To the Editor:—In a recent article by Höhn et al.,1 it was concluded that acute normovolemic hemodilution (ANH) in addition to aprotinin was not beneficial in preventing allogeneic blood transfusions compared to aprotinin alone in cardiac surgery. In this randomized, controlled trial, the patients were hemodiluted to a hematocrit of 28% pre-cardiopulmonary bypass (CPB). The transfusion threshold was set at 17% during CPB and at 25% for post-CPB. The total fluid replacement was in excess relative to the amount of ANH (autologous blood) removed (6.4 ± 2.1 l of crystalloid, 2.0 ± 0.7 l of colloid).2 This led to excessive hemodilution, reducing the hematocrit below the transfusion threshold in the ANH group. Indeed, 50% of the patients in the ANH group required either all (33%) or a portion (22%) of the autologous blood to be transfused during CPB, thus negating its positive effects on erythrocytes and coagulation protection. Consequently, allogeneic erythrocyte transfusion rates and the indirect clinical markers for surgical bleeding (cell saver and 24-h chest tube drainage) were not different between the two groups. One of the goals of ANH is to return it after heparin neutralization. Additional hemodilution occurs with the onset of CPB; therefore, hemofiltration or ultrafiltration and/or diuresis should have been employed to remove excess fluid. Alternatively, ANH can be performed just prior to the onset of CPB; therefore, hemofiltration or ultrafiltration and/or induced diuresis is frequently utilized on CPB to remove excess fluids and to reduce the dilutional effect from the CPB prime. The starting hematocrit averages 39%. We use ε-aminocaproic acid for low-risk cases and reserve aprotinin (TrasyloI®, Bayer, West Haven, CT) for high-risk cases. The total amount of cell saver returned is approximately 200 ml, and 24-h chest tube drainage is 428 ml. Allogeneic transfusion rates for packed erythrocytes, fresh frozen plasma, and cryoprecipitate are 11%, 3%, and less than 1%, respectively.

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Finally, as suggested by Dr. Moskowitz and others,6 we agree that the implementation of a standardized, multidisciplinary approach, including definition of transfusion criteria, administration of antifibrinolytic drugs, use of cell saver devices, and external heating, is most helpful to reduce transfusion of allogeneic blood products in patients undergoing cardiac surgery. However, evidence supporting the addition of ANH as a blood-sparing technique is still lacking, although a consensus is based on data from observational studies and prospective, unblinded, randomized trials.7

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In Reply.—In Dr. Wark’s letter, he states, “hepatic failure after cardiopulmonary bypass is unlikely to be isoflurane hepatitis.” In response, I will borrow an Asian proverb: “A journey of a thousand miles must begin with a single step.” Dr. Wark introduces alternative views of a classic argument. We both agree that hepatotoxicity can occur following halothane administration. However, we disagree about hepatotoxicity when other halogenated volatile anesthetics are considered. Nonetheless, hepatotoxicity following halogenated volatile anesthetics, including isoflurane, is believed to be idiosyncratic, and immune-mediated processes are believed to have a role in this injury, similar to other forms of idiosyncratic drug-induced hepatotoxicity. There are several reports of immune responses and hepatotoxicity following isoflurane administration. More importantly, in this case report, isoflurane was implicated after critically examining other possible causes of hepatotoxicity and was not based on histologic findings alone.

This case report takes forward steps in the journey of a thousand miles to the discovery of the etiologies of drug-induced hepatotoxicity. The first step was also taken by those before us. We demonstrated liver injury following isoflurane administration. Norepinephrine and nitroglycerin were administered, but hypotension or other evidence of ischemia was not demonstrated. The article from 1996 referenced by Dr. Wark clearly states the risk factors for gastrointestinal complications after cardiopulmonary bypass: advanced age, combined coronary artery bypass grafting–valve operation, postoperative low cardiac output, prolonged ventilation time, reexploration of the chest, sternal infection, and a history of peptic ulcer. In our report, the patient was 66 yr old, underwent a three-vessel coronary artery bypass graft, and maintained mean arterial pressures ≥ 70 mm Hg for > 48 h intraoperatively and postoperatively during the initial demonstration of increased aspartate aminotransferase. Therefore, if Dr. Wark’s probability was correct, the only risk factor was age.

