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(Accepted for publication June 21, 1977.)

Anesthesiology
47:477-478, 1977

In reply—The mean total dose of thiopental that we used was 27.3 instead of 17.3 mg/kg body weight. I apologize for having overlooked this typing error. This dose was enough to permit surgical operation without manifestation of discomfort by the dogs. During this anesthesia and ventilation of the lung with pure oxygen, the average control values for mean aortic pressure, heart rate and diastolic coronary blood flow in the working heart were 117 ± 20 (SD) mm Hg; 162 ± 25 beats/min, and 52 ± 21 ml/min, respectively. These absolute values should have been reported.

We used as an index of the effect of halothane on the cardiovascular system the decrease in aortic pressure to a steady value of about 50 per cent of control. This was obtained after 10-20 minutes of halothane, 2-3 per cent. Therefore, the measurements were performed during a marked effect of the drug on the cardiovascular system and at least at a time of a steady aortic pressure. In those dogs in which the administration of halothane was repeated, we waited until the variables returned to control values. This does not mean that halothane was completely eliminated, but the changes produced by the second administration were similar to those produced by the first.

Our measurement techniques were not particularly sophisticated, and we lacked equipment to measure anesthetic concentration or blood pH and P_{CO₂}. Changes in the two last variables may have occurred during halothane. However, since the left ventricular oxygen consumption decreased during halothane, a decrease in myocardial P_{CO₂} and therefore an increase in diastolic coronary resistance could be expected,¹ instead of a decrease as we observed.

In the experiments in which we maintained aortic pressure with aortic coarctation, the decrease in coronary resistance was accompanied by a decrease in the coronary arteriovenous difference of O₂.

In the non-working-heart preparation, the circulation to the central nervous system was discontinued. Even if any central impulses to the coronary arteries

had persisted in this condition, these would not have been influenced by halothane, since the anesthetic did not have access to centers of central autonomic connections.

I think that in the discussion of our report the comments about the results of Vatner and Smith are clear.² They found no increase in diastolic coronary resistance during administration of a high dose of halothane in spite of a large decrease in the magnitude of the variables that determine myocardial oxygen consumption. In other words, coronary vascular resistance did not increase as expected in response to the metabolic coronary autoregulation. The authors interpreted this result as a vasodilatory effect of the anesthetic on the coronary arteries. It seems to me that a similar result is observed in the report of Merin *et al.*³ In fact, in Merin's experiments high concentrations of halothane produced a decrease in myocardial oxygen consumption but no increase in coronary vascular resistance, as should be expected. Our results, mainly those obtained in the arrested heart in which the effect of changes in the mechanical, metabolic and neurohumoral factors that influence coronary resistance can be to a large extent avoided, agree with the concept of a direct vasodilatory effect of halothane on the coronary arteries.

We thought that our results were worthy of being reported because they describe the effect of a very commonly used anesthetic on a very important vascular bed, but we have not suggested that this is the major effect of halothane on the coronary circulation. Both direct effects of the drug superimposed on the indirect effects mediated through the metabolic coronary autoregulation must be considered. The importance of the direct effects may depend on the special circumstance prevailing in the coronary circulation at any point in time.

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(Accepted for publication June 21, 1977.)

Anesthesiology
47:478, 1977

Bronchospasm in the Operating Room

To the Editor:—Sprague's clinical study of the treatment of bronchospasm defined bronchospasm as expiratory wheezing with an increase in peak airway pressure.¹ This kind of bronchospasm can easily be produced in many patients by simply increasing the inspiratory or expiratory flow rate as well as by light anesthesia and inadequate paralysis. In 20 years I have administered more than 12,000 general anesthetics and have encountered only two or three cases of true bronchospasm, *i.e.*, bronchoconstriction severe enough to require treatment with a bronchodilator. Thus, it would seem Dr. Sprague has either collected these cases over a long time span, has a very large anesthetic case load, or practices in an institution with a high incidence of bronchospasm, or that his diagnosis is in error.

The highest peak airway pressure reported was 45 cm/H₂O with a tidal volume of 900 ml, and the decrease in peak airway pressure averaged only 2.2 cm/H₂O. As much as I dislike subjecting my pet biases to statistical analysis, I must admit that analysis of the figures for peak pressure might show whether the decrease was fact or fancy. Both the small change in compliance and tidal volumes of 700 to 900 ml are to me incompatible with true bronchospasm. According to the article "An attempt was made to rule out and correct, if necessary, the presence of

inadequate anesthetic depth." I would not expect deepening nitrous oxide-narcotic anesthesia with more narcotic to have any effect on bronchospasm. I would have expected the nine patients given halothane as the primary anesthetic agent to have improved compliance with increasing anesthetic depth. Nine of the patients, *i.e.*, patients 3, 5, 8, 9, 10, 11, 12, 13, and 14, had peak airway pressure changes of 0-2 cm/H₂O. If these were the patients given halothane, this might explain why their response to treatment with isoetharine was somewhat less than dramatic. Isoetharine has been shown to be effective in the treatment of bronchospasm, but the evidence presented by Dr. Sprague neither proves nor disproves that isoetharine is effective in the treatment of intraoperative bronchospasm.

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(Accepted for publication June 27, 1977.)

Anesthesiology
47:478-479, 1977

In reply—In my study, the diagnosis of bronchospasm was made on the basis of an increase in peak airway pressure associated with the occurrence of expiratory wheezing. Wheezing and increased airway pressure can occur in patients with high airway flow rates, light levels of anesthesia, or inadequate muscle relaxation; however, these factors were ruled out prior to treatment by observing the effects of changing flow rates on and off the ventilator, by increasing the depth of anesthesia when cardiovascular dynamics allowed, and by administering a muscle relaxant when deemed necessary.

Dr. Barbée implies that he would not treat bronchospasm until the compliance was very low. I do not agree with this view. Any degree of wheezing com-

bined with a change in peak airway pressure is abnormal, and measures should be taken to detect and correct the cause. In my study, the early treatment of bronchospasm may account in part for the small changes in peak airway pressure. Statistical examination of these changes was purposely not included in the paper because it was believed that these types of data in the given patient population did not lend themselves to statistical analysis. However, if a *t* value for the difference between means using paired comparisons is calculated, a significant decrease ($P < 0.001$) in peak airway pressure is indeed found.

Dr. Barbée suggests that the small changes in peak airway pressure in nine patients may have been the result of using halothane as the primary anesthetic.