Anesthesiology
60:76, 1984

An Endotracheal Tube Fixation Device Constructed from Discarded Oxygen Tubing and Umbilical Tape

To the Editor:—During the course of clinical practice, an occasional patient presents as a challenge with respect to proper fixation of the endotracheal tube. With a section of discarded oxygen tubing and a length of standard umbilical tape, one can prepare devices suitable for fixation of the endotracheal tube while avoiding the use of tape or adhesives (fig. 1).

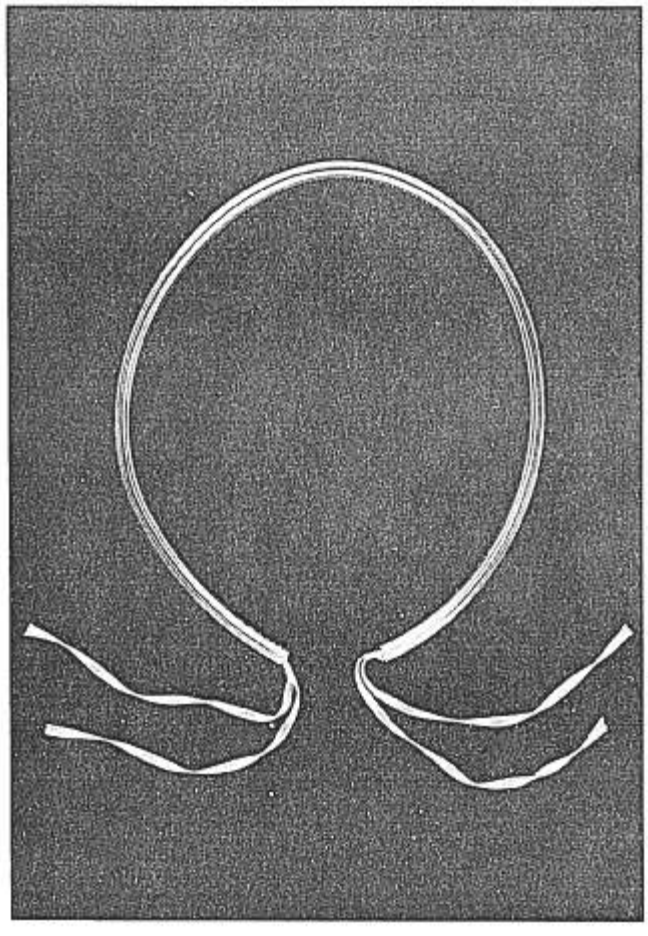

FIG. 1. Endotracheal tube fixation device.

The endotracheal tube tie-down device is useful for use in the patient allergic to adhesives, for the patient with an inconvenient abundance of facial hair, and for the patient with skin conditions that might be aggravated by direct contact with adhesives or adhesive tape. This endotracheal tie-down is comfortable to the patient and might prove useful to the practitioner for use on the intubated patient in the operating room or intensive care setting.

This device is prepared in advance of anticipated use as outlined. A single strand of umbilical tape is passed relatively easily down the oxygen tube lumen. After successful passage, the end is tied to the middle of a 60-inch length of umbilical tape. The single umbilical tape strand then is pulled through, yielding the double-stranded product. The double strand of umbilical tape then is knotted at both ends of the oxygen tube, thereby preventing the inadvertent removal of the tape from the tubing. An adult with a large head requires a tie-down prepared from oxygen tubing 17–19 inches in length, a smaller adult remains at 14–16-inch length, and a child requires a 10–12-inch length.

The oxygen tubing is placed behind the nape of the neck, and the umbilical tape is tied snuggly around the endotracheal tube shaft. Once the physician is satisfied with the proper placement of the tube and once assured of the security of the knot, the ends are trimmed as desired. The oxygen tubing is soft and comfortable to the patient and will not cut or abrade the skin, as might a cloth strip or naked tracheal tape.

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(Accepted for publication June 21, 1983.)

Anesthesiology
60:76–77, 1984

Epinephrine–Halothane Interaction in Children *versus* Adults

To the Editor:—Dr. Karl *et al.* reported on epinephrine–halothane interactions in children,[1] concluding that "children tolerate higher doses of subcutaneous epinephrine than adults during halothane anesthesia" and that "at least 10 μg/kg of epinephrine infiltration may be used safely in normocarbic and hypocarbic pediatric patients without congenital heart disease." Although both of these statements may be true, they should be qualified.

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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(Accepted for publication June 21, 1983.)

Anesthesiology
60:77–78, 1984

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