Seventy-three of 83 patients studied received 1 mg of lidocaine for each microgram of epinephrine. Lidocaine has a protective effect when injected with epinephrine, and the protection increases with higher doses.[2] A patient receiving 10 μg/kg of epinephrine also received 10 mg/kg of lidocaine. This dose of lidocaine is effective in preventing arrhythmias but may be toxic. The toxicity easily could have been masked by the use of barbiturates and halothane.

The comparison of patients in this study to those of Johnston *et al.*[3] also needs qualification. Johnston's patients were unpremedicated, anesthetized for 30 min at 1.25 MAC, injected in the oral and nasal submucosa, and, when given lidocaine, the dose was 5 mg/ml. Karl's patients largely were premedicated with 4 mg/kg of pentobarbital, anesthetized for an undisclosed period with halothane at levels less than 1.25 MAC, injected in a variety of locations, and most received 10 mg/ml of lidocaine.

The authors point out that many factors influence epinephrine-associated dysrhythmias including lidocaine and concomitant anesthetic drugs but then use Bernoulli's trial as a statistic to compare their data with Johnston's. The Bernoulli trial assumes mutually exclusive outcomes (arrhythmias, no arrhythmias), each of which has a constant probability of occurrence (same drug regimens). The probability of arrhythmias was not constant between the two groups.

My conclusion drawn from the data presented is that patients premedicated with barbiturates and given 1 mg of lidocaine for each microgram of epinephrine will tolerate up to one MAC of halothane without ventricular dysrhythmias.

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In reply: The fundamental issue in our survey, "Epinephrine–Halothane Interactions in Children,"[1] is whether the incidence of ventricular arrhythmias after epinephrine infiltration during halothane anesthesia is the same in children as in adults.[2] Calculated probabilities using Bernoulli trials clearly indicate that the incidence is different.

Discovering the reason for this difference is somewhat more difficult. Possible explanations include age, injection site, and the differences in drug regimens. Of the drugs employed, thiopental and nitrous oxide would be expected to make arrhythmias more likely,[3,4] whereas lidocaine in the injectate has been shown to increase the amount of epinephrine that may be administered before arrhythmias occur. The likelihood of each of these being the cause of the difference in incidence can be calculated exactly from a 2 × 2 contingency table using the multinomial distribution of marginal probabilities.[5] When a subset of our patient population is compared with a subset of Johnston's (table 1), one finds that our patients who received no lidocaine are clearly different from Johnston's halothane–saline group ($P = 0.012$). The difference between the groups is probably even greater because our patients received more epinephrine (15.7 μg/kg) than Johnston's (4 μg/kg). We also performed these calculations with the patients in our study who received no barbiturate in their preanesthetic medication (12) and those who were given higher doses of halothane. There was a clear difference.

We too are troubled by the use of drugs at potentially toxic levels. The majority of the patients in our study received high doses of two drugs, and the only untoward effects that we observed were one supraventricular arrhythmia and some tachycardia. Obviously one would only give these high doses if the drugs clearly are needed. "The surgeon injected a volume and concentration of epinephrine . . . sufficient to provide hemostasis."[1] The addition of lidocaine to the injectate may well have contributed to the complete absence of premature ventricular contractions (PVC) in our study as well as providing analgesia that contributed to a smooth anesthetic course. Further study is now needed, including measurement of

TABLE 1. Patients Receiving Epinephrine in Saline

	Johnston	Karl	Total
PVC	5	0	5
No PVC	5	11	16
Total	10	11	21

both lidocaine and epinephrine plasma levels as well as perhaps tighter control of adjunct drugs to clarify the results of our survey.

Until this information is available, we continue to believe that our initial conclusion holds: "10 μg/kg of epinephrine infiltration may be used safely in normocarbic and hypocarbic pediatric patients without congenital heart disease. . . . The occasional presence of premature atrial contractions and tachycardia in the child emphasizes the need for continuous ECG monitoring and caution during halothane anesthesia with epinephrine injection."[1]

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ASA Preceptorship: Success in a Rural Setting

To the Editor:—The goals of the ASA preceptorship program are to 1) expose the medical student to the specialty of anesthesia, 2) build the image of the specialty of anesthesia within medicine, and 3) to act as a "tutorial or Socratic program" for the student.* Medical students are being given the opportunity to see what an anesthesiologist does on a daily basis in hopes of influencing well-qualified applicants to apply for anesthesia residency programs.

Five medical students participated in the ASA preceptorship program at the Dixie Medical Center in St. George, Utah, during the period 1976–1981. St. George, Utah, is a community of 14,000 population with a 65-bed hospital that serves an estimated area of 21,000. Surgical specialties represented during the period covered included: Orthopedics, General Surgery, ENT, OB, Ophthalmology, and Urology. The anesthesia department consisted of two CRNAs and one anesthesiologist.

Each of the five participants enrolled in the 8-week program. Upon beginning the preceptorship, each student was interviewed by me to ascertain their medical goals. Not one of the five medical students wanted to become an anesthesiologist, in fact, all had chosen specialties: *i.e.*, Radiology, Family Practice, or Aerospace Medicine. Their purpose in participating in the program was twofold: to learn endotracheal intubation and to be exposed to critical care patient management.

A flexible program was designed to allow the student to be with the anesthesiologist for preanesthesia and postanesthesia rounds, intensive care rounds, and participate in administering anesthesia in the operating room. The students were given didactic lectures a minimum of two times per week on basic anesthesia subjects. Each student received a list of 21 subjects to choose from for discussion during the preceptorship. A bibliography of 52 basic anesthesia articles was given to the student to serve as a nidus for OR discussion.

Goals for the practical mechanical skills for the student included 1) endotracheal intubation, 2) intravenous therapy, 3) intraarterial cannulation techniques, 4) airway management, and 5) use of anesthesia equipment.

At the conclusion of the 8-week program each student again was interviewed. Three students had not changed their specialty goals, but two students now were considering anesthesiology as an alternate specialty to their initial choice. Follow-up on each student until July 1983 has been rewarding in relation to their initial attitudes toward anesthesiology and the goals of the ASA Preceptorship Program. One student completed a residency in anesthesiology at the University of Arizona and presently is practicing anesthesia in Ames, Iowa; one student completed a Family Practice residency, practiced in a rural area in Utah for 2 years, and is now a resident in anesthesiology at the University at Kansas; one student completed an internship, practiced 6 months as an emergency room physician, and now is a resident in anesthesiology at the University of Utah; one continued in Aerospace Medicine and one will complete a Family Practice residency July 1983.

* Guidelines for Preceptors. Subcommittee on Medical Student Preceptorships American Society of Anesthesiologists, 1976.