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international—with associate editors and an editorial board from many countries. Further information about the journal and a call for papers appear in the advertisement facing p 739 (clinical research), p 734 (general practice), and p 746 (other editions). We invite doctors and others concerned with these central problems of our age to contribute to this journal and so to expand our understanding and resolution of the problems that might otherwise make life unbearable or impossible for our children and grandchildren. We must not be overwhelmed. It is better to light a candle than to curse the darkness.

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Retinopathy of prematurity

Increasing as survival of extremely premature babies increases; cryotherapy improves outcome

Retinopathy of prematurity (previously called retrolental fibroplasia) has been a known and feared cause of visual impairment in children born prematurely since 1942.¹ In the early '50s oxygen was identified as an aetiological factor, and the condition's incidence fell substantially when lower oxygen concentrations were given to premature babies. But better neonatal care has increased the survival of extremely immature children during the past few decades, and consequently the incidence of retinopathy of prematurity is again rising. Recent epidemiological studies report a total incidence of the condition of 30-50% (with severe retinopathy in up to 20%) in children with a birth weight of 1700 g or less; the risk is even higher in the most immature children.²⁻⁴

Retinopathy of prematurity is a disease of immature retinal vessels. The vascularisation of the inner retina is not completed in children born prematurely. Owing to a vascular retinal lesion, normal development may become interrupted, and pathological changes such as vascular shunts, fibrovascular proliferations, and, in the most advanced stages, tractional retinal detachment will occur. Milder stages of the condition may regress spontaneously and do not affect the normal development of the eye. Impaired vision; large refractive errors, particularly myopia; strabismus; and even blindness often follow more severe forms of the condition.

The pathogenesis of the retinopathy is not yet clearly understood. Excessive doses of and prolonged exposure to oxygen still have a role in the disease. Retinopathy of prematurity can, however, develop even without supplementary oxygen, and children who have received high doses of oxygen do not necessarily develop the condition. This suggests the existence of other factors in the pathogenesis of the disease.⁵

Retinopathy of prematurity is thought to be a multifactorial condition that mainly affects the most immature and sick children.⁶ Numerous risk factors, sometimes coexisting, have been documented, including early gestational age,

low birth weight, hyperoxia, hypoxia, hypercarbia, hypocarbia, acidosis, alkalosis, apnoea, infection, intraventricular haemorrhage, Vitamin E deficiency, bright light, blood transfusions, administration of xanthine, maternal bleeding, and administration of β blockers in late pregnancy.^{7,8} Flynn has recently suggested a genetic basis for the condition.⁹ Further research is needed on the pathogenesis of retinopathy of prematurity and, perhaps most importantly, on the aetiology and possible prevention of premature birth.

Continuous monitoring and control of the administration of oxygen have not been shown to prevent the retinopathy; a safe concentration of arterial oxygen has not been defined.⁵ Although antioxidants have been used prophylactically for several decades, they cannot be recommended for routine use. The role of bright light in the aetiology and prevention of the condition is still debated—no randomised trial evaluating the effect of exposure to light has yet been performed.¹⁰ Prophylactic treatment with surfactant in prematurely born children with the respiratory distress syndrome is routine today, but a recently published randomised study showed no change in the incidence of retinopathy.¹¹ Interestingly, benefits have been reported for supplementation with inositol, which may control the surfactant phospholipids in immature lungs.¹² Finally, dexamethasone used to prevent bronchopulmonary dysplasia in children born prematurely might be considered in the prophylaxis of retinopathy, but once again no randomised controlled trial has been performed.¹³

Since the mid-1970s cryotherapy, applied transsclerally to the peripheral, ischaemic part of the retina, has been used to treat advanced stages of the condition. It is thought to arrest the retinopathy by reducing retinal ischaemia and the release of an angiogenic factor that may act as a trigger for the disease process. Cryosurgery did not, however, become an established mode of treatment for advanced retinopathy until an American multicentre study in 1988 showed that it halved the chance of an unfavourable retinal outcome.¹⁴

Assessment three and a half years later has confirmed a structural as well as a functional benefit from the treatment, although a substantial group of children had severely impaired vision despite adequate cryotherapy.¹⁵ Earlier treatment with cryotherapy than that given in the American study has been advocated by some authors,^{16 17} and this might further improve the results.

