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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/H_2O with a tidal volume of 900 ml, and the decrease in peak airway pressure averaged only 2.2 cm/H_2O. 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/H_2O. 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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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.

This in fact was not the case. In the nine patients who received halothane, airway pressures decreased an average of 4.3 cm H_2O, compared with 3.4 cm H_2O in patients anesthetized with nitrous oxide–narcotic. Also, no marked difference in the decreases in airway pressure was found between patients with histories of bronchospastic pulmonary disease and those without. Peak airway pressures decreased an average of 4.4 cm H_2O in the ten patients with pulmonary disease and 4.1 cm H_2O in the six patients without pulmonary disease.

In conclusion, I believe that bronchospasm during anesthesia is a more common entity than suggested by Dr. Barbee.

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Temperature and Density of Tetracaine

To the Editor:—Dr. Landmesser[1] correctly points out that the commonly used hypobaric dibucaine solution contains 0.66 mg/ml, *i.e.*, 0.066 per cent. My reference to this solution[2] was not meant to imply, however, that density was related to drug concentration only. Density is defined as the mass of a unit volume of a material at a given temperature. Obviously, two compounds of different densities can be mixed with a third to make the same concentration. However, given the same solvent the density of this final mixture will be a function of the densities of the solutes.

Dr. Landmesser is critical of the reporting of density of tetracaine solutions at 23–25 C rather than 37 C. However, under usual clinical circumstances tetracaine solutions are not warmed to 37 C prior to injection. Although it probably takes about a minute for the injected solution to reach body temperature,[3] this has not been proven *in vivo*. Even *prior* to warming to body temperature, solutions of 0.33 per cent tetracaine–water and less are lighter than CSF at 37 C. The densities of tetracaine–water mixtures at 37 C are also, as would be expected, a linear function of dilution (fig. 1). However, it remains to be determined whether solutions of tetracaine that, at 37 C, are hypobaric to CSF at 37 C, but at 23–25 C are equidense with CSF at 37 C, behave clinically as isobaric or hypobaric mixtures.

Finally, the term "baricity" is not synonymous with density. Baricity is a relative term: "The weight of one substance compared to the weight of another substance at the same temperature."[4] When used in relation to spinal anesthesia, the "other substance," *i.e.*, CSF, is usually understood. However, for maximum clarity, the temperatures of both the local anesthetic solution and CSF have to be clearly specified.

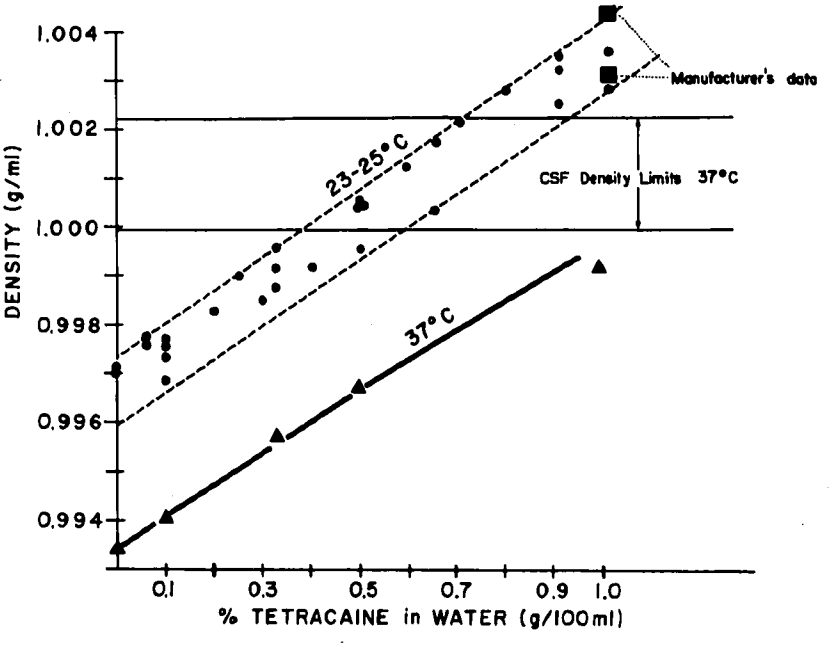

FIG. 1. Densities of tetracaine–water mixtures at 23–25 C and at 37 C compared with CSF at 37 C ± 3 SD.[5] Filled circles are individual determinations at 23–25 C, triangles are means of at least three determinations at 37 C.