tube in respect to the potential danger of high pressure developed on the bronchial wall. After ensuring the correct tube position, the measurements were taken using an aneroid manometer, as described by Cox.$^5$ The observations were taken at the same time, at the beginning of the anesthesia, to avoid the influence of the $\text{N}_2\text{O}$ on the pressure of the cuff.

There was a statistical difference ($P < 0.001$) between the two groups, as the pressures for the PVC tubes were $56.25 \pm 21$ mmHg against $129.75 \pm 41.25$ mmHg recorded for the Carlens tubes. These data showed that the PVC tubes presented smaller pressures in the bronchial cuff than those recorded for the Carlens tubes. These findings suggest that the risk of damage on the bronchus can be decreased by the use of PVC tubes.

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Carcinogenic Potential of Nitrous Oxide

To the Editor.—Baden et al.$^1$ found no evidence that nitrous oxide lifetime exposure has any carcinogenic potential in mice. While this is reassuring to anesthesiologists, our oncology patients may not fare so well. Shapiro et al.$^2$ have shown that anesthetic drugs accelerate the progression of postoperative metastases of mouse tumors after a short, surgical exposure. Halothane, ketamine, thiopental, and nitrous oxide were implicated, although the mechanisms of each may differ.$^3$ The applicability of these findings to humans remains to be clarified, but, as Baden et al.$^1$ state, “Numerous studies have indicated that results of lifetime studies in small rodents predict the carcinogenic potential of a drug in humans.” How much more so a short, surgical exposure?

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In Reply.—We wish to thank Dr. Frankel for his comments. He correctly points out that, although there is no evidence that commonly used inhaled anesthetics are themselves chemical carcinogens, it is possible that they may accelerate the progression of preexisting tumors. The animal studies he cites to support this contention are, in fact, the most recent of a number of similar studies stretching back over 70 years.$^{1–4}$ The possible mechanisms for such acceleration could include changes in neurotranscine function, blood clotting, host immunological re-