References

1. Bell E: Nitroglycerin and uterine relaxation. ANESTHESIOLOGY 1996; 85:683

2. Langevin PB, Wood CE, James CF: Nitroglycerin and uterine muscle relaxation in pregnant sheep (Abstract). ANESTHESIOLOGY 1994; 81:A1137

3. Shin Y, Wang K, Kim Y, et al: Effect of nitroglycerin on a pregnant uterine muscle in vitro: A dose-response study. Anesth Analg 1994; 78:S391

4. Kumar D, Zouorlas PA, Barne AC: In vivo effect of amyl nitrite on human pregnant uterine contractility. Am J Obstet Gynecol 1965; 91:1061-8

5. Langevin PB, Katovich MJ, James CF: Uterine smooth muscle compliance effect of mitroglycerin independent of a contractile mediator (Abstract). ANESTHESIOLOGY 1996; 85:A885

(Accepted for publication May 21, 1997.)

Anesthesiology
1997; 87:717

In Reply:—Dr. Langevin correctly points out that the animal data do not consistently support the clinically observed phenomenon of uterine relaxation resulting from administration of nitroglycerin in human parturients. Dr. Shin *et al.*[1] found a dose-dependent reduction of contractility in isolated human uterine muscle segments exposed to nitroglycerin in vitro, but the mean dose to abolish spontaneous contractions was 4.5×10^{-4} M (250 μg in the 12 ml bath), far exceeding commonly used clinical doses. This may be a result of the scarcity of vascular endothelium present in these preparations.

Dr. Langevin also states that administration of nitroglycerin definitely reduces maternal blood pressure. In our experience, the administration of 50-100 μg of nitroglycerin intravenously or 800 μg sublingually has not resulted in decreased maternal blood pressure consistently, or even frequently, in urgent clinical settings. These include tetanic uterine contraction, extraction of a breech twin, or manual extraction of placenta. I suspect that the anxiety engendered by rapid interventions such as maternal position changes, increased intravenous fluid administration, application of supplemental oxygen, and summoning the obstetrician ameliorate the vasodilatory effects of the nitroglycerin. This is in contrast to our recent trial using nitroglycerin spray in the setting of elective external version of breech position.[2] We saw a high percentage of patients respond with decreased blood pressure (4 of 10 patients experienced a decrease of 20% or greater). It is unclear whether this was in response to the nitroglycerin or mechanical compression from vigorous attempts, and certainly deserves further investigation.

Elizabeth Bell, M.D., M.P.H.
Assistant Professor of Anesthesiology
Division of Women's Anesthesia
Duke University Medical Center
Box 3094
Durham, North Carolina 27710

References

1. Shin Y, Wang K, Kim Y, et al.: Effect of nitroglycerin on pregnant uterine muscle in vitro: A dose-response study. Anesth Analg 1994; 78:S391

2. Redick LF, Bell EA, Livingston E: Sublingual aerosal nitroglycerin for uterine relaxation in attempted external version. Am J Obstet Gynecol 1997; In print.

(Accepted for publication May 21, 1997.)

Anesthesiology
1997; 87:717-8

Conversion of a Nasal to an Orotracheal Intubation Using an Endotracheal Tube Exchanger

To the Editor:—A 57-yr-old woman presented to the emergency room with confusion, weakness and cyanosis. Her history included a muscle biopsy and contracture test consistent with malignant hyperthermia, confirmed hypertrophic obstructive cardiomyopathy, and an undiagnosed neurologic condition characterized by progressive, episodic confusion, somnolence, dysarthria, and headache. She had been started on amiodarone and sotalol by a cardiologist for paroxysmal atrial fibrillation.

Progressive somnolence and hypercapnic acidosis led to a decision to intubate. Airway evaluation revealed mild micrognathism but no other abnormalities. Monitoring consisted of continuous electrocardiography, oximetry, and invasive arterial blood pressure. Lidocaine spray was applied to the oropharynx and hypopharynx, but direct laryngoscopy was poorly tolerated. After preoxygenation, sleep was induced with propofol. Bag and mask ventilation was easily provided, and vecuronium was administered. With appropriate positioning, di-

rect laryngoscopy using Macintosh and Miller blades permitted visualization of the epiglottis but not the vocal cords. Despite external laryngeal pressure and a stylet, the trachea could not be intubated. Oral intubation using a fiberoptic bronchoscope was unsuccessful due to an inability to pass posterior to the epiglottis. Finally, after neosynephrine drops were used to provide nasal vasoconstriction, a 7.5-mm outer diameter nasal tracheal tube was successfully introduced fiberoptically using a vigorous jaw thrust.

