EDITORIAL

The best of the papers given at the 1988 meeting of the Oxford Ophthalmological Congress are to be found in this edition of "Eye".

Thirty seven years ago a session of the Oxford Ophthalmological Congress was devoted to the retinopathy of prematurity although at that time it was starting to be called "retrolental fibroplasia". In those days it was very much the concern of the general ophthalmologist. Papers from the Oxford Region showed an estimated incidence of 0.35% of all live births in 1949. In Birmingham the incidence was given as 4.2% of babies with birthweight less than 3lb (1361g). In fact the figures varied greatly from place to place. A particularly high incidence of "retrolental fibroplasia" was found in Boston, Massachusetts. Various causative factors were suspected including the administration of oxygen. In one review in that same congress session a remarkably clear description of the preliminary stages of the condition was given including appearances now defined in Stage 2 and Stage 3. Perhaps more importantly it was shown that the premature birthrate did not rise significantly from 1945 to 1950 but the survival rate for babies weighing less than 1000g did. Some of us may recall the blind schools of that era and the fact that many of the inmates suffered from retinopathy of prematurity. In a survey of the preschool blind in Massachusetts in the late 1940s, 66% were blind from this condition. In 1956, that is five years after the original session on the subject at Oxford, the incidence of retinopathy of prematurity was shown to be greatly reduced. In fact no fresh cases had been seen in Oxford since 1953. Due recognition had been given to the work of Ashton and others which allowed the elucidation of the cause of the condition and the subsequent improvement in the care and monitoring of newborn premature infants.

Even at that time it was realised that the whole truth may not have been completely revealed and since then cases have started to reappear in spite of careful monitoring of oxygen levels and occasionally they are seen even in the absence of supportive oxygen. By the early 1980s people were beginning to talk of a "second epidemic" and an intense research interest culminated in the publication of the new International Classification of Retinopathy of Prematurity in 1985 and more detailed classification of the late stages in 1987. This classification now enables us to identify "threshold cases" defined as 5 contiguous or 8 continuous clock hours of stage 3 in zone 1 or 2 with "plus" disease. Infants weighing less than 1000g at birth are also at greatest risk. In the current series by Fielder, out of 572 preterm infants weighing less than 1700g, 4.1% showed stage 3 changes but none were blind from retinopathy of prematurity.

More recently, this year we have seen the publication of the preliminary results of the American multicentre trial of cryotherapy. The short term results of cryotherapy in these children have been shown to be promising. It is therefore even more important that children at risk should be identified. Many now agree that careful examination of infants weighing less than 1250g at birth should begin 4-6 weeks after birth or earlier and that the examination needs to be performed by an ophthalmologist experienced in examining the infant retina.

In contrast to the situation in the 1950s, the average ophthalmic surgeon in this country will not have seen many if any of these cases and still less may he or she possess the expertise to examine them. Even if the initial promises of cryotherapy are not borne out by long term studies it will be necessary to identify the patients. It looks as though the Health Service is going to need to employ more paediatric ophthalmologists than exist at present.

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