Tracheostomy without a previously secured endotracheal tube with or without general anesthesia carries certain risks. Since the duration of artificial airway maintenance was expected to be brief, tracheostomy was considered a last resort.

The original idea of a retrograde guide for endotracheal intubation involved passing a catheter through a tracheostomy and into the pharynx as a guide for subsequent endotracheal intubation.[3] Other authors utilized a large bore needle to introduce a catheter through the cricothyroid membrane, passing the catheter between the vocal cords into the mouth and thereby providing a guide for oral endotracheal intubation.[2,4]

This method of retrograde transtracheal intubation possesses some unique advantages. The thin-walled needle is considerably less traumatic than those described previously. Because of this, the frequency of damage to the trachea or underlying structures should be decreased. The use of a wire (vs. a long-line plastic catheter) decreases the elasticity of the guide while the nasotracheal tube is being inserted thereby minimizing the chance of breaking the introducing catheter. This would allow

more control during the time immediately prior to removal of the guide wire. The placement of the guide wire through the side hole of the nasotracheal tube allowed an additional 10–11 mm of nasotracheal tube to be within the trachea prior to removal of guide wire. Because of the short tracheal lengths in the pediatric population, this can be the difference between success and failure of the entire technique. Of final importance is the brief time required for completion of this intubation. Utilization of operating room time is minimal and ability of provide a safe secure airway is enhanced.

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Lidocaine before Endotracheal Intubation: Intravenous or Laryngotracheal?

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Lidocaine, given topically to the larynx and trachea[1] or intravenously,[2] has been shown to blunt the increases in heart rate and blood pressure associated with laryngoscopy and endotracheal intubation. Intravenously administered lidocaine also prevents intracranial hypertension when patients with brain tumors undergo endotracheal intubation.[3] The purposes of this study were twofold: 1) to describe the effects of laryngotracheal lidocaine administration on intracranial pressure, and 2) to determine whether there is a preferred route for administration of lidocaine before endotracheal intubation.

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Methods

Twenty-two patients with brain tumors estimated to be larger than 3 cm in diameter by CAT-scan were studied. All were receiving steroid therapy and were scheduled for elective craniotomy. The protocol was approved by the Human Investigation Committee at the University of Virginia Medical Center, and informed consent was obtained both from the patients and their next of kin on the evening prior to operation. Morphine, 0.1 mg/kg, and atropine, 0.4 mg, im, and diazepam, 0.1 mg/kg, po, were given one hour before arrival in the operating suite. A subarachnoid pressure screw, arterial and central venous catheters were placed using local anesthesia, and pressures were continually recorded. Intracranial compliance was estimated by administering a 1–2 ml bolus of mock cerebrospinal fluid through the subarachnoid screw while the resulting change in intracranial pressure (ICP) was recorded. Once control measurements of ICP, vascular pressures, and heart rate were recorded, arterial blood was obtained for analysis of blood gases and anesthesia was induced with 3 mg/kg thiopental, 1.5 mg/kg

succinylcholine, and 50 per cent nitrous oxide. One minute after induction of anesthesia, eleven of the patients received laryngotracheal 4 per cent lidocaine, 4 ml, under direct vision with a #3 Macintosh laryngoscope through a standard "laryngotracheal anesthesia"§ (LTA®) set. The other eleven received 1.5 mg/kg lidocaine, iv. The type of treatment was chosen randomly. Ventilation was controlled sufficient to maintain a constant end-tidal CO_2 concentration. Two minutes after induction of anesthesia, laryngoscopy was performed with a Macintosh #3 blade and endotracheal intubation was always accomplished within 20 s. The resulting changes in heart rate (HR), ICP, and mean arterial pressure (MAP) were measured over the next five minutes. Analysis of arterial blood gases were repeated 30 s after endotracheal intubation. Statistical comparisons were performed using analysis of variance and critical difference testing. $P < 0.05$ was regarded as significant.

