

uous positive airway pressure (CPAP)." Unfortunately, this dichotomy of terms, non-ventilated lung PEEP^{4,5} and non-ventilated lung CPAP,^{*6} persists in both current texts and journals.

There is nothing in Gregory's⁷ original description of the term CPAP that specifically precludes its use in apneic patients. In fact, many commonly used one-lung CPAP circuits are essentially identical to the circuit described by Gregory. In practice, the airway pressure in the one-lung CPAP circuit fluctuates due to surgical and mediastinal compression of the non-ventilated lung. Thus a pressure-time plot for the system would more closely resemble Dr. Lee's CPAP diagram than his CPP diagram.

We prefer using the term non-ventilated CPAP to non-ventilated lung PEEP because there is no defined expiratory phase, and it causes less confusion in terminology when combined with dependent-lung PEEP.

There are three obvious solutions to this problem: 1) devise a new acronym such as CPP, 2) use the term PEEP, and 3) continue with the term CPAP. None of these is a perfect solution. However, we feel that to change terminology at this late date would only add to the confusion, and we therefore prefer to retain the term CPAP.

* Cohen E, Eisenkraft J, Thys D, Kirschner P, Kaplan J: Oxygenation and hemodynamic changes during one-lung ventilation: Effects of CPAP₁₀, PEEP₁₀ and CPAP₁₀/PEEP₁₀. *J Cardiothorac Anesth* 2:34-40, 1988

Arrhythmogenic Threshold of Epinephrine during Sevoflurane, Enflurane, and Isoflurane Anesthesia in Dogs

To the Editor:—We determined the threshold for epinephrine (EPI)-induced ventricular arrhythmias in dogs anesthetized with isoflurane, enflurane, sevoflurane, or thiopental plus sevoflurane.

Anesthesia was induced and maintained in nine unpremedicated dogs with 1.3 MAC isoflurane, enflurane, sevoflurane, or thiopental (20 mg/kg), plus sevoflurane. End-tidal anesthetic concentration and P_{CO₂} were monitored continuously. Following trachea intubation without muscle relaxants, the dogs were ventilated to maintain normocapnia. A femoral vein was cannulated for infusion of EPI and a solution of 3% dextrose in 0.5% NaCl. A femoral artery catheter was inserted for intra-arterial pressure monitoring and arterial blood sampling. Nasal temperature was maintained at

37.5–38.5° C. Lead II of the ECG was monitored continuously. Arterial pH, P_{O₂}, P_{CO₂}, serum Na, and K were maintained in the range of 7.35–7.45, 90–150 mmHg, 35–45 mmHg, 135–150 mEq/l, and 3.5–4.5 mEq/l, respectively.

Arrhythmogenic dose (AD) of EPI was established by logarithmically spaced infusions of EPI lasting 3 min with at least 10 min between infusion.¹ In this procedure, the infusion was started at the minimum dose of 2.2 μg · kg⁻¹ · min⁻¹, and the dose was increased by e^{0.4} (e = 2.72) (3.3, 5.0, 7.4, and 11.1 μg · kg⁻¹ · min⁻¹) until the arrhythmias occurred. If there were the arrhythmias at one of these doses, a smaller dose, which was given by that dose divided by e^{0.2}, was tested. When there were the arrhythmias at 3.3 μg · kg⁻¹ · min⁻¹, a

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TABLE 1. Arrhythmogenic Thresholds of Epinephrine during Four Types of Anesthesia in Dogs (Mean \pm SEM)

Anesthesia	N	Arrhythmogenic Threshold of Epinephrine	
		Dose ($\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	Plasma Level (ng/ml)
Sevoflurane	9	8.57 \pm 1.18*	175.2 \pm 27.7*
Thiopental plus sevoflurane	8	5.11 \pm 0.68†	106.8 \pm 15.1†
Enflurane	9	5.17 \pm 1.00*	100.5 \pm 24.5*
Isoflurane	6	9.81 \pm 1.26*	207.3 \pm 31.3*

* $P < 0.05$, compared to each other.† $P < 0.05$, compared to sevoflurane value.TABLE 2. Mean Blood Pressure and Heart Rate at the Time of Arrhythmias (Mean \pm SEM)

Anesthesia	N	Mean Blood Pressure (mmHg)	Heart Rate (rate/min)
Sevoflurane	9	214 \pm 15.1	123 \pm 12.8
Thiopental plus sevoflurane	8	210 \pm 10.2	137 \pm 9.0
Enflurane	9	209 \pm 14.6	143 \pm 9.3
Isoflurane	6	229 \pm 24.6	128 \pm 15.1

smaller dose of $2.7 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($3.3 \div e^{0.2}$) was tested, and, similarly, in the case of 5.0, 7.4, or $11.1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, a smaller dose of 4.1, 6.0, or $9.0 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ was tested, respectively. Unless the arrhythmias were observed at $11.1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, maximum dose tested was $13.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. The AD was defined as the dose that produced four or more premature ventricular contractions within 15 s.¹ The AD of EPI during each of four types of anesthesia was obtained on separate days (minimum 7 days interval) in each of the animals under similar conditions. A blood sample was collected at the time when the criterion for the AD was satisfied. The plasma EPI concentration was measured by automated fluorimetric analysis.²

The Wilcoxon signed rank test was used for comparing arrhythmogenic threshold with isoflurane and other anesthetics. *t* test for paired data was used for all other comparisons. $P < 0.05$ was considered significant.

