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REFERENCES

1. Seagard JL, Elegbe EO, Hopp FA, Bosnjak ZJ, von Colditz JH, Kalbfleisch JH, Kampine JP: Effects of isoflurane on the baroreceptor reflex. ANESTHESIOLOGY 59:511–520, 1983
2. Seagard JL, Hopp FA, Kalbfleisch JH, Kampine JP: Halothane and the carotid sinus reflex: Evidence for multiple sites of action. ANESTHESIOLOGY 57:191–201, 1982

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In reply:—As indicated in the original article,[1] thiopental was found to blunt the depressor response of the baroreceptor reflex produced by increasing blood pressure through infusion of phenylephrine. The slope of the bradycardic heart rate–blood pressure curve is the depressor component of the baroreflex and was found to be significantly depressed by thiopental alone at 0.0% inspired isoflurane. This action may be due in part to the proposed vagolytic effect of thiopental. The unfortunate error of noting "at 1 MAC (isoflurane) the bradycardic responses to decreases in pressure were not different from control" is a mistake that should have been corrected but was not noticed in the revisions. We hope that the presentation of data in table 1 and the discussion in "Results" was sufficient to convey the information in spite of the error in the "Discussion."

The missing numeric values for carotid sinus nerve activity recordings, which should have been presented in Table 2, were included in the original manuscript but not in the final revision of the paper. The authors apologize for their apparent omission. The values, presented as the slopes of the carotid sinus nerve activity *versus* carotid sinus pressure (spikes · 100 $ms^{-1} \cdot mmHg^{-1}$) are 0.22 ± 0.05 (0.0% isoflurane), 0.36 ± 0.06* (1.3% isoflurane), and 0.42 ± 0.06† (2.6% isoflurane), with * = $P < 0.01$ *versus* 0.0% isoflurane and † = $P < 0.01$ *versus* 0.0% isoflurane and $P < 0.05$ *versus* 1.3% isoflurane. Figure 2 presents the data from one animal, while the slopes are the mean results from six animals. All individual slopes utilized in the analysis were significant, with correlation coefficients of 0.7 or greater, as indicated in the earlier study.[2]

The presentation of preganglionic and postganglionic sympathetic efferent nerve activity in table 3 included both "*baseline*" and "*reflex changes*" in activity, with both sets of data presented as % of baseline (control) levels at 0.0% isoflurane. This is similar to the method used in a previous article[2] (tables 2 and 3), although the arrangement of the table was changed to present a more concise summary of the results. It was assumed, perhaps wrongly, that the direction of the reflex change would be apparent, based on the general understanding of the baroreflex, the discussion in "Results," and the accompanying figure 3.

The infusion rates were actually in μg/min and the authors apologize for this error. The actual dose employed, while informative, is not as important as the knowledge that the same degree of hypotension or hypertension was produced in all the animals.

Finally, the study presented in this article is a complex series of smaller studies designed to determine the effects of isoflurane on the entire baroreflex arc. By necessity, this requires the use of different methods to investigate the complete actions of this agent. Many previous studies examining the actions of anesthetics on cardiovascular reflex control generally have been studies examining at best only a few actions of an anesthetic. This has produced a large amount of conflicting data due to the variety of anesthetic techniques, including inductional agents, basal anesthetics, and different levels of inhalational anesthesia. In some studies variables such as blood pressure, preload, and cardiac output have been regulated, while in others, these factors were allowed to vary with anesthesia. The authors of this article feel, therefore, that although the present study is indeed complex, it is important to present a thorough and complete investigation in which similar preparations were utilized to examine the multiple actions of the anesthetic on baroreflex regulation. This type of study hopefully will provide some important information that will contribute to a clearer picture of the effects of inhalational anesthetics on cardiovascular reflex control.

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REFERENCES

1. Seagard JL, Elegbe EO, Hopp FA, Bosnjak ZJ, von Colditz JH, Kalbfleisch JH, Kampine JP: Effects of isoflurane on the baroreceptor reflex. ANESTHESIOLOGY 59:511–520, 1983

2. Seagard JL, Hopp FA, Kalbfleisch JH, Kampine JP: Halothane and the carotid sinus reflex: Evidence for multiple sites of action. ANESTHESIOLOGY 57:191–201, 1982

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A Simple Device for Oxygen Insufflation with Continuous Positive Airway Pressure during One-lung Ventilation

To the Editor:—Hypoxemia is a significant clinical problem in approximately 20% of patients for whom a one-lung ventilation (OLV) anesthetic technique is utilized. Oxygen insufflation to the upper, nonventilated lung with continuous positive airway pressure (CPAP) is reported to increase Pa_{O_2} during OLV.[1,2] We have constructed an inexpensive, reusable, and readily available device that provides O_2 insufflation with variable CPAP to the upper, nonventilated lung during OLV (fig. 1).

