blood lidocaine/bupivacaine concentration also should be available in order to label this as a case of unexplained postoperative apnea in the neonate.

Unexplained postoperative apnea in the preterm neonate is a well-recognized potential problem. In the full-term infant it is, as Coté and Kelly say, rare. While the case for intensive postoperative monitoring of all such patients can hardly be disputed, the case for equally impeccable intraoperative attention to detail is well illustrated. More importantly, as with halothane hepatitis, unexplained postoperative apnea remains a diagnosis of exclusion when every other more common cause has first been considered and ruled out satisfactorily. In order for a case to be considered unexplained postoperative apnea, the burden of proof lies in establishing that the known events could not have caused the observed signs.

BUPESH KAUL, M.B., B.S., M.D., F.F.A.R.C.S.
Clinical Lecturer
RANDALL C. CORK, M.D., PH.D.
Associate Professor
Department of Anesthesiology
University of Arizona Health Sciences Center
1501 North Campbell Boulevard
Tucson, Arizona 85724

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In Reply—In response to Kaul and Cork’s letter, the date of this anesthetic was well before our “enlightened” views to preoperative fasting. This was a pediatric outpatient who had, in fact, been kept non per as from midnight, and her estimated fluid deficit prior to beginning surgery was approximately 150 ml. That, plus the hour in the surgical procedure itself, makes a total fluid deficit of approximately 145 ml. The patient’s entire deficit and maintenance fluids were replaced over a brief period of time (2 h), and although this may not be “standard” anesthetic practice, this is not an uncommon event in many pediatric operating rooms. Because the patient had a completely uncomplicated anesthetic, and because we did, in fact, auscultate the chest, which was clear, there was no apparent reason to perform invasive studies such as blood gases, electrolytes, osmolality, or even a chest x-ray. In fact, many of these studies were later obtained in the intensive care unit while the child was carefully observed and demonstrated to have continued apnea spells.

This child was believed to have all the stigmata by history, physician examination, and abnormal pneumogram, of a child with sudden infant death syndrome (SIDS). However, the only reason for not labeling the child as a SIDS baby was that technically, without a history of a SIDS event, the child could not be so-labeled. Interestingly, as we noted in the Case Report, with the institution of theophylline therapy, the child’s pneumogram became normal.

We agree with Kaul and Cork that other factors may have contributed to the child’s manifesting this respiratory abnormality, but that does not detract from the fact that the child had an abnormality of ventilation, regardless of what factor stressed the system sufficiently to cause clinical manifestation. Iatrogenesis did not cause an abnormality of the respiratory center. Iatrogenesis may have made it clinically manifest, which was fortunate for this child, because it may well have saved this child’s life.

CHARLES J. COTÉ, M.D.
Associate Professor of Anaesthesia
Associate Anaesthetist
Department of Anesthesia
Massachusetts General Hospital
and Harvard Medical School
Boston, Massachusetts 02114

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Concerns Regarding Barotrauma during Jet Ventilation

To the Editor—We previously recommended several possible high oxygen pressure jet ventilation systems for use with intravenous catheters and tube exchangers (i.e., a jet stilet). Although one of these systems included an additional in-line regulator for reducing the 50-psi wall pressure to some lower value (see fig. 1 of ref. 1), we did not state that the pressure should routinely be reduced. However, additional scientific data and several anecdotal reports strongly indicate that the initial driving pressure should be less than 50 psi (as adjusted by the in-line regulator). First, in a lung model with a clinically relevant (and variable) lung compliance and airway resistance (i.e., moderate to severely deranged), we have shown that the tidal volume through 14- and 16-G intravenous catheters and large- and medium-sized tube ex-
changers with a 50-psi oxygen source and a 1-s inspiratory time may be as large as 2,000 ml. Second, the distal tip of a tube exchanger may be in one lung or one lobe of a lung, thereby greatly limiting the amount of lung that participates in the tidal volume. Finally, we are aware of several cases of pneumothoraces that occurred in association with the use of 50-psi jet ventilation.

For all of these reasons, it is clear that a reliable means of regulating the jet ventilation tidal volume, such as an additional in-line regulator, is necessary to reduce the risk of jet ventilation induced barotrauma. We do not think it is possible to reliably limit the tidal volume by trying to depress mechanically the inspiratory button for less than 1 s, especially in anxiety-laden resuscitation situations. We now recommend the routine inclusion of an in-line pressure regulator (cost is $40) and the use of an initial driving pressure of less than 50 psi. The initial driving pressure used should depend on such factors as predicted lung compliance and airway resistance, size of the patient, and in the case of a tube exchanger, depth of insertion. The tip of the tube exchanger should not be distal to the trachea carina; i.e., one must be cognizant of the external centimeter markings. After the first few breaths, one can either decrease or increase the driving pressure depending on subsequent clinical and monitoring observations. When in doubt, it seems prudent to start with the driving pressure at a low level, increasing it as dictated by the clinical response.

JONATHAN L. BENUMOF, M.D.  
Professor of Anesthesiology

SHEILA D. GAUGHAN, M.D.  
Resident in Anesthesia  
Department of Anesthesiology  
UCSD Medical Center  
Mail Code 8812  
225 Dickinson Street  
San Diego, California 92103-8812

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ANNOUNCEMENT

The American Board of Anesthesiology (ABA) will administer its written examination for certification of Continued Demonstration of Qualifications (CDQ) on Saturday, May 15, 1993, and Friday, October 1, 1993. Diplomates of the ABA who are interested in participating in the voluntary CDQ program may request an application by writing to the Secretary, American Board of Anesthesiology, 100 Constitution Plaza, Hartford, Connecticut 06103-1796. The deadline for receipt in the Board office of completed applications for the May examination is November 15, 1992. The deadline for receipt of applications for the October examination is April 1, 1993.