By showing that the patient had several risk factors for volatile anesthetic-induced liver injury, additional steps were taken. The patient had received two anesthetics in 3 months: developed a relapsing fever postoperatively, which could suggest the presence of an immune-mediated process; and also developed significant increases in serum transaminases. The immunohistochemical evidence was supportive since the trifluoroacetyl-modified proteins were detected in centrilobular areas, suggesting drug-induced liver injury, and were associated with cytoplasmic organelles, suggesting specificity. Surprisingly, the trifluoroacetyl-modified proteins were detected after 72 h at concentrations previously not seen with isoflurane. Surely, the increased amount of trifluoroacetyl-modified proteins may have been caused by reduced hepatic clearance from global hepatic dysfunction induced by other causes. However, if this was the case, anesthetic metabolism should have been impaired as well.

Our gastroenterology colleagues completed another step in the journey by documenting identical histologic evidence of hepatotoxicity in a patient 5 days after isoflurane exposure for a different surgical procedure. We would like to thank Dr. Wark for his comments. We will only move forward by having these kinds of discussions.

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References


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Oral to Nasal Endotracheal Tube Exchange in a Difficult Airway: A Novel Method

To the Editor—There is a significant risk of losing a proven difficult airway during nasal to orotracheal tube exchange. Various methods are described to achieve such conversions, but they are associated with technical disadvantages intrinsic to the method, lack of an optimal aid, procedural complications, and the stage at which the need for conversion arises.

We successfully converted an oral endotracheal tube (ETT) to a nasal ETT in an 11-yr-old girl presenting for reconstruction of a postmandibulectomy defect secondary to osteogenic carcinoma. Airway assessment revealed agnathia, bucked incisors, a large tongue occupying the oral cavity, postradiotherapy contracture and scarring of the anterior neck, and a posttraechecotemy scar. Neck mobility was adequate, and the nares were patent. The lateral view radiograph of the head and neck showed a large tongue base, an anteriorly placed larynx, and a noncompressed air column.

In view of the aforementioned findings, a flexible fiberoptic bronchoscope (FFB)–guided nasotracheal intubation was planned under sedation. Premedication included 150 mg ranitidine orally and intramuscular glycopyrrolate 1 h prior to the procedure. Informed consent was obtained from the parents after explaining the procedure. The child was moved to the operating room, and routine monitoring was applied. The nasopharynx was anesthetized with 10% lidocaine spray. Propofol, 1 mg/kg, was administered, and the FFB (FB-10X; Pentax, Tokyo, Japan) was introduced via the right naris and advanced further while the child breathed 100% oxygen via a nasal cannula placed in the left naris. The fibroscope repeatedly met the large tongue base, obstructing the view. Oral fibroscopy also failed to negotiate the base of the tongue on multiple attempts. By this time, the child had desaturated to an oxygen saturation of 90%. Flexible fiberoptic bronchoscopy was abandoned. Direct laryngoscopy (Miller blade #2) showed a Cormack and Lehane grade III cords view. A 5.5-mm uncuffed ETT was blindly placed into the trachea with the help of a stylet. One hundred percent oxygen was given, and satuartion improved. As per the requirement of the procedure, oral to nasotracheal tube conversion was then performed. The FFB was advanced via the right nares and retrieved orally. The proximal end of the existent oral ETT holding the connector was cut. The fibroscope was passed through the oral tube to just above the carina. The tube-FFB assembly was grasped with fingers inside the oral cavity, and the tip of the fibroscope was anteriorly flexed to prevent dislodgment. The fibroscope and the ETT were gradually withdrawn retrogradely through the nasal passage and out of the nares. The FFB was removed, bilateral air entry was reconfirmed,
and the ETT was secured. The conversion took 80 s and was atraumatic and without any desaturation. Thereafter, anesthesia was maintained with 1–2% halothane and 66% nitrous oxide in oxygen.

