracheal Administration

	Control*	Minutes after Laryngotracheal Lidocaine Administration (2 mg/kg) 1	4	9	15	Maximum Measured Lidocaine Elevation	Time of Maximum Measured Lidocaine Elevation (Minutes)	Laryngotracheal Lidocaine (mg)
Laryngotracheal lidocaine and nonlubricated tracheal tube								
Patient 1	Trace	0.4	0.5	1.1	1.3	1.3	15	160
Patient 2	Trace	0.6	1.4	2.5	2.0	2.5	9	160
Patient 3	Trace	1.0	1.0	1.7	1.9	1.9	15	120
Patient 4	Trace	0.5	0.7	0.9	1.0	1.0	15	160
Patient 5	Trace	0.4	0.7	1.3	2.0	2.0	15	140
Patient 6	Trace	0.4	0.5	1.2	1.7	1.7	15	110
MEAN		0.6	0.8	1.5	1.7	1.7		142
SE		0.1	0.1	0.2	0.2	0.2		9
Laryngotracheal lidocaine and viscous lidocaine-lubricated tracheal tube								
Patient 7	0.2	0.8	2.0	2.0	1.2	2.0	4	100
Patient 8	Trace	0.2	2.7	2.6	2.5	2.7	4	160
Patient 9	Trace	1.3	1.4	2.0	1.7	2.0	9	100
Patient 10	0.4	1.0	1.5	2.0	2.7	2.7	15	150
MEAN		0.8	1.9	2.2	2.0	2.4		128
SE		0.2	0.3	0.2	0.3	0.3		16

* Trace = small gas chromatograph peak which could not be accurately measured (less than 0.1 μg/ml). Lidocaine present in the control measurement probably represented systemic absorption of the local anesthetic used for infiltration before placement of arterial and venous catheters.

TABLE 2. Lidocaine Concentrations in Arterial Blood (μg Lidocaine Base/ml) after Intravenous Administration

	Control	Minutes after Intravenous Lidocaine (2 mg/kg) 1	4	9	15	Intravenous Lidocaine (mg)
Patient 11	0.1	6.6	4.0	2.7	1.9	130
Patient 12	0.1	8.5	4.3	3.7	3.0	200

less persistent (not diminished) pressor response after laryngoscopy and intubation, with toleration of the endotracheal tube without immediate additional anesthesia.§

Lidocaine concentrations in arterial blood were somewhat greater when laryngotracheal administration was followed by intubation with a viscous lidocaine-lubricated tracheal tube. Nevertheless, sustained therapeutic lidocaine levels were not achieved. Furthermore, lubrication of a tracheal tube of a proper size to facilitate passage through the larynx is of questionable value.[5]

The maximum measured concentration of lidocaine in arterial blood was present in six of the ten patients 15 minutes following laryngotracheal administration. Since measurements were not obtained after 15 minutes, we cannot state with certainty that these data reflect the maximum lidocaine concentrations in every patient.

§ Stoelting RK, Peterson C: Personal communication.

Adriani and Campbell asserted that local anesthetic absorption through mucous membranes could simulate intravenous administration.[6] However, systemic absorption may be slowed through laryngeal and tracheal mucosa, and lidocaine dissolved in secretions lining the upper airway may impede systemic absorption. Indeed, administration of lidocaine resulted in a peak lidocaine concentration within 1 minute, while laryngotracheal administration was associated with later peaks and lower but more sustained arterial-blood lidocaine concentrations.

Mr. J. B. Keenaghan, Astra Pharmaceutical Products, Inc., Worcester, Massachusetts, and Dr. J. F. Nash and Ms. M. K. Brunson, Eli Lilly and Company, Indianapolis, Indiana, performed the arterial lidocaine analyses.

References

1. Pelton DA, Daly M, Cooper PD, et al: Plasma lidocaine concentrations following topical aerosol application to the trachea and bronchi. Can Anaesth Soc J 17:250–255, 1970
2. Keenaghan JB: The determination of lidocaine and prilocaine in whole blood by gas chromatography. ANESTHESIOLOGY 29:110–112, 1968
3. Gianelly R, von der Groben JO, Spivack AP, et al: Effect of lidocaine on ventricular arrhythmias in patients with coronary heart disease. N Engl J Med 277:1215–1219, 1967
4. Grossman JI, Cooper JA, Frieden J: Cardiovascular effects of infusion of lidocaine on patients with heart disease. Am J Cardiol 24: 191–197, 1969
5. Nicholson MJ: Questions and answers. Anesth Analg (Cleve) 53:573–574, 1974
6. Adriani J, Campbell D: Fatalities following topical application of local anesthetics to mucous membranes. JAMA 162:1527–1530, 1956

Use of Succinylcholine in the Presence of Atypical Cholinesterase

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Succinylcholine is a short-acting neuromuscular blocking agent because after injection it is rapidly hydrolyzed in the circulation by plasma cholinesterase. Succinylcholine is usually given in doses of 0.5 to 2 mg per kg body weight so that despite metabolism in the circulation, an adequate concentration of drug is achieved at the motor endplate to produce its effect. Atypical cholinesterase is unable to metabolize succinylcholine rapidly. Therefore, in a patient with the atypical enzyme, doses of the order noted comprise an overdose. This is a report of a patient with atypical cholinesterase who required repeated anesthesias for electroconvulsive therapy (ECT), in whom we were able to observe the responses to graded doses of succinylcholine.

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Received from the Departments of Anaesthesia, Peter Bent Brigham Hospital and Harvard Medical School, Boston, Massachusetts 02115. Accepted for publication April 30, 1975.

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Report of a Case

An 80-year-old man of physical status 2 with depression resulting in severe anorexia and weight loss required anesthesia for ECT. On the first occasion, after atropine premedication and oxygenation, methohexital sodium, 50 mg, and succinylcholine, 60 mg, were injected intravenously. Following ECT the patient remained apneic and needed assisted respiration for 2 hours. Blood was drawn for measurement of serum electrolyte concentrations and serum cholinesterase levels and for characterization of the cholinesterase. Serum electrolytes were normal. Before the results of the cholinesterase studies were available, anesthesia was given for a second treatment. On this occasion the dose of succinylcholine was reduced to 40