Oxygen and Retrolental Fibroplasia in Neonates

A SPECIAL CONFERENCE of North American pediatricians, ophthalmologists, physiologists, pathologists, and biophysicists was convened in Des Plaines, Ill., in June 1967 to consider current practices of oxygen administration in the management of hypoxemic prematurely born infants. The objectives of the conference were to promote improved communication and cooperation among these groups in the investigation of the results of present-day oxygen therapy, with particular reference to the risk of retrolental fibroplasia.

Retrolental fibroplasia, first described in 1942, occurs almost exclusively in premature infants during the first 3 months of postnatal life. About 30 percent of the infants who develop the disorder become blind. Oxygen administered in concentrations in excess of that in air for prolonged periods of time was identified in the early 1950's as the sole and sufficient causative agent.

At a symposium on retrolental fibroplasia held during the 1954 meeting of the American Academy of Ophthalmology and Otolaryngology, participants recommended that (*a*) routine administration of supplemental oxygen to premature babies be discontinued, (*b*) it be given only if infants are cyanotic or show signs of respiratory distress, and (*c*) oxygen therapy be discontinued as soon as respiratory distress is relieved.

As the use of oxygen was drastically curtailed in nurseries throughout the world, the incidence of retrolental fibroplasia dropped sharply, although blindness from too much oxygen is still seen in some children. Since the current trend in the use of oxygen is toward a determinative policy, rather than a rigid, restrictive policy, a critical evaluation of the risk of retrolental fibroplasia in infants was considered imperative.

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Respiratory Distress Syndrome

A respiratory distress syndrome is observed in roughly 10 percent of neonates born before 37 weeks of gestation and is the most frequent single cause of death among neonates. Evidence suggests that deaths associated with this disorder increased during the decade of rigid oxygen restriction (1955–65) in nurseries for premature infants, but evidence also suggests that the frequency of neurologic sequelae, that is, cerebral palsy, rose as the incidence of retrolental fibroplasia fell.

In the past 2 or 3 years, a concerted effort has been made to improve the outcome in hypoxemic infants with neonatal respiratory distress by administering supplemental oxygen in concentrations sufficient to relieve arterial desaturation. Moreover, asphyxia in the immediate neonatal period is treated by resuscitation and exposure to high concentrations of oxygen (80 to 90 percent) in an effort to forestall the development of the respiratory distress syndrome by decreasing pulmonary arterial tone and thereby increasing pulmonary blood flow.

If the infant's condition stabilizes, the oxygen concentration is lowered slowly after 24 hours. In some nurseries, oxygen in high concentrations is continued until the expiratory grunt, virtually a constant sign in respiratory distress syndrome, disappears. In others, treatment with oxygen in high concentrations continues for only the first 2 or 3 hours after birth.

These oxygen administration practices are

usually monitored by serial measurement of oxygen tension and saturation in arterial blood or arterialized capillary blood. The facilities for reliable measurements of arterial oxygenation, however, are not available in most hospitals, and it is feared that undetected hyperoxia may occur under these circumstances. Moreover, there is considerable disagreement concerning the interpretation of arterial oxygen measurements in various sites, such as the radial artery, the temporal artery, and the abdominal aorta below the ductus arteriosus, with respect to the risk of retrolental fibroplasia. Evidence concerning the untoward effects of hyperoxia on other organs, especially the lungs and the brain, is accumulating, and the evidence of these effects undoubtedly will also influence eventual decisions concerning the optimum method for treating hypoxemia in newborn infants.

Considerable discussion centered around the following issues: (*a*) the need for criteria for supplemental oxygen administration; (*b*) the need for accumulating evidence on the association of clinical signs, arterial oxygen measurements, funduscopic appearance, and psychomotor development in oxygen-treated infants; (*c*) the need for improved devices for monitoring ambient oxygen concentration; (*d*) the need for caution in adminstering supplemental oxygen when appropriate observations, as indicated in (*b*) cannot be made; and (*e*) the need for basic research in factors which control vasomotion.