The FFB may be a less than ideal device to gain airway control in certain difficult airway situations.⁵ Oraly or nasally guided flexible fiberoptic bronchoscopy may fail to locate the glottis adequately in conditions in which the airway anatomy is distorted.⁵,⁶ Partial and complete airway obstruction add to the difficulty of fiberoptic, and certain maneuvers⁷,⁸,¹² and intubation¹³ have proven effective in opening up the airway.

Our patient was a child with major airway abnormalities. This resulted in the following problems: (1) the maneuvers designed to aid oral and nasal flexible fiberoptic bronchoscopy were inapplicable, (2) we could not have kept the child awake before airway control, and (3) nasotracheal intubation was necessary. The former two presumably led to the failure of our FFB-aided intubation attempts. The last problem posed a significant challenge. We were able to convert the existing oral ETT to a nasotracheal tube with the help of the FFB, but the limitations of this method can be manifold, such as breakage of optical fibers, trauma, failure, and/or inadvertent extubation. An uncuffed polyvinyl chloride ETT is flexible enough to allow bending without transmitting tension to the FFB. The absence of jaw and tongue retraction led to minimal oral space in our patient, which significantly decreased the arc of negotiation of the FFB-ETT unit and consequently prevented damage to the optical fibers. Moreover, the possibility of accidental extubation was lessened, as the tube and the FFB were firmly grasped, the tip of the fibroscope was anteriorly flexed, and the pull of the FFB through nose was gentle. FFBS have been utilized previously to effect safer conversion of a nasal tube to an oral tube.⁵,¹¹ Ours is probably the first case in which an oral to nasal ETT conversion was carried out with the help of a FFB. This case not only reiterates the few significant limitations of the FFB while gaining a difficult airway, but it also shows that deft handling of this delicate equipment may improve outcome.

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Use of the Arndt Wire-guided Endobronchial Blocker


Reference

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Fig. 1. Sheared balloon of the Arndt endobronchial blocker. Insert shows the endobronchial blocker passing through the Tuohy-Borst valve (arrow) of the special bronchoscopy port.
To the Editor:—For difficult airway management in pediatric patients, a technique using a laryngeal mask airway (LMA™; Laryngeal Mask Company, Nicosia, Cyprus), a fiberoptic bronchoscope (FOB), wire insertion, and an endotracheal exchange catheter has been described.1,2 We have employed a similar technique in adults when dealing with an unanticipated difficult airway after induction of anesthesia. In an anesthetized patient, the technique is as follows: insertion of an appropriate-sized LMA™ and ventilation of the patient; passage of a FOB through a collapsible elbow connector, through the LMA™, and into the trachea; passage of a guide wire (0.081-cm diameter, 145-cm length; Cook Urological, Spencer, IN) through the FOB suction port and into the trachea; withdrawal of the FOB, leaving the wire in place; passage of an endotracheal tube exchange catheter (Cook Critical Care, Bloomington, IN) over the wire and into the trachea; withdrawal of the LMA™, and insertion of a standard endotracheal tube over the exchange catheter. Direct laryngoscopy is useful at this point to guide the endotracheal tube through the mouth and under the epiglottis. Assuming that ventilation with a LMA™ is successful, this technique allows almost continuous control of the airway with manual or mechanical ventilation except during the last step; an unhurried sequential approach allowing deliberate use of the FOB; minimal trauma, as the devices employed are designed for soft tissue use; and final intubation with a conventional endotracheal tube of adequate size. Of note, we have observed that a standard LMA™ works best rather than an intubating type.

Larger endotracheal tubes (sizes ≥ 7.0) may be difficult to insert directly through a LMA™ over a FOB, and the endotracheal tubes used with intubating airways have a higher pressure cuff than standard tubes. This is of greater importance when postoperative ventilation is anticipated. Smaller endotracheal tubes placed over a FOB and through a LMA™ may secure an airway but may result in problems with weaning or may be generally unsuitable for prolonged ventilation, especially in large patients.

We have used this technique successfully many times during the last 3 yr, and it is now our initial approach to the unanticipated difficult airway in patients undergoing cardiovascular surgery.

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