RESULTS

There was no difference between the groups with respect to age, intracranial compliance, heart rate, mean arterial pressure, and PA_{CO_2} before intubation of the trachea. Despite the presence of large intracranial neoplasms, ICP decreased after iv lidocaine administration and did not significantly increase after endotracheal intubation (fig. 1). In contrast, the group given lidocaine laryngotracheally developed a significant increase in ICP after endotracheal intubation, with three of these patients sustaining ICPs in excess of 40 torr. Laryngoscopy and laryngotracheal administration of lidocaine during the light nitrous oxide–barbiturate anesthesia, resulted in significant increases in heart rate and mean arterial pressure which then persisted after endotracheal intubation. Although iv lidocaine did not entirely prevent cardiovascular stimulation in response to endotracheal intubation, significant increases in heart rate and mean arterial pressure occurred only within the first minute after intubation. In contrast, the group which received laryngotracheal lidocaine sustained significant increases in cardiovascular variables which persisted for at least two minutes (figs. 2 and 3).

DISCUSSION

Laryngoscopy and endotracheal intubation can cause striking changes in hemodynamics[4] and intracranial pressure,[5] probably as a result of intense sympathetic nervous system responses to stimulation. In most patients these changes are transient, highly variable, and probably of little consequence. In patients who are at risk for de-

§ Abbott Laboratories, Chicago, Illinois.
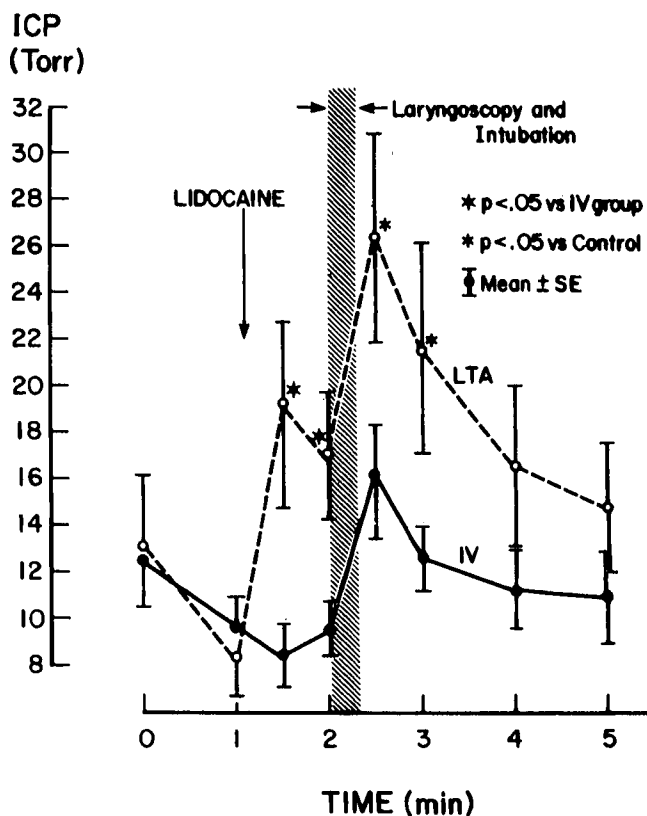

ICP
(Torr)

FIG. 1. Intracranial pressure (ICP) response to laryngoscopy and endotracheal intubation after administration of either laryngotracheal (LTA®) or intravenous (iv) lidocaine. No increase in ICP occurred after intravenous lidocaine, whereas direct instillation of lidocaine into the trachea caused a significant increase in ICP and did not prevent elevated ICP after intubation.

veloping increased intracranial pressure, arterial hypertension or myocardial ischemia, however, these changes may be life-threatening.[6] Although deep levels of volatile anesthetics may limit the cardiovascular response to endotracheal intubation, patients with intracranial mass lesions present a particular challenge since such an anesthetic technique may cause intracranial hypertension and seriously reduce cerebral perfusing pressure.[5] Administration of a larger dose of thiopental than that used in this study might have effectively prevented arterial and intracranial hypertension after endotracheal intubation, but there would have been an increased risk of causing arterial hypotension in at least some of the patients studied. It is because of clinical considerations such as these that lidocaine is often administered either intravenously or laryngotracheally before endotracheal intubation.

