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Retrolental Fibroplasia and Oxygen Administration during General Anesthesia

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Retrolental fibroplasia is a major cause of blindness and impaired vision in low-birth-weight infants. When the retinal vasculature is immature, elevated inspired oxygen concentrations ($F_{I_{O_2}}$), resulting in arterial oxygen tensions (Pa_{O_2}) above normal, increase the incidence of retrolental fibroplasia.[1] This case report describes a twin premature infant without cardiopulmonary disease in whom retrolental fibroplasia developed following administration of oxygen during and after general anesthesia. The infant's twin sister required an elevated $F_{I_{O_2}}$ during a three-day course of treatment for respiratory distress syndrome, yet retrolental fibroplasia did not develop.

REPORT OF A CASE

Twin "A". A white female 1,140-g infant of 32 weeks' gestational age had occasional apneic spells during the first two days of postnatal life. Respiratory distress syndrome did not develop and the infant received no increase of $F_{I_{O_2}}$ until the age of 3 days. At that time, the infant showed signs of high-intestinal obstruction, attributed to duodenal atresia. During nitrous oxide–*d*-tubocurarine anesthesia, and in the immediate postoperative period, the infant received an elevated $F_{I_{O_2}}$ for a total of 275 minutes. Pa_{O_2}'s of radial arterial blood samples obtained percutaneously ranged from 64 to 325 torr (fig. 1). The infant's postoperative course was uneventful except for the development of retrolental fibroplasia, first seen in the active stage when the infant was 7 weeks old (table 1), and confirmed in the cicatricial stage (grade II) when she was 7 months old (fig. 2) by examination under general anesthesia.

TABLE 1. Ophthalmologic Examination Results, Twin "A"

Age	
16 days	Premature eyes
7 weeks	Stage II active retrolental fibroplasia
7 months	Grade II cicatricial retrolental fibroplasia
	Refraction: OD myopia (−4.25s)
	OS myopia, astigmatism (−3.50 + 1.00/135°)

Twin "B". The patient's twin, a 1,440-g female infant, had respiratory distress syndrome at birth, and an elevated $F_{I_{O_2}}$ was necessary until she was 79 hours of age. Pa_{O_2}'s of blood samples obtained via an umbilical-artery catheter ranged from 23 to 235 torr ($F_{I_{O_2}}$ 0.3–0.6). The first Pa_{O_2} after discontinuing oxygen administration was 47 torr. This infant recovered fully, without development of retrolental fibroplasia, and was discharged with normal eyes.

DISCUSSION

To our knowledge, the case of Twin "A" is the first reported case of an infant in whom retrolental fibroplasia developed following administration of oxygen limited to the course of general anesthesia for an operation. The recent literature[1–6] emphasizes that many factors may be involved in the development of retrolental fibroplasia besides elevated $F_{I_{O_2}}$, and the disease is better described as "retinopathy of prematurity."[7] During breathing of air in otherwise normal low-birth-weight infants, mean Pa_{O_2} increases from 60 ± 8 (SD) torr at age 3 hours to 78 ± 16 torr at 3 days, where it remains throughout the first month.[8] Even at these Pa_{O_2}'s, retrolental fibroplasia will develop in some premature infants. Severe stages of retrolental fibroplasia have occurred in infants with cyanotic

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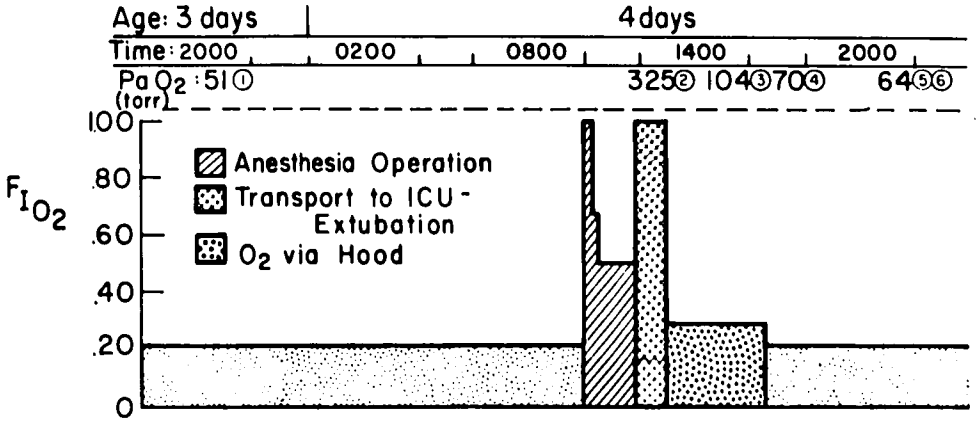

