Is Bright Room Light Related to Retrolental Fibroplasia?

To the Editor—I enjoyed reading Dr. Flynn’s excellent discussion of oxygen and retrolental fibroplasia. It is established that acute retrolental fibroplasia is more common in the premature infant than in the baby born at term and that the disease sometimes can be related to high concentrations of inhaled oxygen. Not being trained in ophthalmology, I should like to question Dr. Flynn concerning another possible etiologic factor of retrolental fibroplasia, namely prolonged or intense exposure to bright room light.

The proper development of an organ or part would seem to depend upon its receiving a suitable, but not excessive, workload at suitable intervals. If the required work becomes excessive, compensatory growth and vascularization can result. Thus, the heart hypertrophies from sustained hypertension, and the thyroid gland becomes very vascular during hyperthyroidism.

The workload placed upon the retina would seem to be a direct function of the intensity of received light. The eyelids of a premature baby are thin and might transmit considerable light. In addition, the retina of a premature infant might be stressed readily by bright light.

I have noticed that the rooms in which premature infants were cared for often were brightly lit during many hours of the day and night. Also, the infants eyes often were not shielded from the room light.

I wonder whether premature eyes have poor tolerance of bright room lighting. Could such exposure, especially if continuous and in the presence of elevated oxygen tension, contribute to retinopathy and loss of vision?

I also wonder whether sustained bright room light can interfere with the baby’s resting, sleeping, and other activities.

Albert D. Warshauer, M.D.
1608 East Fifth Street
Greenville, North Carolina 27834

Reference
(Accepted for publication July 16, 1984.)

In reply—Dr. Warshauer raises the interesting point about the role of light and prolonged light exposure in the newborn intensive care unit that possibly could contribute to the development of retinopathy of prematurity. It is interesting that Terry, the man who originally described retrolental fibroplasia, first suspected light as the etiologic agent. A study done by Locke and Reese attempted to answer this question by patching one eye of a group of high-risk infants. Their results showed no difference in the incidence of cicatrinal retrolental fibroplasia. This subject recently has been explored by Glass, et al., who reexamined the question in a group of neonates kept in an incubator covered with a neutral density filter versus a group kept in normal, nonfiltered incubators. They found that the incidence of retinopathy of prematurity (the acute disease) was statistically higher in the unprotected group at each birthweight level. The key factor in their experience may have been in the restriction of light to the whole body surface area.

The role of light quanta in the production of toxic forms of oxygen, such as singlet oxygen and O₂-, is unclear but potentially an important one. The role of light therapy in neonatal jaundice is well established and therapeutically beneficial. It may be that excessive light for some premature infants may have harmful side effects as well.

John T. Flynn, M.D.
Professor of Ophthalmology
Department of Ophthalmology
Bascom Palmer Eye Institute,
University of Miami School of Medicine
Miami, Florida 33101
REFERENCES


(Accepted for publication July 16, 1984.)

Iatrogenic Airway Foreign Body

To the Editor—Aspiration of a foreign body is a common problem confronting pediatric anesthesiologists. We wish to report a case of iatrogenic airway foreign body.

A 10-year-old with “short bowel syndrome” presented for placement of a central line for parenteral nutrition. He was brought to the operating room without premedication. Through a previously placed iv, anesthesia was induced with ketamine, and continued with nitrous oxide, oxygen, and halothane by mask. Using a Laryngo-O-Jet® (International Medication Systems, Ltd., South El Monte, California), the vocal cords were sprayed with 4% lidocaine. The spray device was noted to be shortened markedly when it was withdrawn. Repeat laryngoscopy did not reveal the fragment in the pharynx. Bag and mask ventilation with 100% oxygen and halothane was resumed without difficulty. The surgeon performed a rigid bronchoscopy, retrieving a 7.5-cm length of plastic tubing from the right mainstem bronchus. The procedure was completed without incident. There were no long-term sequelae.

Subsequent examination of the spray device revealed that a break had occurred at one of the side holes (fig. 1). The rigid plastic tubing may have been cracked by an attempt to curve it to facilitate its passage through the vocal cords. In addition, several instances of damage in shipping have been reported to the manufacturers.

We recommend that anesthesiologists using these devices avoid attempts to reshape the tubing and that they examine the spray carefully both before and after use.

Cecelia Hard, M.D.
Clinical Fellow in Anesthesia
Harvard Medical School
Fellow in Anesthesia
The Children’s Hospital

Charles D. Nargoziian, M.D.
Instructor in Anesthesia
Harvard Medical School
Assistant in Anesthesia
The Children’s Hospital

300 Longwood Avenue
Boston, Massachusetts 02115

(Accepted for publication July 16, 1984.)

The Jury Is Still out in the Case of Isoflurane versus Halothane in Neurosurgical Patients

To the Editor—Todd and Drummond1 draw questionable conclusions from their observations on the effects of isoflurane and halothane on cerebral circulation and metabolism. The authors’ main observations were: 1) Halothane and isoflurane in equipotent concentrations caused similar rises in the intracranial pressure (ICP). 2)