A decision was made to convert the nasal tube to an oral tube. A fiberoptic bronchoscope, LMA, and cricothyroidotomy kit were immediately available. After ventilation at a FiO_2 1.0, unconsciousness and paralysis were maintained with supplemental propofol and vecuronium bromide. An endotracheal tube exchanger, (endotracheal ventilation catheter, ETVC™, CardioMed Supplies, Gormley, ON) was inserted through the nasal tube. The nasotracheal tube was then withdrawn, leaving the ETVC™ *in situ*. Direct pharyngoscopy was performed, and the ETVC™ was grasped and secured with Magill forceps. A second Magill forceps was used to pull the ETVC™ in through the nose and out the mouth. This was readily accomplished without significant change in arterial saturation. A 8.0 mm OD ETT was then loaded onto the ETVC™. The ETVC™ was connected to oxygen tubing and insufflation at 4 LPM was commenced. The endotracheal tube was easily threaded over the ETVC™ to a depth of 22 cm. Before withdrawing the ETVC™, it was threaded through a bronchoscopic adaptor and carbon dioxide was detected during positive pressure ventilation. The ETVC™ was withdrawn, and the position of the endotracheal tube was confirmed bronchoscopically. Arterial saturation remained in excess of 98% throughout the tube exchange.

Seven days later, she was extubated uneventfully, using the ETVC™ to maintain airway access.[1]

Novella has described the intraoperative conversion of a nasotracheal to orotracheal tube in a patient with Klippel-Feil syndrome, using a Sheridan tube exchanger.[2] Although his conversion was achieved within 90 s, the patient desaturated from 100% to 85%. The provision of oxygen by insufflation, or jet ventilation if necessary[3] provides an additional measure of safety should the conversion be prolonged or the patient unstable. It is equally important to be prepared for possible reintubation. In the present case, a tube exchanger was used to maintain airway access after extubation.

It is important to point out that commercial tube exchangers vary in their stiffness and diameters.[4] Such physical properties may prevent this maneuver from being successful with a stiffer or larger caliber catheter.

Richard M. Cooper B.Sc., M.Sc., M.D., F.R.C.P.C.
Assistant Professor
University of Toronto
Department of Anaesthesia
The Toronto Hospital and Mount Sinai Hospital
600 University Avenue
Toronto, Ontario M5G 1X5

References

1. Cooper RM: Clinical use of an endotracheal ventilation catheter for airway management: 202 consecutive cases. Can J Anaesth 1996: 43:90–3

2. Novella J: Intraoperative nasotracheal to orotracheal tube change in a patient with Klippel-Feil syndrome. Anaesth Intensive Care 1995: 23:402–4

3. Cooper RM, Cohen DR: The use of an endotracheal ventilation catheter for jet ventilation during a difficult intubation. Can J Anaesth 1994: 41:1196–9

4. Cooper RM: Extubation and Changing Endotracheal Tubes, Airway Management: Principles and Practice. Edited by Benumof JL. St. Louis, Mosby-Year Book, 1996

(Accepted for publication June 16, 1997.)

Anesthesiology
1997; 87:718–9

Delayed Subarachnoid Migration of an Epidural Arrow FlexTip Plus Catheter

To the Editor:—The "migration" of epidural catheters into undesirable locations is well-documented in the literature. The three most commonly reported sites of catheter misplacement are intravascular, subdural, and subarachnoid. However, most of these reports provide details consistent with introduction of the Tuohy needle, either partially or completely, into these spaces prior to the placement of the catheter. Others have reported what appeared to be a block produced by a normally functioning epidural catheter which, after a few subsequent bolus doses, "suddenly" produced a far more extensive block, *i.e.*, from lumbar to cervical. To our knowledge, only one instance of an epidural catheter perforating into the subdural space after functioning normally for a prolonged period of time (4 days) has been reported.[1] We report an apparent delayed perforation into the subarachnoid space involving the new soft FlexTip PlusO epidural catheter (Arrow International).

The patient was a 66-yr-old woman with malignant mesothelioma presenting for thoracotomy and pleural decortication. Multiple attempts were made to place an epidural catheter in the T7–T8 interspace using the "loss of resistance" technique and normal saline. At no time did we note paresthesias, blood, or cerebrospinal fluid (CSF). A catheter was threaded but subsequent doses of local anesthetic failed to confirm placement in the epidural space. The surgery proceeded under general anesthesia without incident. Several hours after surgery, a repeat attempt at catheter placement at T11–T12