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.

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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.
3. Determine the natural history of patients with PAH or HPS who do not undergo liver transplantation.
4. Identify cost-effective evaluation of patients with PAH and HPS before liver transplantation.
5. 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 $\cdot$ s $\cdot$ 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

greater than 3 beats after right ventricle opacification) or positive ^{99m}Technetium lung scan documenting greater than 5% shunt uptake over the brain or kidneys

Survey forms can be obtained from: Michael J. Krowka, M.D., 200 1st Street, SW, Rochester, Minnesota, 55905; telephone: 507 284-2921; E-mail: Krowka.Michael@Mayo.Edu

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A Choking Hazard during Nasal End-tidal CO_2 Monitoring

To the Editor:—Goldman[1] initially described the monitoring of end-tidal CO_2 ($ETCO_2$) in awake and sedated patients *via* nasal cannula by inserting a shortened intravenous catheter into the lumen of one of the nasal prongs and connecting this to the sampling tube from the capnograph. I used this technique to monitor expired CO_2 in a patient undergoing dilation and curettage during intravenous sedation. Intraoperatively, the patient became apneic, so the nasal cannula was replaced immediately with face mask delivering 100% oxygen. While attempting to provide assisted ventilation, I noticed a small object at the corner of the patient's oral cavity that turned out to be the shortened intravenous catheter, which had apparently been dislodged from the nasal cannula. It was removed quickly without incident. In the process of switching from nasal cannula to face mask oxygen, the $ETCO_2$ sampling tube had to be disconnected first from the adapted intravenous catheter in the nasal cannula and then connected to the face mask. The nasal cannula was then removed and replaced by the face mask. In the process, the adapted intravenous catheter was unknowingly dislodged from the nasal cannula, falling into the oral cavity.

I urge anesthesiologists using this adapted method for $ETCO_2$ monitoring to be careful about the potential dangers that could result from dislodgement of the catheter hub.

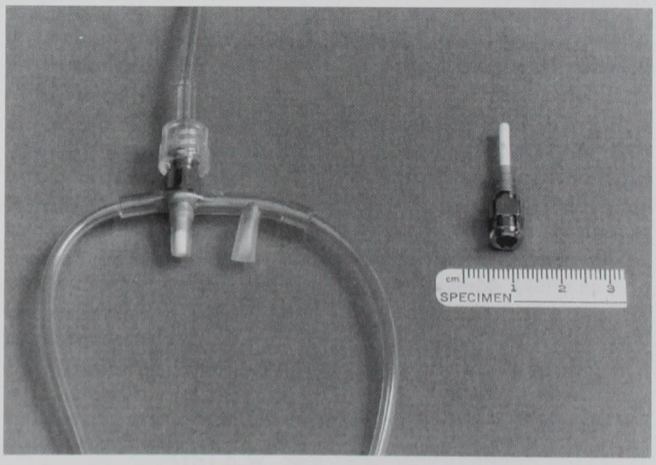

Fig. 1. Nasal cannula adapted with a shortened intravenous catheter for nasal $ETCO_2$ monitoring. The size of the adapted intravenous catheter is shown next to the nasal cannula.

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