CORRESPONDENCE

Anesthesiology 1997; 87:449 © 1997 American Society of Anesthesiologists, Inc. Lippincott-Raven Publishers

The Use of a Nasogastric Tube as an Aid in Blind Nasotracheal Intubation: A Postscript

To the Editor-We would like to propose an improvement to our original method of intubation presented in 1992. We originally proposed first inserting an endotracheal tube into the esophagus, then pulling the tube back into the pharynx until breath sounds are audible. The cuff is then inflated, and^{2,3} the nasogastric tube is passed into the larynx through the endotracheal tube. At this point, the Rusch tube can be inserted into the trachea together with the nasogastric tube after deflating the cuff. Now we would like to recommend an alteration for situation in which the nasogastric tube is mistakenly inserted into the esophagus. When this occurs the endotracheal tube cuff should be deflated, the tube advanced into the esophagus, and rotated approximately 180°. It should then be withdrawn until breath sounds are heard, and the nasogastric tube is then reinserted. We emphasize the importance of rotating of Rusch endotracheal tube in the esophagus because most failures may occur when the tip of the endotracheal tube, when drawn back from the esophagus, is located on the improper side of the pharynx to permit passage of the nasogastric tube. Following this recommendation, we have found the success rate to be greatly improved

Naoki Sugiura, D.D.S.
Department of Oral and Maxillofacial surgery
Morimasa Yamada, M.D., D.D.S.
Motoshi Kainuma, M.D.
Toshiyuki Miyake, M.D.
Department of Anesthesiology
Fujita Health University School of Medicine
Toyoake, Aichi 470-11, Japan

References

- 1. Sugiura N, Miyake T: The use of a nasogastric tube as an aid in blind nasotracheal intubation. Anesthesiology 1992; 77:613-4
- 2. Gorbach MS: Inflation of endotracheal tube cuff as an aid to blind nasal endotracheal intubation. Anesthesia Analg 1987; 66:913-22
- 3. Van Elstraete AC, Pennant JH, Gajraj NM, Victory RA: Tracheal tube cuff inflation as an aid to blind nasotracheal intubation. Br J Anaesth 1993; 70.691-3

(Accepted for publication March 31, 1997.)

Anesthesiology 1997; 87:449-450 © 1997 American Society of Anesthesiologists, Inc. Lippincott-Raven Publishers

Suction Catheter-guided Nasotracheal Intubation

To the Editor—Despite the popularity of fiberoptic bronchoscope for nasotracheal intubation, "blind nasal" intubation still remains a necessary technique in maintaining an airway in certain conditions. Although various methods and devices have been developed, most of the concerns are focused on the safety of operators without considering the trauma to the patients. 5-7

We would like to present a simple, easy, inexpensive technique that would be more favorable for the patient and the operator. The whole process takes only a few minutes and is faster than placing the fiberoptic bronchoscope. It is an ideal technique for the patients on whom the use of fiberoptic bronchoscope is impossible because of copious bloody secretions.

The patient is prepared for an awake intubation as described elsewhere. ^{1,2} An appropriate size of endotracheal tube is inserted through the nostril until the tip of the endotracheal tube reaches the oropharynx where the nonturbulent smooth breathe sounds can be heard. At that point, instead of directly passing the endotracheal tube into the larynx blindly as the conventional method describes, a soft suction catheter is inserted (Professional Medical Products, Inc, Suction Catheter with control port, 14-French TM) through the tube into the trachea. The predetermined length of the catheter is advanced to reach the end of the endotracheal tube. The patient is asked to take deep respirations, and the catheter tip is advanced slowly while

applying gentle suction. The catheter tip will go into the trachea easily. After the catheter tip is advanced >10 cm from the tip of the endotracheal tube, the catheter is connected to the gas sampling tubing of the capnograph, and the control port is sealed. The capnograph confirms the placement of the catheter tip in the trachea with reassuring CO₂ wave forms. If the catheter tip is placed in the esophagus, very small (if any) CO₂ wave forms will be noticed on the capnograph. The operator will also feel more resistance as the catheter tip passes through the esophagus. If the patient has not been adequately topicalized, the patient will cough as the tip of the catheter approaches the glottis. The operator also may feel some degree of resistance while advancing the catheter through the glottis. Once the catheter tip is placed in the trachea, the endotracheal tube can be passed over the catheter to the trachea using the Seldinger technique.

Reasons for successful placement of the catheter in the trachea appear to be as follows:

First, the nasopharyngeal route is a natural airway; air passes directly from *via* the oropharynx to the trachea. This explains why a nasogastric tube, intended to be placed in the esophagus, often goes to the trachea and why a fiberoptic bronchoscope inserted through the nasopharyngeal route often enters the trachea without much guidance. Second, we have noticed that while working to clear copious bloody secretions under direct laryngoscopy for difficult intuba-

CORRESPONDENCE

tion, the soft suction catheter tip tends to move toward the trail of secretions that is coating the glottis and eventually finds its way to the trachea. Finally, strong inspiratory air movement on awake patients may further facilitate the catheter tip to move into the trachea.