Although cryotherapy is usually not associated with any problems, complications such as bradycardia, apnoea, chemosis, periorbital oedema, scarring, and late onset retinal detachment may occur. Another treatment of advanced retinopathy is laser treatment, which has recently been reported to be at least as effective as cryotherapy and may give rise to fewer systemic and ocular complications.¹⁸ Finally, some cases, despite cryotherapy or laser treatment, progress to retinal traction and detachment and will need surgery with scleral buckling or vitrectomy. Although this form of surgery may produce good anatomical results, the functional results are still poor.^{19 20}

As cryotherapy substantially reduces the visual handicap of premature children, paediatric ophthalmologists should be responsible for identifying those who would benefit from it. Studies on the natural course and epidemiology of the condition have resulted in screening programmes that should be in use in every neonatal unit.^{4 21 22}

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Deregulating emergency contraception

Justified on current information

The statistics on unintended pregnancy in Britain make worrying reading. Almost half of all conceptions are unplanned.¹ Over 170 000 pregnancies are terminated every year, and soon a quarter of 25 year olds will have had an abortion.² Unwanted pregnancy affects mainly (though by no means exclusively) people under 30. In London and Oxford abortion rates are highest in 20-24 year olds,³ but elsewhere the peak rate is among 19 year olds.⁴

Little is done to help young people avoid pregnancy. They are given the impression that it is normal to be sexually active, but contraception is marketed inadequately to the public.⁵ Nevertheless, about half of unwanted pregnancies result from contraceptive failure rather than a lack of use of contraception.⁶⁻⁸ The methods most used by younger couples—oral contraception and condoms—have failure rates of at least 1% even among experienced users, and failure rates are higher among young and less well educated people.⁶

About 70% of unwanted pregnancies are predictable because the woman realises that she is at risk after unplanned intercourse or an accident with a condom.⁶⁻⁸ In such cases emergency contraception offers a 98% chance of preventing pregnancy. The most effective method—insertion of an intrauterine device—is not ideal for young women, but hormonal postcoital contraception is suitable. (As it can be used 72 hours after intercourse the term "morning after pill" has been abandoned.⁹) Its failure rate, probably around 2% per cycle,¹⁰ is much higher than that of standard contraceptive methods, and postcoital contraception is therefore unsuitable for repeated use.¹¹

Surveys of women with unwanted pregnancies have shown

that 70% knew about emergency contraception but only 3% tried to use it.⁶⁻⁸ The woman may deny to herself that she is really at risk. She may not know where to obtain postcoital contraception, particularly if (as often happens) unprotected intercourse has occurred at a weekend or away from home. Another obstacle is that access to emergency contraception is controlled by doctors.

Family planning is now part of general practice, but a woman who is ashamed or embarrassed may prefer to accept what she hopes is a small risk rather than explain to a receptionist why she wants an emergency appointment with her general practitioner. Young people may prefer the relative anonymity of family planning clinics, few of which are open seven days a week. Emergency contraception may be available in accident and emergency departments, but these are not the best places for counselling about sexual behaviour.

An editorial in the *British Journal of Obstetrics and Gynaecology* has suggested that emergency contraception should be available without prescription from pharmacists.² This would be safe. The "Yuzpe" regimen (the only one currently approved by the Committee on Safety of Medicines¹³) was introduced in 1972 and has been licensed in Britain since 1984. There are no published reports of death or serious illness after its use and it does not affect clotting factors.¹⁴ It should not be given to a woman who is already pregnant (so the woman should also buy a pregnancy test), but 17% of British family planning clinics say that no other contraindications exist and 41% regard thromboembolism as the only other contraindication.¹⁵ Even if the woman is already pregnant the risks are only theoretical,