What the obstetrician does have is the freedom of choice to make decisions when he is confident that the quality of neonatal care will be excellent, however small the baby. This need not and must not be the sole prerogative of obstetricians in regional centres. Even if there were several perinatal centres in each region they would not be likely to cope with all or even most of the high-risk pregnancies presenting at district maternity hospitals. Uncritical expansion of antenatal referral services erodes the experience of medical and nursing staff in the management of common pregnancy complications—yet at the same time accommodation for such patients at the regional centre can never be guaranteed.

There would be little justification for the transfer of premature babies who are not suffering from respiratory failure and for many examples of antenatal referral if the standards of neonatal care in district maternity hospitals were improved. The essential neonatal facilities that must be maintained in district maternity hospitals can be spelt out: constantly available skilled resuscitation and short-term ventilatory support; the ability to anticipate neonatal problems and recognise them early; reliable monitoring for and prophylaxis against apnoeic attacks; scrupulous control of oxygen treatment in babies with respiratory distress; awareness of the individual baby's thermal homeostasis; and a critical approach to gavage feeding in small babies. The provision of such basic care is not costly, and it is to midwives and nurses we must turn to maintain these standards. An increase in their numbers is certainly necessary in most district maternity hospitals, but ensuring that existing staff are appropriately trained is just as important. Present training programmes, in their attempt to be comprehensive, frequently fail to teach the nuances of neonatal care. Yet in this specialty it is often nuances that dictate the difference between appropriate and poor care.

We have reached a crossroads in the regional organisation of perinatal care in Britain. Our health service administrators must not be allowed to lead us towards too much centralisation in the belief that money can be saved by diverting resources from the district maternity hospitals. What then should be our policy on neonatal and antenatal referral? Neonatal transfer for impending respiratory failure is totally justified. We need a more selective policy for antenatal transfer that would include, for example, women with fetuses severely affected by rhesus disease; those with complications of pregnancy in whom preterm elective delivery is contemplated in the face of uncertainty about pulmonary maturation or a known risk of hyaline membrane disease based on biochemical analysis of amniotic fluid; and the presence of a fetal malformation amenable to surgical treatment or with a predicted need for medical treatment. Finally, undue emphasis on neonatal and antenatal transfer should not preclude regional perinatal centres from maintaining other roles. Firstly, they should find time to improve standards of care at district maternity hospitals through the training of midwives and nurses and postgraduate medical education; secondly, they need to look at innovations in the prevention and treatment of perinatal disorders—stimulated by close working contact between geneticist, obstetrician, paediatrician, neurodevelopmental specialist, paediatric pathologist, and others; and, thirdly, they should collect and analyse regional and district statistics so that more can be learnt about the influence of different patterns of perinatal care on mortality and neurodevelopmental handicap.

MALCOLM L. CHISWICK

Consultant Paediatrician,
North-western Regional Perinatal Centre,
St Mary's Hospital, Manchester M13 0JH

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Beta-blockers in the treatment of chronic simple glaucoma

We ought not to be over anxious to encourage innovation in cases of doubtful improvement, for an old system must ever have advantages over a new one: it is established and understood. C C Colton 1825.

A preparation of timolol maleate for topical use was released in Britain in January 1979. By December 1981, 50 000 patients with glaucoma were thought to be receiving treatment with this drug. We need, therefore, to review the action of beta-blockers in glaucomatous eyes, to emphasise possible unwanted effects, and to try to identify the place of these drugs in the management of chronic simple glaucoma.

Chronic simple glaucoma is a disease treated in the main by topically applied drugs which lower the intraocular pressure. The side effects on the eye of long-term drug administration include blurred vision, irritation, and pain—and since the disease itself is largely asymptomatic except in the late stages, the side effects discourage patients from taking their drugs regularly. The results are poor control of the intraocular pressure^{1 2} and progression of the disease. Against that background there has been a great need for a tolerable, effective drug for glaucoma that can be administered topically.

It is now nearly 14 years since the first report of the effect of intravenous and oral propranolol on the intraocular pressure.³ This and other beta-blockers have had their ocular hypotensive effect confirmed both when given by mouth and when applied topically.^{4 5} Not until the advent of timolol, however, did the pharmaceutical industry come up with an acceptable combination of hypotensive effect, duration of action, and relative freedom from local side effects.

