CORRESPONDENCE

Anesthesiology
78:603–604, 1993

Ventilation *Via* a Mouth Mask Facilitates Fiberoptic Nasal Tracheal Intubation in Anesthetized Patients

To the Editor:—Fiberoptic tracheal intubation (FTI) is usually performed while a patient is awake. However, at times it must be utilized during general anesthesia, *e.g.*, when difficult intubation is recognized after induction or when a patient refuses or cannot tolerate awake tracheal intubation. Several types of face masks incorporating a diaphragm,[1–3] through which the insertion of a fiberscope and an endotracheal (ET) tube is undertaken, have been designed for this purpose. These masks make it possible to maintain ventilation and anesthesia during FTI. However, none are wholly satisfactory because the mask and diaphragm reduce the maneuverability of a fiberscope and an ET tube.[4]

We have developed a method by which ventilation is performed *via* only the mouth and FTI can be done *via* the nostril with no hindrance from the mask in anesthetized patients. Following induction of anesthesia, a mask applied only over the mouth (mouth mask) is substituted for a standard anesthesia mask. We have been using an infant or child type Seal Mask® (Gibeck Respiration) for the mouth mask because its elasticity and pliability facilitate the fit over the mouth. An oral airway is usually inserted to overcome the airway obstruction. The nostrils are plugged with cotton if air leakage from the nostrils disturbs ventilation. FTI is performed by another anesthesiologist. An ET tube, which is capped with a rubber diaphragm to protect against air leakage, is passed through the nostril, and a fiberscope is inserted through the ET tube (fig. 1). The subsequent technique is the same as that of the usual FTI for awake patients.

The key to success with this method is whether ventilation is maintained with the mouth mask. With our experience, in almost all cases, ventilation is as well maintained with the mouth mask as with a standard anesthesia mask. There has been one failure in 123 attempts. In this case, the patient suffered from advanced rheumatoid arthritis and was undergoing "halo" traction for atlantoaxial dislocation and C3, C4 vertebral compression fracture. Ventilation was well maintained with a standard anesthesia mask, but not with the mouth mask.

We have been using this method for difficult intubation for 2 yr and have found that this is the easiest and safest FTI approach for difficult intubation under general anesthesia.

Takumi Nagaro, M.D.
Associate Professor of Anesthesiology and Resuscitology

Gen Hamami, M.D.
Instructor of Anesthesiology and Resuscitology

Yasufumi Takasaki, M.D.
Instructor of Anesthesiology and Resuscitology

Tatsuru Arai, M.D.
Professor and Chairman of Anesthesiology and Resuscitology

Ehime University School of Medicine,
Shigenobu-cho, Onsen-gun, Ehime-ken
791-02, Japan

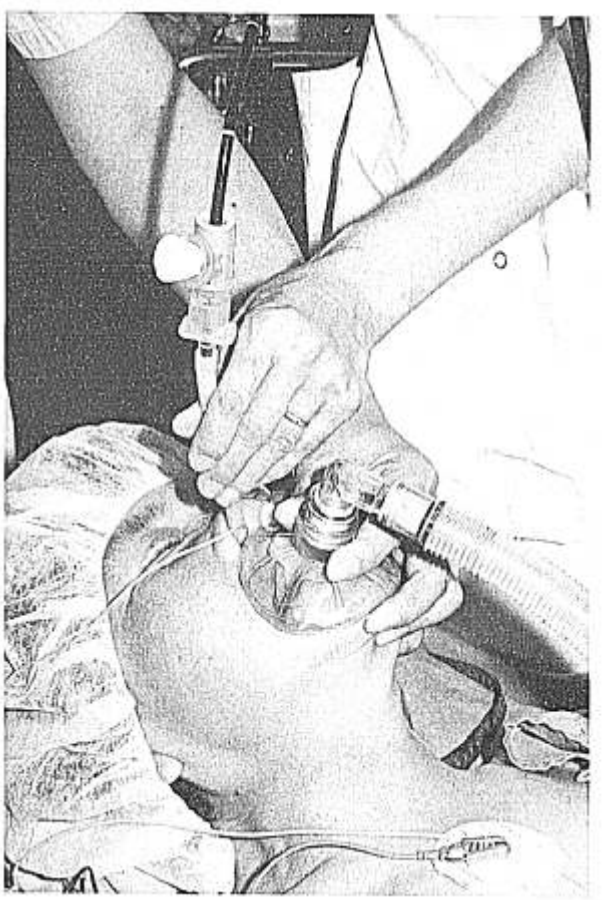

Fig. 1. Mouth mask ventilation method for nasal endotracheal (ET) intubation for anesthetized patients. Ventilation and anesthesia are maintained by a mask applied only over the mouth (an infant type Seal Mask®) with aid of an oral airway during fiberoptic tracheal intubation (FTI). FTI is performed by another anesthetist. An ET tube, which is capped with a rubber diaphragm, is passed through one nostril. The other nostril is plugged with cotton. A fiberscope is inserted through the ET tube. The subsequent procedure is the same as that of the usual FTI for awake patients.

