REFERENCES


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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. 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 cmH₂O with a tidal volume of 900 ml, and the decrease in peak airway pressure averaged only 2.2 cmH₂O. 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 cmH₂O. If these were the patients given halothane, this might explain why their response to treatment with isothiopane was somewhat less than dramatic. Isothiopane has been shown to be effective in the treatment of bronchospasm, but the evidence presented by Dr. Sprague neither proves nor disproves that isothiopane is effective in the treatment of intraoperative bronchospasm.

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REFERENCE


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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. Barbee implies that he would not treat bronchospasm until the compliance was very low. I do not agree with this view. Any degree of wheezing combined 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. Barbee suggests that the small changes in peak airway pressure in nine patients may have been the result of using halothane as the primary anesthetic.