

expiration can occur. One means of doing this is to attach an anesthesia bag to the patient port and inflate the bag until it has positive pressure at the end of inspiration. This patient illustrates that faulty manual respirators may appear normal and can result in compli-

cations. To the anesthesiologist it stresses the importance of carefully checking equipment before and during its use. Pulmonary barotrauma should be high on the list of differential diagnosis in cardiorespiratory emergencies.

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Appraisal of Epinephrine Administration to Patients under Halothane Anesthesia for Closure of Cleft Palate

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To achieve optimal local hemostasis in patients undergoing general anesthesia, epinephrine frequently is injected. During the closure of a cleft palate in pediatric patients, the injection of epinephrine is important in this regard. However, when halothane is concomitantly used, cardiac arrhythmias may occur.

Katz *et al.*¹ suggested that 1 $\mu\text{g}/\text{kg}$ epinephrine is a safe dose for adults under halothane anesthesia. Johnstone *et al.*² demonstrated that 2.1 $\mu\text{g}/\text{kg}$ was the dose producing premature ventricular contractions (PVCs) in 50% of patients (ED50) when epinephrine in saline was injected for hemostasis during halothane anesthesia. When 0.5% lidocaine was added to the injection solution, the ED50 increased to 3.7 $\mu\text{g}/\text{kg}$. Melgrave³ and Wallbank⁴ found that children could tolerate greater amounts of epinephrine on a body-weight basis than adults. They used from 2.5 to 5.5 $\mu\text{g}/\text{kg}$ of epinephrine together with local anesthetics for pediatric patients under halothane anesthesia and found no arrhythmias.

The frequency of cardiac arrhythmias when epinephrine is used during halothane anesthesia in pediatric patients has not been determined. We, therefore, determined the incidence of cardiac arrhythmias when epinephrine was given together with halothane anesthesia for closure of cleft palate.

MATERIALS AND METHODS

The subjects studied were 50 patients (27 boys and 23 girls) who underwent closure of a cleft palate. All the patients were considered to range in risk from ASA I to II. The age range was from 13 to 20 (15.9 ± 1.5 SD) months, and the body weight range was 8.4 to 14.5 (10.4 ± 1.1 SD) kg. Parental consent was obtained for each child. The study design was approved by a human experimentation committee.

The patients were premedicated with im injections of atropine, 0.25 mg, and hydroxyzine, 12.5 mg. Halothane was inhaled in 100% oxygen via a non-rebreathing system. The patients breathed spontaneously at all times. Halothane 4% from a factory-calibrated vaporizer was given for 10 min and orotracheal intubation was performed without use of a muscle relaxant. Anesthesia was maintained with 2.5% halothane. One percent lidocaine with 1:100,000 epinephrine (LID-E) was injected by the surgeon. A dental block syringe with a 27-gauge needle was used for the LID-E injection. One milliliter of LID-E was administered every 30 s, and the volume of LID-E for closure of cleft palate was limited to 8 ml. The ECG was monitored continuously with an oscilloscope. If PVCs occurred during the injection, the injection was halted until the arrhythmia disappeared. After completion of the LID-E infiltration, we waited 5 min before starting surgery so that the effect of the epinephrine and lidocaine could be observed. The concentration of the inhaled halothane was gradually decreased to 0.7% during surgery, and the halothane discontinued 5 min before the end of surgery. The trachea was extubated when the patient regained consciousness.

RESULTS

The amount of LID-E used in this study varied from 6 to 11 (8.04 ± 1.06 SD) ml. The limit of 8 ml of LID-

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Key words: Anesthetics, volatile: halothane. Anesthetics, local: lidocaine. Anesthesia: pediatric. Sympathetic nervous system: catecholamine epinephrine. Complications: arrhythmia.

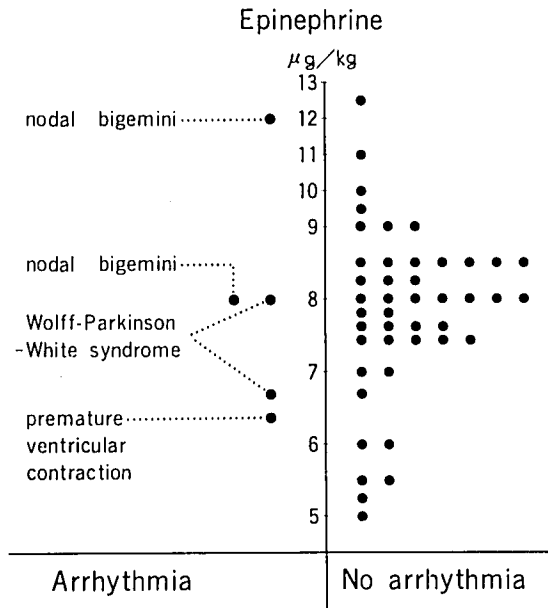


FIG. 1. Number of patients and amount of epinephrine used in this study. Each dot represents one patient.

E was surpassed in nine cases to perform trimming on a previously operated cleft lip at the same time. The injected epinephrine dosage varied from 5.0 to 12.4 (7.8 ± 1.5 SD) $\mu\text{g}/\text{kg}$ (fig. 1).

Changes in the heart rate appeared within 2 to 3 s after starting the LID-E injection and reached a peak in 2 min. The heart rate increased from 5 to 10% in 19 patients, and more than 10% in six patients. The increased heart rate of these 25 patients returned to the control rate in 5.2 ± 1.6 (SD) min. There was one patient whose heart rate decreased. No change in heart rate was observed in 16 patients (fig. 2).

