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. 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 Carlen tubes. These data showed that the PVC tubes presented smaller pressures in the bronchial cuff than those recorded for the Carlen 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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REFERENCES


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Carcinogenic Potential of Nitrous Oxide

To the Editor.—Baden et al. 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. 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. The applicability of these findings to humans remains to be clarified, but, as Baden et al. 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. The possible mechanisms for such acceleration could include changes in neuroendocrine function, blood clotting, host immunological re-

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Detection of Contaminated Nitrous Oxide

To the Editor:—Recently, during the early stages of induction of anesthesia, with only nitrous oxide and oxygen flowing, it was noted that a previously calibrated and properly zeroed infrared anesthesia agent monitor (AAM-222, Puritan Bennett Corporation) was displaying 0.5% halothane prior to the agent being turned on. The machine was zeroed to air, but, when the sampling tube was replaced in the line, it again read 0.5%.

To trouble-shoot the problem prior to calling for repairs, the AAM was re-calibrated and re-zeroed. However, when the nitrous oxide wall source was turned on, the AAM immediately read 0.5%.

An outside firm was contacted to analyze our nitrous oxide for contamination. An infrared scan revealed a peak at 3.4 microns in the nitrous oxide coming from the main supply, which was not present in the E cylinder nitrous oxide. Further analysis by gas chromatography revealed the following contaminants: methane—detected, but not <0.02 PPM; ethane—0.2 PPM; propane—3.4 PPM; isobutane—0.02 PPM; N-butane—41.7 PPM; pentane—46.0 PPM; and hexane—0.8 PPM.

The concentrations as shown do not constitute a hazard. Other than removing the questionable lot from use, no further steps were taken by this hospital, inasmuch as it was not warranted.

These impurities do, however, show that there was defective quality control in manufacturing and/or processing of nitrous oxide, which was subsequently found and corrected. Thus, although not designed as such, the AAM-222 can prove invaluable in the detection of hydrocarbon contaminants of anesthetic gases.

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