Conclusions

The conference emphasized the current importance of increased understanding of mutual problems by pediatricians and ophthalmologists. Premature infants with major illnesses can be treated adequately only in optimally manned and fully equipped intensive care units. Current support in terms of trained manpower, funds, and equipment for these facilities is woefully inadequate. Ophthalmologists must examine every premature baby receiving supplemental oxygen, and the eyes of children born prematurely should be examined regularly for the first 2 years of life.

Participants in the conference agreed that revised recommendations for oxygen administration are highly desirable but that currently available data are insufficient to justify a revision of the present cautious recommendations. A vital need exists for extensive research in these areas, along with the accumulation of considerably more clinical data.—Dr. William A. Silverman, *Columbia University College of Physicians and Surgeons.*

Conference Participants

Dr. Peter A. M. Auld
New York Hospital-Cornell
University Medical Center
New York City, N.Y.

Dr. Philip G. Banister
Department of Health, Education, and Welfare
Ottawa, Ontario, Canada

Dr. Richard Behrman
University of Oregon Medical School
Portland, Oreg.

Dr. William C. Cooper
Presbyterian Hospital
New York, N.Y.

Dr. John J. Downes
Children's Hospital of Philadelphia
Philadelphia, Pa.

Robert W. Flower
Johns Hopkins Hospital School of Medicine
Baltimore, Md.

Dr. Anita P. Gilger
Cleveland, Ohio

Dr. Margaret Henry
University of California
San Francisco, Calif.

Dr. Donald R. Johnson
University of Oregon Medical School
Portland, Oreg.

Dr. V. Everett Kinsey
Institute of Biological Sciences
Rochester, Mich.

Continued

Dr. Marshall Klaus
University Hospitals of Cleveland
Cleveland, Ohio

Dr. Jonathan T. Lanman
State University of New York
Downstate Medical Center
Brooklyn, N.Y.

Dr. John C. Locke
McGill University
Montreal, Quebec, Canada

Dr. Andrew McCormick
University of British Columbia
Vancouver, B.C., Canada

Dr. Richard E. Nachman
The Johns Hopkins Hospital
Baltimore, Md.

Dr. Thomas K. Oliver, Jr.
University of Washington School of Medicine
Seattle, Wash.

Dr. Arnall Patz
Johns Hopkins Hospital School of Medicine
Baltimore, Md.

Dr. David B. Schaffer
University of Pennsylvania Hospital
Philadelphia, Pa.

Dr. Sydney Segal
University of British Columbia
Vancouver, B.C., Canada

Dr. William A. Silverman
Columbia University College of Physicians and Surgeons
New York City, N.Y.

Dr. John C. Sinclair
Columbia University College of Physicians and Surgeons
New York City, N.Y.

Dr. Mildred T. Stahlman
Vanderbilt University School of Medicine
Nashville, Tenn.

Dr. Earl Stern
University of California Medical Center
San Francisco, Calif.

Dr. Leo Stern
Montreal Children's Hospital
Montreal, Quebec, Canada

Dr. Avron Y. Sweet
Cleveland Metropolitan General Hospital
Cleveland, Ohio

Dr. Paul Swyer
University of Toronto
Toronto, Ontario, Canada

Dr. William H. Tooley
University of California Medical Center
San Francisco, Calif.

Dr. Leonard Apt, Observer
University of California School of Medicine
Los Angeles, Calif.

Staff

Dr. J. S. Drage
National Institute of Neurological Diseases and Blindness
Bethesda, Md.

Dr. Eileen G. Hasselmeyer
National Institute of Child Health and Human Development
Bethesda, Md.

Dr. John W. Ferree
National Society for the Prevention of Blindness, Inc.
New York City, N.Y.

Mrs. Virginia S. Boyce
National Society for the Prevention of Blindness, Inc.
New York City, N.Y.