Five cardiac arrhythmias were observed in five patients immediately after starting the LID-E injection. The heart rate of these patients did not increase. Two patients had nodal bigemini, two other patients had Wolff-Parkinson-White syndrome (WPW), and the remaining one patient had PVCs. The PVCs were not of the multifocal type and appeared regularly every five beats. These arrhythmias disappeared spontaneously before specific treatment was needed, and the total dose of LID-E was administered without further problem (fig. 1, table 1).

Two other arrhythmias appeared during the surgical procedure in two patients. One was PVCs induced by obstruction of the endotracheal tube, and the other was WPW due to vigorous pharyngeal suctioning. These two arrhythmias were corrected immediately by eliminating the causative problems.

All of the patients were able to tolerate the anesthesia and surgery without any complications.

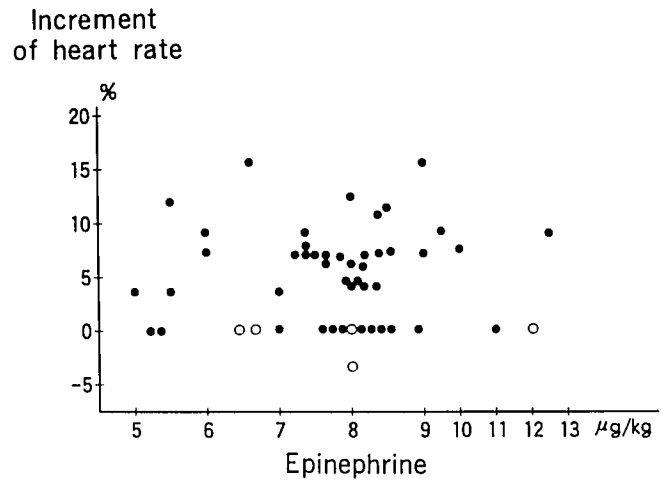


FIG. 2. Per cent increase in heart rate following epinephrine injection. Each circle represents one patient. Empty circles indicate the patients who developed arrhythmias.

DISCUSSION

This study shows that some patients develop cardiac arrhythmias following the use of epinephrine for the closure of cleft palate during halothane anesthesia.

Wallbank⁴ concluded that the circulatory changes that occur following epinephrine injection for harelip and cleft palate surgery are mainly due to systemic absorption of the epinephrine. In our study, however, changes in the heart rate and development of arrhythmias following LID-E injection were apparently caused by the irritation from the LID-E injection rather than by the exogenous epinephrine itself. We came to this conclusion because there was almost no difference in time between the start of the LID-E injection and the onset of the arrhythmias or the changes in heart rate. Also, the arrhythmias were transient and further administration of LID-E did not aggravate them. Our conclusion agrees with the findings of Kaufman,⁵ Forbes,⁶

TABLE 1. Arrhythmias and Changes in Heart Rate that Appeared in Five Patients Following Epinephrine Injection

Type of Arrhythmia	Duration (s)	Change in Heart Rate	Total Dose of Epinephrine ($\mu\text{g}/\text{kg}$)
Premature ventricular contraction	30	160 → 155	6.4
Wolff-Parkinson-White syndrome	10	160 → 160	6.7
Wolff-Parkinson-White syndrome	90	150 → 150	8
Nodal bigemini	10	130 → 130	8
Nodal bigemini	10	150 → 150	12

All arrhythmias appeared at the very beginning of the epinephrine injection.

and Alexander *et al.*^{7,8} They reported that a stimulus to the trigeminal nerve induced cardiac arrhythmias.

For the closure of cleft palate in our practice, the epinephrine solution was injected not only to minimize surgical bleeding but also to separate the periosteum and mucosa from the bone. The rather high concentration of halothane was necessary to maintain adequate anesthesia with such a potent surgical stimulus. We probably gave deeper anesthesia than did Melgrave³ or Wallbank,⁴ who used 0.5% halothane together with 50 to 60% nitrous oxide. A higher concentration of halothane may produce hypercarbia and greater sensitization of the myocardium to epinephrine; however, sympathetic discharge induced by stress can be suppressed with deeper anesthesia.⁹ These conflicting effects prevent us from making any conclusions regarding the influence of deep halothane anesthesia on the incidence of arrhythmias.

The heart rates of the patients who developed cardiac arrhythmias were either unchanged or decreased following LID-E injection. No arrhythmias were observed in the group of patients whose heart rates were increased by the injection (fig. 2, table 1). These findings support the explanation regarding the mechanism of development of arrhythmias under halothane anesthesia proposed by Hashimoto and Hashimoto.¹⁰ Halothane is known to depress sinus node activity. Any mechanism that produces further depression of the sinus node or activation of automaticity other than the sinus node of the heart creates favorable conditions for the occurrence of arrhythmias.

Johnstone *et al.*² demonstrated that lidocaine increased the ED₅₀ of epinephrine. They used 0.5% lidocaine in their study. One per cent lidocaine was used in this study, aiming at a stronger antiarrhythmogenic effect. Conversely, we avoided using nitrous oxide because of our clinical impression that this might enhance the arrhythmogenicity of epinephrine during halothane anesthesia. This impression has been supported by animal study results recently reported by Puerto *et al.*¹¹

and Liu *et al.*¹² Further studies are needed to clarify the effect of the lidocaine concentration in the epinephrine injection solution, and the arrhythmogenic effect of nitrous oxide in humans.

We conclude that a mean dose of epinephrine of 7.8 $\mu\text{g}/\text{kg}$ together with lidocaine can be used safely for the closure of cleft palate. The mechanical stimulus produced by the epinephrine injection can cause cardiac arrhythmias.

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