

on the cart. These components included a suction male quick coupler, a receptacle, a length of suction tubing, and a Yankauer suction. The participants were told of the test beforehand but were not provided with practice trials. A stop watch was used for timing each participant.

The average time required for assembling the suction unit was 95.8 s (table 1). No individual took less than 35 s, one took more than 200 s. Only three individuals were able to assemble the unit to function in less than 1 min.

It seems unlikely that aspiration could be avoided in an actual emergency when using this equipment considering the time delays noted. The presence of disassembled suction units on a cart should not be construed as "availability" of wall suction. We recommend that assembled wall suction units be kept in all labor rooms, ready for immediate use whenever regional anesthesia is administered. We further recommend that routine drills in the use of emergency equipment be carried out for all members of anesthesia departments, regardless of their level of prior training or experience.

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Complex Effects of Isoflurane on Baroreceptor Reflex Compounded by Errors

To the Editor:—The article by Seagard *et al.*¹ on the effect of isoflurane on the baroreceptor reflex addresses an important physiologic mechanism. Although the methods are well designed and suggest sophisticated technical accomplishments, the data are presented poorly, with errors and omissions, making subsequent conclusions of dubious value.

The authors state that thiopental anesthesia significantly depressed the depressor but not the pressor response of the baroreceptor reflex. The data in table 1 show just the opposite, namely a significant reduction in bradycardic response—pressure slope to increasing blood pressure from 59.4 ± 16.5 ms/mmHg in conscious dogs to 23.4 ± 12.8 ms/mmHg in thiopental dogs. In their discussion, the authors reason that the vagolytic effect of thiopental was responsible for the depression of the depressor response. Any vagolytic effect would attenuate the bradycardic response of increasing pressures, not the depressor response as the authors suggest. Further in the discussion, they note "at 1 MAC (isoflurane) the bradycardic responses to decreases in pressure were not different from control." Surely they mean to suggest that the bradycardic response to *increase* in pressure is unaffected by 1 MAC isoflurane anesthesia as is indicated by the data.

In their results on the carotid sinus nerve recordings, the authors present no numeric data to clarify the confusing scatter of points on the nerve activity—carotid sinus pressure graph in figure 2 (we are referred to table 2 but the data are absent). The best fit lines, if indeed they are accurately drawn, would seem to have very wide 95% confidence limits. To conclude from this that "isoflurane produces a dose dependent increase in baroreceptor activity" is questionable.

In table 3 the legend states preganglionic and postganglionic nerve activity is expressed as per cent baseline level. Stating that sympathetic efferent nerve activity fell to $6.27 \pm 3.07\%$ of control in response to decreases in blood pressure must be an error. In an earlier publication,² these authors present similar data as a per cent of *change*, in which case the reader needs to know the direction of change.

The infusion rates of nitroprusside and phenylephrine (100–300 mg/min and 10–50 mg/min, respectively) seem lethal. Since we do not know the duration of infusion, however, total dosages are unknown.

Finally, we feel that considerable confusion is caused by the combination of four different methods into one publication. In our opinion, it would be more appropriate to present each as a separate publication so the reader can better analyze the data and conclusions presented.

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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 · 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 article² (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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