FIG. 1. Oxygen administration and Pa_{O_2} in Twin "A" (birth weight 1,140 g). 1) Admission to The Children's Hospital of Philadelphia at $F_{I_{O_2}}$ 0.21. 2) Thirty minutes post-anesthesia, prior to tracheal extubation, breathing unassisted at $F_{I_{O_2}}$ 1.00. 3) Thirty minutes post-extubation at $F_{I_{O_2}}$ 0.28. 4) Thirty minutes at $F_{I_{O_2}}$ 0.21. 5) Six hours at $F_{I_{O_2}}$ 0.21. 6) Pa_{O_2} 65 torr after 18 hours at $F_{I_{O_2}}$ 0.21.

congenital heart disease,[3] stillborn infants,[4] and infants who received no additional oxygen.[4,5] However, an increased Pa_{O_2} is an important contributory factor,[1] and any anesthesiologist dealing with infants less than 40–44 weeks of post-conceptual age must be aware of this hazard, because the retinal vasculature is not mature in the temporal periphery until after birth of the *full-term* infant.[9]

We advocate frequent determination of Pa_{O_2} whenever $F_{I_{O_2}}$ exceeds 0.21 during and after anesthesia in infants less than 44 weeks of post-conceptual age. Samples should be obtained from the radial artery, preferably the right, or a temporal artery.[1] These sites avoid the discrepancies in Pa_{O_2} obtained with blood from an umbilical-artery catheter placed below the ductus arteriosus, which may yield lower values because of intermittent pulmonary-to-aorta shunting.[10] Anesthesia machines should be equipped with a means (*e.g.*, air flowmeters) to permit reduction of $F_{I_{O_2}}$ to 0.21 when necessary to maintain

