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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 (V_E) *vs.* alveolar P_{O_2} ($P_{A_{O_2}}$). 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 (Sa_{O_2}).[2–4] Thus, by plotting $\dot{V}_E$ *vs.* Sa_{O_2}, 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 Sa_{O_2} during experiments.[5]

We have taken the data presented in figure 1 of Yacoub's article and converted $P_{A_{O_2}}$ to Sa_{O_2}. We then replotted Yacoub's data in the format suggested by Rebuck and Campbell[3] and Rebuck and Woodley[4] and obtained a linear function of the ventilatory response to hypoxia. Severinghaus has recently used a similar method to reexamine his previous data and has confirmed Rebuck's observations.[6] Using this method, the ventilatory response to hypoxia (without added resistance) in figure 1 of Yacoub's article becomes 0.41 l/min/l per cent decrease in Sa_{O_2}. The correlation coefficient of the line is 0.9. With nitrous oxide, the slope decreases to 0.11 l/min/l per cent decrease in Sa_{O_2}. The decrease in the ventilatory response produced by nitrous oxide inhalation is highly significant ($P < .001$).

When the ventilatory response to hypoxia is expressed in terms of Sa_{O_2} rather than alveolar oxygen pressure, there are further advantages other than linearization of the response curves. It no longer becomes necessary to assume a level of arterial oxygenation from alveolar P_{O_2} measured at the mouth. When Sa_{O_2} is displayed continuously by the ear oximeter throughout hypoxic exposures, hypoxic studies in man will be safer and more readily accepted.

DAVID Z. N. FRANKEL, M.D.
Physiological Monitoring Resident
Department of Anesthesia
Toronto General Hospital
University of Toronto
Toronto, Ontario, Canada

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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 Corssen[1] 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-