A modified Ayre's T-piece (essentially a Mapleson E system) is connected to the lumen of a double-lumen endotracheal tube that supplies the nonventilated lung. The fresh gas port of the T-piece is connected to an oxygen source, either a tank or a wall outlet, via an appropriate flowmeter. A reusable valve designed to produce positive end-expiratory pressure (Boehringer Laboratories, Wynnewood, Pennsylvania) is connected to the corrugated tubing at the end of the T-piece assembly.[3] Using this system clinically, we have observed

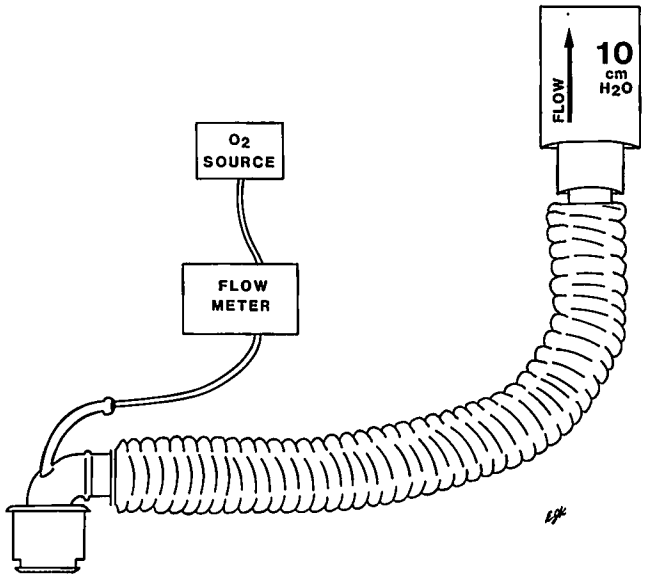

FIG. 1. A modified Mapleson "E" open-ended anesthesia circuit is connected to an oxygen source via a flow meter, and to a Boehringer "PEEP" valve at the distal end. When the system is connected to the nonventilated lung with an oxygen flow rate of 8 $l \cdot min^{-1}$, this circuit will provide oxygen insufflation; the level of CPAP is indicated on the valve, provided the valve is fixed in the vertical position.

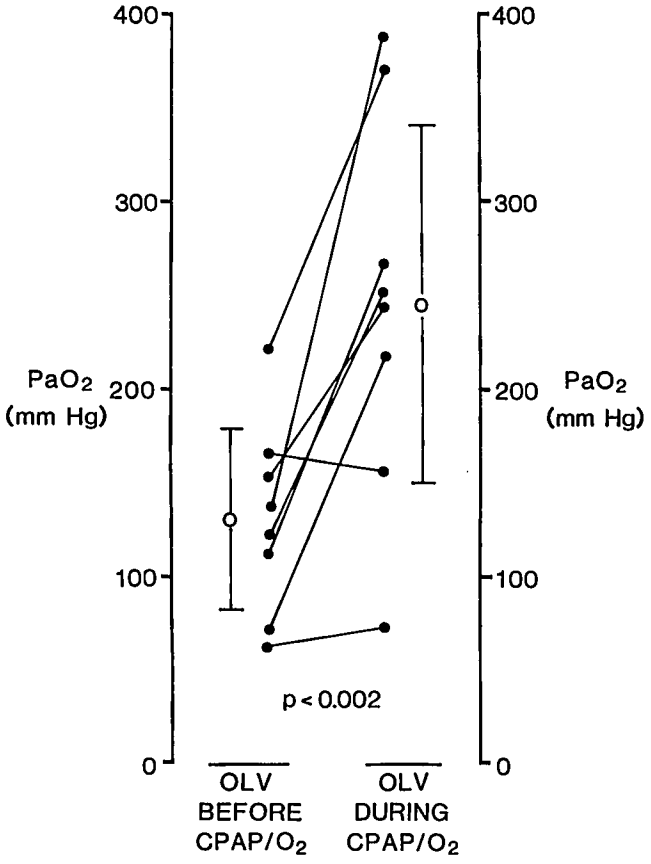

FIG. 2. Arterial oxygen tension (Pa_{O_2}) measured after 20 min of (OLV) without CPAP/O_2 is shown compared with similar data obtained 20 min after starting CPAP/O_2 to the nonventilated lung. Open symbols show mean values for each group with brackets to indicate one standard deviation.