

Phototherapy: an ocular hazard revisited

Phototherapy is an established and effective treatment for neonatal jaundice.¹ There remains concern, however, that this necessary light exposure may pose a potentially noxious stimulus to the eyes of neonates.^{2–4} Recent studies have suggested that, irrespective of phototherapy, prolonged exposure to 'bright' ambient light may damage cone photoreceptors³ and increase the incidence of retinopathy of prematurity.⁵ In view of these findings it is perhaps timely to reconsider the potential ocular hazard posed by phototherapy and the practices required to minimise the likelihood of damage occurring.

The efficacy of 'patching'

The eyes of phototreated infants are routinely 'patched', because it has been shown that the intensity of light in phototherapy units can cause retinal damage in animals.² In a study of 12 different eyeshields obtained from neonatal units within the United Kingdom, Chin *et al*⁶ found that all shields tested were extremely effective in reducing the intensity of incident light. They and others,⁷ however, noted that patches are prone to slip. Such occurrences are explicable on two counts: it is often difficult to secure eyeshields effectively and, in addition, nursing staff often think of 'patching' as of secondary importance to more immediate and potentially life preserving interventions.

To what extent does inadequate patching—or indeed no patching at all—increase the ocular hazard posed by phototherapy? In fact there is little evidence that phototherapy has caused retinal damage in human infants. This may be due partly to limited follow up testing, and partly to the fact that subtle damage may go undetected. Most studies, however, have reported carefully 'patched' infants who may not be expected to suffer damage.⁸

Animal experiments

The case for routine patching of infants' eyes during phototherapy is mostly based on extrapolations from animal studies. Here an important distinction must be made between the substantial number of reports about ocular phototoxicity^{9 10} and the relatively few studies that have, in an attempt to mimic clinical practice, used an appropriate (that is, neonatal) animal with light exposure obtained from

a phototherapy unit. Of the latter more relevant studies, Sisson *et al*¹¹ exposed newborn piglets (36 hours old) to phototherapy for 72 hours. In contrast to human neonates, the piglets' pupils were dilated to compensate for their orientation at 90° to the light source. At necropsy pronounced histological damage to rod and cone photoreceptors was found, and this was attributed to the light exposure. In a similar study, Messner *et al*¹² found retinal damage in the eyes of newborn monkeys that were exposed to phototherapy for up to seven days; in this experiment, however, the animals were restrained so that they directly faced the light source.

Neither of these studies accurately represents the conditions prevailing in clinical practice. Firstly, human neonates undergoing phototherapy are never purposely restrained to face the light source, nor will their pupils be kept dilated during treatment. Secondly, the amount of time the experimental animals spent with their eyes open was not addressed; studies in humans have shown that premature neonates may spend more than 80% of the time with their eyes fully closed.^{13 14} If the transmission of light through the closed eyelids is similar to that of the adult, there will be a wavelength dependent reduction in the intensity of light reaching the cornea of between 10 to 100 times.¹⁵

The developing human eye

Consideration of the developing human eye emphasises that further caution is warranted when extrapolating from animal studies. For example, corneal haze¹⁶ and the transient tunica vasculosa lentis (present in the eye of the premature neonate until about 34 weeks' gestation) may reduce the intensity of light reaching the retina. Though photoreceptors in the peripheral retina of the human neonate are relatively well developed at full term, the macular region remains poorly differentiated.^{17 18} Similarly, retinal vascular development is not complete until just after term.¹⁹ The relevance of these observations to ocular phototoxicity in human neonates has yet to be addressed but they highlight the difficulties of extrapolating from animal experiments.

Conclusions

We conclude that further research is required to evaluate the ocular hazard posed by phototherapy.

Firstly, future animal studies should be designed in such a manner as to ensure that the conditions prevalent in clinical practice are appropriately simulated. Secondly, estimates of typical exposures of the retina received by infants during phototherapy are currently unavailable and would be of great benefit in evaluating the potential hazard. To this end we are attempting to measure both the environmental (for example, the intensity and duration of light exposure) and biological (for example, eyelid opening, pupil size, transmittance of the eyelids, and optic media) factors that influence retinal exposure during phototherapy. It is hoped that such data can be related to known photobiological dose response associations obtained from in vivo and in vitro laboratory studies.

Should phototherapy be found to pose an important ocular hazard then the use of eyeshields needs to be afforded the appropriate clinical priority. If it is not, then the economics of 'patching' and its unknown effects on visual development may not justify the continuation of the practice.

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M J MOSELEY and A R FIELDER
 Department of Ophthalmology,
 University of Leicester,
 Clinical Sciences Building,
 PO Box 65, Leicester Royal Infirmary,
 Leicester LE2 7LX