Intravenous lidocaine is a suppressant of the cough reflex[7,8] and is effective in preventing or mitigating the arterial hypertension and tachycardia seen with endotracheal intubation.[2] Lidocaine blood levels of 3–6 µg/ml are known to potentiate the effects of nitrous oxide
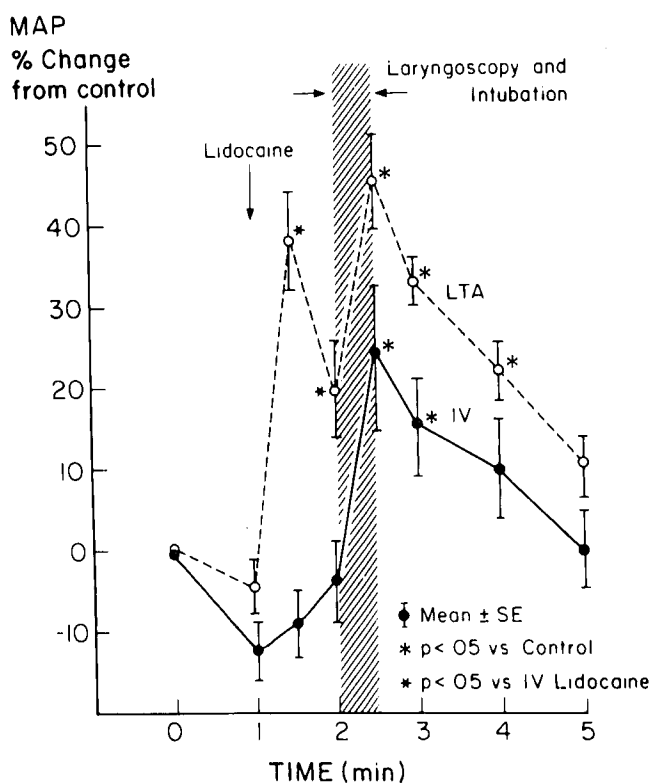
FIG. 2. Mean arterial pressure (MAP) was significantly higher after direct laryngotracheal administration of lidocaine than after iv lidocaine and then remained higher than control values for a minute and a half after intubation. After intravenous lidocaine MAP was significantly increased for less than a minute after intubation was completed.
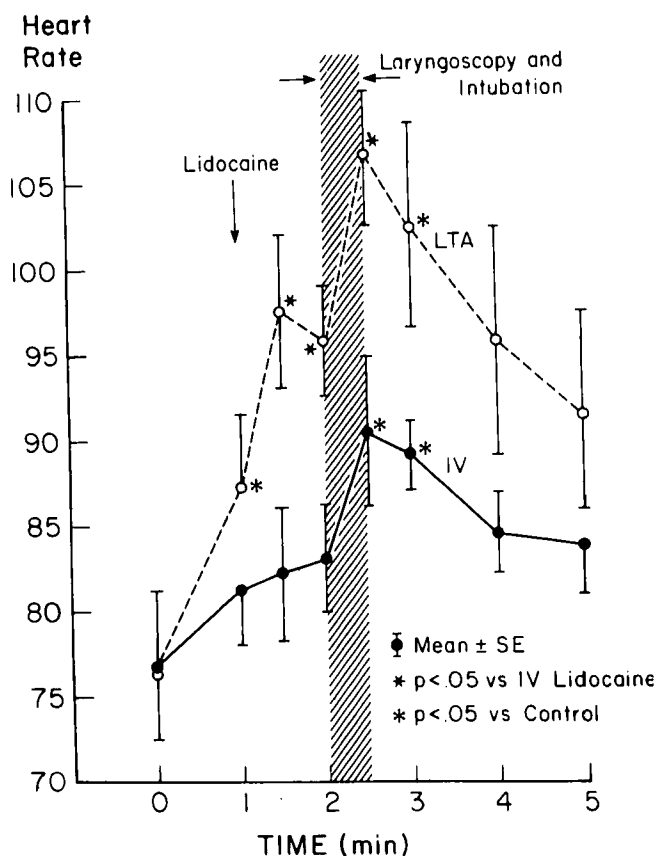

FIG. 3. Heart rate increased significantly in response to laryngotracheal injection of lidocaine, and peak heart rate after endotracheal intubation was significantly higher than that in the intravenous lidocaine group.

anesthesia in human and a 10–28 per cent reduction in halothane MAC has been observed in dogs with blood lidocaine levels between 3 and 10 μg/ml.[9] In a previous study[3] a mean blood lidocaine level of 3.2 μg/ml ± 0.6 SE was observed 1.5 min after a 1.5 mg/kg iv bolus. Thus, it appears that iv lidocaine prevents cardiovascular stimulation at least partially by causing an increase in the depth of general anesthesia.