Timolol is a beta₁-adrenergic and beta₂-adrenergic blocker which has neither any intrinsic sympathomimetic action nor any important local anaesthetic properties. Each 1 ml of 0.25% solution contains 0.34 mg timolol maleate, and the drug is

given twice daily.⁶ The initial clinical studies^{6,7} and later clinical trials have shown that the hypotensive effect compares favourably with that of pilocarpine⁸ and adrenaline,⁹ while adding timolol to "maximum medical therapy" further lowers intraocular pressure.¹⁰ This additive effect is seen with both pilocarpine and acetazolamide but is less certain for combinations of adrenaline and timolol.¹¹⁻¹⁴ Long-term studies have shown that the hypotensive effect is maintained for years, though some patients develop tolerance and in them the hypotensive effect may be reduced by up to 25% after one or more months of use.¹⁵

Local side effects remain remarkably few. No patient has developed the ocular syndrome seen with oral practolol. A few patients have complained of pain and blurred vision, while punctate keratitis and corneal anaesthesia have been seen on rare occasions.¹⁶ Systemic side effects appear more common; these are due to absorption of the drug through the nasal mucosa, which allows 80% of the total dose to pass directly into the circulation without deactivation by the liver.¹⁷ Systemic side effects reported in one 11-month study included depression, anxiety, and confusional states.¹⁶ Bradycardia (affecting both resting and exercise rates), arrhythmias, and airways obstruction have also been reported.^{16,18} Timolol should not be used in known asthmatics or in patients with any form of reversible airways obstruction. Similarly it should be given with caution in patients with known heart disease.

What, then, does the future hold for beta-blockers in eye disease? The twice-a-day regimen is probably optimum, frequent enough for the patient to remember it but not so frequent as to be a burden. The hypotensive effect may not be improved on. Nevertheless, the ocular penetration of timolol is much less than that of other topically applied beta-blockers.¹⁹ Any increase in penetration (for example, development of prodrug such as dipivalyl adrenaline—converted to adrenaline within the eye)²⁰ might allow a lower total dose to be given to achieve the same hypotensive effect with a reduction in the systemic side effects. Even without these possible improvements, however, the advent of topical beta-blockers in general and timolol in particular must be seen as a major advance in the medical treatment of chronic simple glaucoma. They are likely to be responsible, along with laser surgery and better techniques for conventional glaucoma surgery, for a major realignment of ophthalmic ideas in the management of this disease.

ROGER A HITCHINGS

Consultant Ophthalmic Surgeon,
Moorfields Eye Hospital,
London WC1V 7AN

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Where am I?

Transient global amnesia is a benign phenomenon whose diagnosis depends on the recognition of characteristic clinical features. Once the diagnosis has been made expensive and unpleasant investigations may be avoided.

The syndrome was described by Fisher and Adams in 1958¹ but despite a mass of publications since that time its pathophysiology and causation remain unknown. Transient global amnesia is an alarming medical emergency for the victim and his family; for the doctor the temptation is to refer to hospital, where the patient may be overinvestigated.

The typical patient is a man aged between 50 and 70 with no relevant antecedent medical history, though an association with hypertension has been noted. The attack may be provoked by taking a shower^{2,3} or physical exertion.³ The onset is sudden, with the patient behaving in a confused manner and repeatedly asking where he is and what he is doing. Usually he is agitated and restless but with no other psychiatric features nor the purposeless automatic behaviour associated with psychomotor epilepsy. The attack lasts a few hours and recovery is rapid. During the attack complex motor skills are retained, so that the patient may dress, prepare food, use a typewriter, or drive a car safely. Usually the attack is single, but as many as three or four episodes may occur spread out over several years.

The differential diagnosis includes epilepsy, head injury, alcoholism, hypoglycaemia, encephalitis, hysteria, and migraine—and the exclusion of most of these other causes relies on clinical findings. Thus a psychomotor epileptic seizure generally lasts less time, and the patient's behaviour during the attack is characterised by automatisms and depersonalisation. Major seizures may be part of the antecedent history, but the distinction may be impossible and an electroencephalogram may be helpful.

Accidental head injury with concussion and retrograde