

then proceeded uneventfully with this new machine.

A technician checked the original gas machine and after stripping down the breathing circuit reported that it was working satisfactorily and that he was unable to determine the cause of the trouble. There had been difficulty with the canister on-off switch, which had jammed, and it was thought that maybe this was in some way responsible for the difficulty experienced.

Two months later an attending anesthesiologist went to prepare the same gas machine in the same OR for an open-heart operation. He attached the sterilized corrugated tubing, Y-piece, and breathing bag to the machine. On inflating the breathing bag with oxygen, he was surprised to see that it did not deflate, but instead remained full. Aware that there had been trouble with this machine before, he checked the absorber, but still could not understand what was wrong. It wasn't until the C.R.N.A., who has charge of the equipment, arrived on the scene, that the correct diagnosis was made—*two sets of valves*, one on the canister, the other in the Y-piece,

but *in opposition*. By then the build-up in gas pressure had forced one of the valves in the Y-piece off its seating.

Enquiries disclosed that the supervisor of the cardiothoracic OR recently had ordered a number of new metal swivel Y's (Anesthesia Associates) and in error specified they be fitted with valves. All the other 24 gas machines are fitted with metal swivel Y's, but without valves because the absorbers contain valves.

We were lucky that in each case two anesthesiologists and a spare machine were available to solve the immediate difficulty. However, it must be clear that under less fortunate circumstances a tragedy could easily have occurred. It is also clear that this simple error is NOT easy to detect in the heat of the moment unless one has it in mind when trouble occurs. Surely it is worth some temporary inconvenience to eliminate this hazard for all time by a simple mechanical change in design.

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## Brachial Plexus Block

*To the Editor:*—It was with a great pleasure that I read the report of DeKrey, Schroeder, and Buechel on continuous brachial plexus block in the March issue of ANESTHESIOLOGY.<sup>1</sup> Although they cite Ansbro,<sup>2</sup> who described a technique of continuous supraclavicular brachial block using a metal needle in 1946, they were apparently unaware of our description in 1964<sup>3</sup> of the use of plastic needles to provide both continuous axillary and continuous subclavian perivascular block. Nonetheless, with regard to their technique of continuous brachial plexus block, it is imperative to point out that once a paresthesia has been obtained, an initial injection of anesthetic solution should be made, not only to ascertain that the needle is in the sheath, but also to expand the sheath and push the nerve trunk away from the needle. Following this, the needle should not be advanced (or at *most* only for a distance

of 1 mm) but should be held firmly in place while the catheter is advanced over it; the catheters currently available are rigid enough to advance along the sheath. This minor detail is important if one is to avoid either lacerating a nerve with the needle or making an intraneural injection.

Since development of the interscalene technique,<sup>4</sup> we have preferred continuous brachial plexus block by this approach, for should the plastic catheter advance when the patient turns his head or moves his neck, there are no vital structures at *this* level that can be damaged. Our primary indication for continuous brachial block has not been simply for prolonged operation, but more often for severe trauma to the extremity, particularly with vascular damage, where continuous sympathetic block, as well as sensory and motor block, is desirable for prolonged periods.

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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### References

1. DeKrey, J. A., Schroeder, C. F., and Buechel, D. R.: Continuous brachial plexus block, *ANESTHESIOLOGY* 30: 332, 1969.
2. Ansbro, F. P.: Method of continuous brachial plexus block, *Amer. J. Surg.* 71: 716, 1946.
3. Winnie, A. P., and Collins, V. J.: The subclavian perivascular technic of brachial plexus anesthesia, *ANESTHESIOLOGY* 25: 353, 1964.
4. Winnie, A. P., and Collins, V. J.: The Perivascular Technic of Brachial Plexus Anesthesia. Scientific Exhibit, Annual Meeting of the American Society of Anesthesiologists, Denver, Colorado, October 27, 1965.

### 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

Pa <sub>CO<sub>2</sub></sub>		pH	
Control	33	Control	7.45
10 min	39	10 min	7.47

(? increase in Pa<sub>CO<sub>2</sub></sub> with increase in pH).

#### Patient 10

Tidal volume	Pa <sub>O<sub>2</sub></sub>	Pa <sub>CO<sub>2</sub></sub>	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 Pa<sub>O<sub>2</sub></sub> of 22 mm ? increase in pH of 0.01 with 11 mm decrease in Pa<sub>CO<sub>2</sub></sub> and normal Pa<sub>O<sub>2</sub></sub> ? increase of Pa<sub>CO<sub>2</sub></sub> with increased tidal volume and a drop of minute ventilation of 0.0 l/min, when P<sub>O<sub>2</sub></sub> has increased. Also, again an increase of pH from 7.38 to 7.45, with an increase in Pa<sub>CO<sub>2</sub></sub> of 47 mm to 52 mm?)

#### Patient 11

A decrease in Pa<sub>CO<sub>2</sub></sub> from 54 mm to 45 mm produced (over 20 minutes), an increase of 0.01 pH units in a patient with a control Pa<sub>CO<sub>2</sub></sub> 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 Pa<sub>CO<sub>2</sub></sub> and pH. I think that it is clear that the observed changes in pH were not as consistent as the changes in Pa<sub>CO<sub>2</sub></sub>, as noted. In table 1 all 15 patients showed increases in Pa<sub>CO<sub>2</sub></sub> ten minutes post-diazepam. That the pH changes were not as consistent indicates that we had not measured pH as accurately as Pa<sub>CO<sub>2</sub></sub>. 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, Pa<sub>O<sub>2</sub></sub> and Pa<sub>CO<sub>2</sub></sub>. 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 Pa<sub>O<sub>2</sub></sub> at 10 minutes could increase without a change in minute ventilation. Similarly, changes in Pa<sub>CO<sub>2</sub></sub> were not consistent with the observed changes in Pa<sub>O<sub>2</sub></sub>. I believe that the unusual changes in this patient could well have been related to the fact that