The effectiveness of intravenously administered lidocaine in preventing increased intracranial pressure, however, has only been described recently.[8,10] Lidocaine causes a 10–27 per cent reduction in cerebral metabolic rate for oxygen and a similar reduction in cerebral blood flow when given to dogs in doses of 3–15 mg/kg.[11] It is possible then, that lidocaine prevents intracranial hypertension by increasing cerebral vascular resistance and decreasing cerebral blood volume during a period of profound cardiovascular stimulation.

The lack of efficacy of topically applied laryngotracheal lidocaine in preventing increased ICP, hypertension, and tachycardia after intubation was unexpected, particularly in view of the widespread clinical use of the LTA® kit. We found, however, that there was no dif-

ference in peak HR, MAP, or ICP between the patients intubated after laryngotracheal lidocaine in this study and the patients in a previous report[3] who received a similar anesthetic technique except that no lidocaine was given.

Personal experience with lidocaine administered in the oropharynx indicates that nearly maximal topical anesthesia occurs within approximately 60 s, and for this reason we used a 60-s time interval between laryngotracheal lidocaine instillation and endotracheal intubation. In contrast, studies which have shown laryngotracheal lidocaine to be effective in preventing hypertension and tachycardia after intubation have used a 5-min time interval between instillation of lidocaine and endotracheal intubation.[1] Viegas and Stoelting[12] have shown that blood lidocaine levels are low one minute after laryngotracheal lidocaine spray, but that they gradually rise to a peak value of 1–2.7 μg/ml between 4 and 15 min thereafter. Thus, only with systemic drug absorption is laryngotracheal lidocaine apparently effective, such as when it precedes endotracheal intubation by five minutes.

The results of the present study indicate that during

light barbiturate–nitrous oxide anesthesia, topical laryngotracheal administration of lidocaine using a laryngoscope and LTA® kit causes significant increases in ICP, HR, and MAP and does not protect against potentially harmful cardiovascular and intracranial pressure changes induced by endotracheal intubation. Intravenous lidocaine, 1.5 mg/kg, given one minute before intubation, both prevents intracranial hypertension and also limits the intensity and duration of cardiovascular stimulation. These data indicate that the intravenous route is the preferred technique for administering lidocaine prior to endotracheal intubation.

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Obstruction of Anomalous Tracheal Bronchus with Endotracheal Intubation

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Several authors have described clinical symptoms in unanesthetized patients associated with a tracheal bronchus.[1-6] In this paper, we describe a case where an otherwise asymptomatic patient developed a right upper lobe collapse with endotracheal intubation during anesthesia. The diagnosis of an anomalous tracheal bronchus acutely obstructed by the endotracheal tube was made by fiberoptic bronchoscopy.

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Report of a Case

A 12-year-old male child was scheduled for excision of maxillary and mandibular cystic lesions. He had no history of pneumonia, airway distress, or known congenital anomalies. Preoperative chest roentgenograph was normal. A 6.5-mm nasotracheal tube was inserted into the trachea with direst visualization utilizing the McGill forceps. The endotracheal tube cuff was palpated in the trachea just superior to the sternal notch. Breath sounds were initially decreased over the right upper lobe but appeared to become equal after withdrawal of the endotracheal tube by about 1 cm. Breath sounds were never decreased on the left side, and chest expansion appeared symmetric. Surgery proceeded during 60 per cent N_2O and 1–2 per cent enflurane anesthesia and spontaneous ventilation.

Approximately 15 min after the start of surgery, the patient became cyanotic. Auscultation of the chest revealed absence of right upper lobe breath sounds, and percussion revealed dullness over the area. Further withdrawal of the endotracheal tube with palpation of cuff further above sternal notch failed to resolve the problem. Chest roentgenogram revealed complete collapse of the right upper lobe.

While the trachea was still intubated, fiberoptic bronchoscopy was performed during which time an anomalous tracheal bronchus originating about 1 cm above and anterolateral to the right main bronchus was visualized (figs. 1 and 2). The endotracheal tube was removed.