Don S. Lee, M.D., M.P.H. Associate Professor Charles I. Yang, M.D. Resident Department of Anesthesiology George Washington University Medical Center 901 23rd Street, NW Washington, DC 20037-0170 Second edition. Edited by Barash PG, Cullen BF, Stoelting RK. Philadelphia, JB Lippincott Company, 1992, p 698

- 2. Benumof JL: Management of the difficult airway with special emphasis on awake tracheal intubation. Anesthesiology 1991; 75:1087-110
- 3. Gilbert TB: An earpiece modification to facilitate nasotracheal intubation [letter]. Anesth Analg 1990; 70:334-5
- 4. Shapiro H, Unger R: Blind, but not deaf or dirty, intubation. Anesthesiology 1986; 64:297
- 5. King H-K, Wooten DJ: Blind nasal intubation by monitoring endtidal carbon dioxide. Anesth Analg 1989; 69:412-3
- 6. Omoigui S, Glass P, Martel DLJ, et al: Blind nasal intubation with audio-capnometry. Anesth Analg 1991; 72:393
- 7. Berger JM, Stirt JA: Aid to nasotracheal intubation. Anssthesiology 1983; 58:105-6

(Accepted for publication April 1, 1997.)

References

1. Stehling LC: Management of the Airway, Clinical Anesthesia.

Anesthesiology 1997; 87:450–1 © 1997 American Society of Anesthesiologists, Inc. Lippincott–Raven Publishers

Formation of a National Database on Pulmonary Hypertension and Hepatopulmonary Syndrome in Chronic Liver Disease

To the Editor: - Pulmonary arterial hypertension (PAH) occurs in approximately 2% of patients who present for liver transplantation.¹ Intraoperative management is difficult and associated with mortality rates of up to 80%.2 Different from pulmonary hypertension but equally challenging is the hepatopulmonary syndrome (HPS), a triad of hypoxemia, pulmonary vasodilation, and hepatic dysfunction that occurs in approximately 30% of potential transplantation recipients.³ Marked hypoxemia that occurs during transplantation surgery often complicates the postoperative course and leads to an increase in perioperative death.4 Resolution of pulmonary hypertension and hepatopulmonary syndrome has occurred in some patients after liver transplantation.^{5,6} However, because cases are limited in any given institution, factors that predict either intraoperative or perioperative survival have not been identified.⁷ Therefore, many institutions deny these high-risk patients liver transplantation. Apart from liver transplantation, there is no other known management.

To learn more about the natural history and outcome of these special patients, a group of interested investigators has established a database to collect information from multiple liver transplantation centers. The principal investigators are Michael J. Krowka, M.D., at the Mayo Clinic, Rochester, Minnesota, and M. Susan Mandell, M.D., Ph.D., at the University of Colorado, Denver.

Members of the steering committee for The Multicenter Hepatopulmonary/Pulmonary Hypertension Database for Liver Transplant Candidates/Recipients include Gary Abrams, M.D., (Hepatology) University of Alabama, Jeffery Crippin, M.D., (Hepatology) Baylor University, Dallas, Marie Csete, M.D., (Anesthesiology) University of California at Irvine, Andre DeWolf, M.D., (Anesthesiology), Northwestern University, Chicago, John Lake, M.D., (Hepatology) University of California at San Francisco, David Plevak, M.D., (Anesthesiology) Mayo Clinic, Rochester, Minnesota, Jeffery Plotkin, M.D., (Anesthesiology)

University of Maryland, Victor Scott, M.D., (Anesthesiology) University of Pittsburgh, James K. Stoller, M.D., (Pulmonary) Cleveland Clinic, Terry Therneau, Ph.D., (Biostatistics) Mayo Clinic, Minnesota, and Russell Weisner, M.D., (Hepatology), Mayo Clinic, Minnesota.

The database will collect information regarding the severity of liver and pulmonary disease, transplantation status, and outcome. The database will address following issues:

- 1. Identify factors that predict the survival of patients with PAH or HPS during and after liver transplantation surgery.
- 2. Identify characteristics that predict pulmonary disease resolution.
- Determine the natural history of patients with PAH or HPS who do not undergo liver transplantation.
- Identify cost-effective evaluation of patients with PAH and HPS before liver transplantation.
- Initiate a multicenter therapeutic trial with medications suggested to be effective in pulmonary hypertension of other etiologies.

All patient data are confidential and will be presented periodically at national meetings. We are currently recruiting patients with liver failure that meet the following criteria for identification of pulmonary hypertension and hepatopulmonary syndrome:

- 1. Pulmonary hypertension
 - · Mean pulmonary artery pressures greater than 25 mmHg
 - Pulmonary vascular resistance greater than 120 dynes·s·cm⁻⁵
 - Pulmonary, capillary wedge pressure less than 15 mmHg
- 2. Hepatopulmonary syndrome
 - PaO_2 less than 70 mmHg, or hemoglobin saturation less than 92% breathing room air

AND

· Positive enhanced echocardiogram (left atrial opacification