References

1. Patil V, Stehling LC, Zauder HL, Koch JP: Mechanical aids for fiberoptic endoscopy. ANESTHESIOLOGY 57:69–70, 1982
2. Rogers SN, Benumof JL: New and easy techniques for fiberoptic

endoscopy-aided tracheal intubation. ANESTHESIOLOGY 59:569–572, 1983

3. Imai M, Kemmotsu O: A new adapter for fiberoptic endotracheal intubation for anesthetized patients. ANESTHESIOLOGY 70:374–375, 1989

4. Mcalpine G, Williams RT: Fiberoptic assisted tracheal intubation under general anesthesia with IPPV. ANESTHESIOLOGY 66:853, 1987

(Accepted for publication November 20, 1992.)

Anesthesiology
78:604, 1993

Pharmacodynamics of Propofol and Free Drug Concentrations

To the Editor:—The study by Vuyk *et al.*[1] on the concentration-effect relationship of propofol was interesting because it attempted to achieve blood-brain equilibrium of propofol concentrations. However, the authors did not comment on the wide range of concentrations required to produce the same effect in a presumably homogeneous group of patients. The highest whole blood propofol concentration for loss of eyelash reflex was 3.92 μg/ml, more than double the lowest concentration of 1.88 μg/ml.

The effects of highly protein bound drugs may correlate better with the free fraction of drug.[2] Propofol has a more shallow dose-response curve compared with thiopental,[3] but it has not been generally acknowledged that this may be a result of propofol being highly protein bound. The protein binding of propofol may vary from 97% to 99%,[4,5] there being a 300% increase in free fraction from 1% to 3%. The free drug exerts the effect, and so there may be marked variation in the total propofol concentrations for a given effect while the free concentrations are similar. If data on the free concentration-effect relationship of propofol proves to be less variable, it would then be useful to try and predict the degree of protein binding in a particular patient.

Propofol binds to albumin and erythrocytes. The induction dose of propofol was correlated significantly with albumin concentrations,[6] although the correlation coefficient was only 0.28. There are no studies correlating protein binding of propofol with physiologic data such as albumin and hemoglobin concentration. I do not disagree with the important results from Vuyk *et al.*, but suggest that it would be valuable to measure free propofol concentrations in future studies.

Tony Gin, M.B.Ch.B., M.D., B.Sc., F.R.C.Anaes., F.A.N.Z.C.A.
Director of Research
Department of Anaesthesia and Intensive Care
The Chinese University of Hong Kong
The Prince of Wales Hospital
Shatin, New Territories
Hong Kong

References

1. Vuyk J, Engbers FHM, Lemmens HJM, Burm AGL, Vletter AA, Gladines MPRR, Bovill JG: Pharmacodynamics of propofol in female patients. ANESTHESIOLOGY 77:3–9, 1992

2. Wood M: Plasma binding and limitation of drug access to site of action. ANESTHESIOLOGY 75:721–723, 1991

3. Servin F, Desmonts JM, Haberer JP, Cockshott ID, Plummer GF, Farinotti R: Pharmacokinetics and protein binding of propofol in patients with cirrhosis. ANESTHESIOLOGY 69:887–891, 1988

4. Gin T, Yau G, Jong W, Tan P, Leung RKW, Chan K: Disposition of propofol at caesarean section and in the postpartum period. Br J Anaesth 67:49–53, 1991

5. Leslie K, Crankshaw DP: Potency of propofol for loss of consciousness after a single dose. Br J Anaesth 64:734–736, 1990

6. McCleane GJ, Fogarty DF, Watters CH: Factors that influence the induction dose of propofol. Anaesthesia 46:59–61, 1991

(Accepted for publication November 20, 1992.)

Anesthesiology
78:604–605, 1993

In Reply:—We studied the concentration-effect relationships for loss of eyelash reflex and loss of consciousness of propofol in healthy female patients and indeed found a twofold interindividual variability[1] in our patients with respect to the response to similar blood propofol concentrations. However, this variability, in our opinion, is small compared to the four- to five-fold interindividual variability previously described for other intravenous anesthetic agents.[2,3]

The range of the concentrations required to produce a similar effect in this group of patients can be partially explained by the following factors. First, as stated in the paper, some patients in our study lost eyelash reflex and consciousness before blood-brain equilibration had occurred. In these patients, the blood propofol concentration still exceeded the theoretical effect compartment concentration, thereby contributing to the above mentioned interindividual variability. A second factor is the variation in the analysis of the blood propofol concentration by high-performance liquid chromatography (a coefficient of variation ≤ 7%). Third, we agree with Gin that, for highly protein-bound drugs, interindividual differences in protein binding might be a factor influencing the magnitude of the pharmacodynamic interindividual variability as well. For alfentanil, for example, Lemmens *et al.* showed that 45% of the variability in the CP50 of alfentanil for the intraabdominal part of surgery could be explained by the variability in the protein binding of alfentanil.[4]

In conclusion, we agree with Gin that differences in protein bind-