The AD and the corresponding plasma level of EPI during each anesthesia are presented in the table 1. During thiopental plus sevoflurane anesthesia, no data were obtained in one of the nine dogs because of circulatory arrest. During isoflurane anesthesia, no AD was reached in three dogs at the maximum dose of EPI, and the results of the remaining six dogs were used to calculate the mean value. There were no significant differences among the hemodynamic data during the four types of anesthesia (table 2).

The results show that, among three anesthetics without thiopental induction, the arrhythmogenic plasma

level of EPI was highest during isoflurane anesthesia, lowest during enflurane anesthesia, and intermediate for sevoflurane. The different potencies for myocardial sensitization among these anesthetics were apparently not due to differences in the effect of EPI on blood pressure or heart rate.

The arrhythmogenic threshold of EPI during isoflurane, enflurane, and sevoflurane in this study was much higher than that reported previously during halothane anesthesia.³ The results also show that thiopental lowered the arrhythmogenic threshold during sevoflurane anesthesia, as has also been reported when administered during anesthesia with halothane,⁴ enflurane,⁵ or isoflurane.⁵

The AD of EPI for enflurane and isoflurane were similar to those reported by Atlee *et al.*⁵ The enflurane value was lower than that reported by Sumikawa *et al.*³ This difference may be due to the different manner in which EPI was administered, *i.e.*, in our present study, we started the EPI infusion at a higher dose and the number of challenges with EPI to obtain AD was decreased. Furthermore, a longer recovery period (10–40 min) was allowed for hemodynamic parameters to return to control values. It is likely that these differences would reduce the tolerance to EPI.⁵

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How to Make Tape Stick to Sandpaper

To the Editor:—Although the literature on how to properly tape an endotracheal tube to a patient's face is plentiful, we would like to add another report and a self-explanatory pertinent illustration on this subject.

Whenever a patient's skin is either greasy, hairy, or otherwise so uneven that normal plastic or cloth tape will not adhere properly, we use the following technique.

Once endotracheal intubation has been performed and proper tube position is confirmed, two small rectangles (3 × 7 cm) of transparent dressing (Tegaderm™, 3M, St. Paul, Minnesota) are placed on the skin overlying the cheeks or the zygomatic arch, forming a "second skin." The endotracheal tube can then be secured by the operator in the usual fashion, with the adhesive tape applied to the Tegaderm™. Contrary to regular tape, adhesion of the Tegaderm™ "second

skin" does not deteriorate with time. Even after prolonged intubation, contact remains excellent and tube displacement is very unlikely to occur. For extubation, tape is easily removed together with the Tegaderm™.

We recommend this as an efficient and clean technique to make tape stick to any patient's skin.

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A Method of Facilitating Intravenous Regional Bretylium

To the Editor:—Ford *et al.*¹ recently described intravenous (iv) regional bretylium for treatment of patients with reflex sympathetic dystrophy (RSD). We have used this technique several times with varying success. However, major problem in utilizing this technique is difficulty in establishing venous access due to the hypersensitivity and vasoconstriction caused by the RSD. We describe an approach to facilitate iv access in these patients.

A young woman presented to our Pain Clinic with a 2-month history of RSD of the right foot following an industrial accident. An iv bretylium blockade of her leg was accomplished only after great difficulty in establishing iv access. The patient had excellent pain relief for 36 h, and a repeat block was planned for the following week. Multiple attempts, including the use of nitro-paste ointment, to start an iv were unsuccessful. After

appropriate consent was obtained and adequate prehydration given a 17-gauge Tuohy needle was inserted into the epidural space *via* the L₃₋₄ interspace. Ten milliliters of .25% Marcaine was administered with the patient in the sitting position. The patient developed partial pain relief of her foot and minimal vasodilation. A 22-gauge angiocatheter was inserted and an iv bretylium block was done resulting in complete analgesia.

In summary, establishing an iv in a limb afflicted with reflex sympathetic dystrophy is often difficult because of hypersensitivity and vasoconstriction. Epidural sympatholytic block producing venodilation and some analgesia facilitates obtaining iv access for a regional block. A possible extension of this technique for an upper extremity would be sympatholysis *via* stellate ganglion blockade.