Nitrous Oxide and Hypoxic Ventilatory Responses

To the Editor:—The article by Yacoub and colleagues (Anesthesiology 45: 385–389, 1976) is both interesting and important because nitrous oxide has long been regarded as a harmless gaseous anesthetic, causing minimal physiologic derangement. When describing the ventilatory response to hypoxia, Yacoub plotted ventilation (Ve) vs. alveolar PaO2 (Pao2). A hyperbolic function was obtained and an “A” value derived as described by Weil et al.1 It is now recognized that the ventilatory response to hypoxia varies linearly with arterial oxygen saturation (SaO2).2–4 Thus, by plotting Ve vs. SaO2, a linear function may be obtained. The straight lines thus generated are suitable for analysis by simple, conventional, statistical methods. Ear oximetry, with its advantages of non-invasiveness and immediate information, may be used to measure SaO2 during experiments.5

We have taken the data presented in figure 1 of Yacoub’s article and converted PaO2 to SaO2. We then replotted Yacoub’s data in the format suggested by Re buck and Campbell6 and Re buck and Woodley7 and obtained a linear function of the ventilatory response to hypoxia. Sev eringhaus has recently used a similar method to reexamine his previous data and has confirmed Re buck’s observations.8 Using this method, the ventilatory response to hypoxia (without added resistance) in figure 1 of Yacoub’s article becomes 0.41 l/min/1 per cent decrease in SaO2. The correlation coefficient of the line is 0.9. With nitrous oxide, the slope decreases to 0.11 l/min/1 per cent decrease in SaO2. The decrease in the ventilatory response produced by nitrous oxide inhalation is highly significant (P < .001).

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Removal of an Inflated Endotracheal Tube Cuff

To the Editor:—As developers of the Bivona Cuff, we were perturbed by the report by Doctors Tavakoli and Corssen1 of an isolated case in which a “hazardous complication” occurred because of separation of the pilot tube from the body of the endotracheal tube, thereby making routine deflation of the cuff impossible.

Directions for the use of a Bivona Fome-Cuf tube, accompanying each package, clearly state that the unit may be removed without cuff deflation. In no way is extubation hampered by an accidental loss of the pilot tube. The foam-filled cuff can be contracted either by applying suction to the pilot tube or by squeezing the cuff. The former, of course, is the common clinically accepted practice. But, in the event the pilot port is accidentally sheared off, the cuff will still collapse as it is gently pulled out (squeezed) through the vocal cords. This can be easily demonstrated by pulling a lightly lubricated cuff through one’s partially opened fingers.

Several of our colleagues have commented that they prefer not to deflate the cuff prior to extubation because they believe that accumulated secre-
Local Anesthetic vs. Spinal Fluid

To the Editor:—In a recent letter (Anesthesiology 44:451, 1976), Dr. Reisner indicates the value of using a urine test strip to distinguish cerebrospinal fluid from local anesthetic solution. I am writing to call attention to another simple method for distinguishing the two solutions. If the fluid dripping from the hub of the needle is allowed to come in contact with a solution of thiopental (pH 10), turbidity will occur immediately when the fluid is local anesthetic (pH 5) but not when it is cerebrospinal fluid (pH 7).

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Cuffs Do Not Seal the Trachea Airtight

To the Editor:—In 31 consecutive patients receiving closed-circuit anesthesia I measured the concentration of nitrous oxide in the pharynx with a Foregger Nitrous Oxide Monitor. In every case I found between 100 and 800 ppm of nitrous oxide in the pharynx in spite of zero concentrations at all other areas such as outside the mouth, around the circle system, and at the ventilator. In all cases, the cuffs were inflated to the point of no audible leak with positive pressure applied to the endotracheal tube.

Mehta found that cuffs do not protect against aspiration of contrast medium, but attributed this to its presence in the ventricle between false and true cords at the time when the cuff is deflated. Egnatinsky believes that dilatation of the trachea normally seen with inspiration allows aspiration around a cuff, and therefore recommends deliberate overinflation of high-volume cuffs. Pavlin et al. reported failure of a large-volume cuff to protect against aspiration in a spontaneously breathing patient. Stanley et al. recommend a pressure-relief valve to prevent overinflation of cuffs due to disproportionately rapid inward diffusion of nitrous oxide into cuffs, compared with slow outward diffusion of nitrogen. They rightly caution against overinflation in view of possible compression of the wall of the endotracheal tube or its orifices. The membranous posterior wall of the trachea offers the least resistance to overinflation, and when 20 ml of air were injected into an ordinary cuff on a #9 Magill tube a 7 cm-long rupture of the trachea resulted.

It seems timely to emphasize that cuffs do not provide an airtight seal except perhaps prior to utmost stretching of the trachea just preceding its rupture. My measurements show that the usual clinical maneuver of inflating cuffs just to the point of abolishing audible leakage with positive pressure to the airway does not result in an airtight seal. These results are easily verifiable with closed systems, which are without exception associated with leakage in the order of 20 to 100 ml/min. The practicing anesthetist should not place reliance on cuffs to seal the larynx, but should continue to employ additional safeguards against aspiration such as throat packs and avoidance of spontaneous respiratory efforts whenever feasible.

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