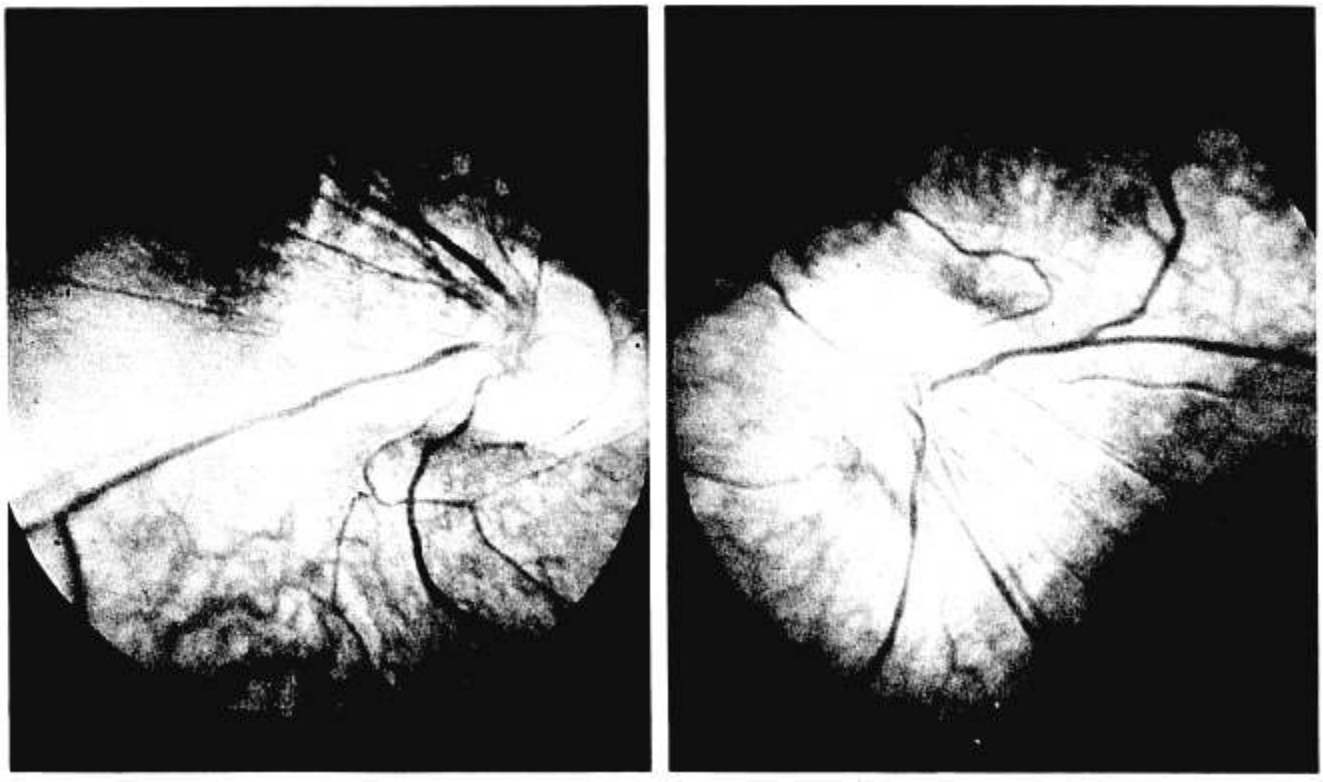

FIG. 2. Fundi of Twin "A". *Left*, right eye. *Right*, left eye. Photographed by DBS when the infant was 7 months old. Both show grade II cicatricial retrolental fibroplasia with temporal dragging of the retina resulting in macular distortion and impaired vision.

the Pa_{O_2} in the normal range of 60 to 80 torr.[8] A lower Pa_{O_2} may result in increased pulmonary vascular resistance[11] and retard circulatory adaptation of the newborn;[12] a higher Pa_{O_2} may increase the incidence of retrolental fibroplasia.[1]

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Serum Cholinesterase Activity Following the Use of Methoxyflurane in Obstetrics

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Serum cholinesterase activity tends to be low in normal women during pregnancy and labor and in the postpartum period.[1,2] The exact mechanism for this decrease is unclear, but occasionally the levels are sufficiently reduced so as to result in prolonged paralysis following normal clinical doses of succinylcholine.[2,3] Recently, it has been suggested that the use of methoxyflurane may result in a further decrease in serum cholinesterase activity (Shnider, S. M., personal communication). One of the metabolic products of methoxyflurane, inorganic fluoride ion, is capable of inhibiting normal cholinesterase, a property utilized in the identification of the fluoride-resistant variant of serum cholinesterase.[4] Because of the continuing frequent use of methoxyflurane and succinylcholine in patients undergoing labor and delivery or cesarean section, we determined the effect of methoxyflurane administration on serum cholinesterase activity in normal parturients undergoing elective cesarean section.

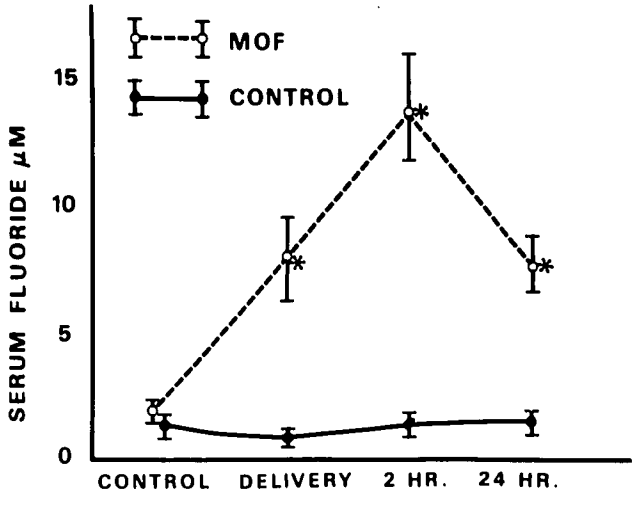

Fig. 1. Serum inorganic fluoride levels ± SE. * = significantly different from the control value for the methoxyflurane group and significantly different from the non-methoxyflurane group.

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