Correspondence

IPPB-induced Respiratory Alkalosis

To the Editor:—The stated purpose of the article entitled: "Urinary Output and Plasma Levels of Antidiuretic Hormone during Intermittent Positive-pressure Breathing in the Dog" (ANESTHESIOLOGY 32: 17, 1970), by Baratz *et al.*, was "to determine if IPPB would produce changes in the intrathoracic blood volume or airway mechanics that could be correlated with changes in plasma ADH and urinary output."

If the authors were primarily interested in the effect of IPPB on ADH release mediated through increased activity of the volume receptor mechanism, the results would be more definitive had Pa_{CO_2} been kept constant throughout the experiment. Since IPPB as applied in this study "did not impose any stress on the circulatory system," other possible causes for the changes observed should be sought.

Two variables which differed significantly from control values were Pa_{CO_2} and *p*H. The degree of alteration (Pa_{CO_2} 23 mm Hg and *p*H 7.52) from control values (Pa_{CO_2} 39 mm Hg and *p*H 7.35) was such that significant alterations of cerebral blood flow, renal blood flow, and activity of the sympathetic nervous system could be expected. Furthermore, the effect of respiratory alkalosis on pharmacodynamics of ADH must be considered. Therefore, perhaps a more appropriate title for the article would have been "The Effects of IPPB-induced Respiratory Alkalosis on Urinary Output and Plasma Levels of Antidiuretic Hormone in the Dog." Unfortunately, the authors have ignored respiratory alkalosis in both experimental design and discussion of results. As a result, I can only conclude that IPPB sometimes increases ADH secretion (Baratz and Ingraham, Amer. J. Physiol. 198: 565, 1960), and at other times decreases ADH secretion (Baratz *et al.*, ANESTHESIOLOGY 32: 17, 1970). The mechanism by which IPPB has produced diametrically opposed results remains an area of speculation, *i.e.*, respiratory alkalosis *vs.* alteration of intrathoracic blood volume.

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To the Editor:—The purpose of Period 2 in the experimental design was to try to match IPPB with spontaneous respiration, so that pulmonary mechanics and blood gases during the two periods would be similar. The results, shown in tables 2 and 3 (ANESTHESIOLOGY 32: 17, 1970), show that this was accomplished, and accordingly there was no difference between the renal studies in Periods 1 and 2.

During Period 3, when IPPB was doubled by increasing the tidal volume, the idea was to try to change both pulmonary mechanics and blood gases and thus simulate the conditions produced during clinical use of IPPB. That the degree of respiratory alkalosis during this period can produce significant changes in cerebral blood flow, renal blood flow and sympathetic nervous system activity cannot be denied. We also felt this had to be investigated, and recently published further work (ANESTHESIOLOGY 33: 345, 1970) which defines more clearly the mechanism by which IPPB produces changes in plasma ADH levels and urinary output.

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