

I do not mean for these comments to detract from the report of DeKrey, Schroeder, and Buechel. They are to be commended for extending the safety of brachial plexus anesthesia to patients who might otherwise have had to undergo general anesthesia.

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The Effects of Diazepam

To the Editor:—Dr. James Dalen's article the Hemodynamic and Respiratory Effects of Diazepam, *ANESTHESIOLOGY* 30: 259, 1969, was most interesting.

Several data in table 1 are puzzling. Perhaps you can clarify apparent inconsistencies:

Patient 9

P_{aCO_2}		pH	
Control	33	Control	7.45
10 min	39	10 min	7.47

(? increase in P_{aCO_2} with increase in pH).

Patient 10

Tidal volume		P_{aO_2}	P_{aCO_2}	pH
Control	385 (20/min)	89	47	7.38
10 min	389 (19/min)	111	52	7.45
30 min	626 (15/min)	98	41	7.46

(? increase P_{aO_2} of 22 mm ? increase in pH of 0.01 with 11 mm decrease in P_{aCO_2} and normal P_{aO_2} ? increase of P_{aCO_2} with increased tidal volume and a drop of minute ventilation of 0.0 l/min, when P_{O_2} has increased. Also, again an increase of pH from 7.38 to 7.45, with an increase in P_{aCO_2} of 47 mm to 52 mm?)

Patient 11

A decrease in P_{aCO_2} from 54 mm to 45 mm produced (over 20 minutes), an increase of 0.01 pH units in a patient with a control P_{aCO_2} of 49 mm and a pH of 7.51 (presumably a metabolic alkalosis).

These inconsistencies are troublesome. Unless there is some other obvious reason, it

would seem a likely explanation is laboratory error.

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To the Editor:—We appreciate the questions of Dr. Weaver regarding our article.

The first question relates to inconsistencies in P_{aCO_2} and pH. I think that it is clear that the observed changes in pH were not as consistent as the changes in P_{aCO_2} , as noted. In table 1 all 15 patients showed increases in P_{aCO_2} ten minutes post-diazepam. That the pH changes were not as consistent indicates that we had not measured pH as accurately as P_{aCO_2} . I believe that one of our major points, namely, that we observed transient hypoventilation after diazepam, could well be made on the basis of the changes in tidal volume, P_{aO_2} and P_{aCO_2} . The changes in pH, I believe, are not necessary to establish this point.

Patient 10 certainly does stand out as showing unusual responses. I am at a loss to explain how the P_{aO_2} at 10 minutes could increase without a change in minute ventilation. Similarly, changes in P_{aCO_2} were not consistent with the observed changes in P_{aO_2} . I believe that the unusual changes in this patient could well have been related to the fact that

his cardiac lesion was an atrial septal defect with a very large left-to-right shunt. In patients with atrial septal defect, in the absence of pulmonary vascular disease, not only is pulmonary blood flow very large (in his case, ranging from 18 to 25 l/min), but in addition, the tremendous increase in flow is quite labile. It seems likely that his cardiac output was quite labile secondary to the left-to-right shunt. This may well be the explanation for the discrepancies in his ventilatory changes and arterial blood gases.

Our major point in regard to the respiratory effects of diazepam was that it caused a consistent, but modest, degree of hypoventilation. I hope that the apparent inaccuracies in the measurement of pH have not obscured this finding. We thank Dr. Weaver for his interest.

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Life Expectancy among Anesthesiologists

To the Editor:—After reading Dr. Bruce's article, "Causes of Death Among Anesthesiologists," in the May-June, 1968 issue of *ANESTHESIOLOGY*, I was interested to note the unusually high number of deaths listed in the *ASA Newsletter* for January, 1969.

In the Directory of the American Medical Association, I found the ages of 26 of the 28 deceased. It is alarming to note that the majority of these members died before reaching the average national life expectancy:

- 3 in the 30-year age group
- 3 in the 40-year age group
- 12 in the 50-year age group
- 5 in the 60-year age group
- 3 in the 70-year age group

Thus, 18 of the 26 died before the age of 60. In view of these figures, it is interesting to speculate on the causes of death: were they due to professional hazard, or the recent epidemic of respiratory disease? Or, perhaps, doctors who had been in poor health had switched from other medical practice into anesthesiology with the expectation that it would be less strenuous? I think the subject merits further study.

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Obstetrics and Pediatrics

FETAL ACIDOSIS The maternal and fetal cardiovascular and acid-base changes resulting from spinal hypotension and its subsequent correction with ephedrine were studied in eight ewes. When ephedrine was used to correct spinal hypotension, the fetal deterioration which had already occurred was arrested. With the restoration of maternal arterial pressure there was usually an improvement in fetal oxygenation, carbon dioxide elimination and fixed acid excretion. However, when the spinal hypotension was accompanied by maternal hypoxia and hypercarbia, the correction of maternal hypoxia with maternal oxygen administration was not accompanied by correction of fetal hypoxia until maternal blood pressure was restored with ephedrine. Thus, ephedrine, when used to correct maternal hypotension, prevents fetal metabolic acidosis. (Shnider, S. M., and others: *Vasopressors in Obstetrics. 1. Correction of Fetal Acidosis with Ephedrine during Spinal Hypotension*, *Amer. J. Obstet. Gynec.* 102: 911 (Dec.) 1968.)