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 pilocarpine[8] 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.

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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 1958[1] 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

amnesia encompassing the injury may result in a postconcussive confusional state and amnesia, but there are usually signs of injury. Alcoholism with Korsakow's mental state should be recognisable by confabulation, lack of insight, and the associated malnutrition, neuropathy, and ophthalmoplegia. In hysteria the amnesic attacks are usually multiple and occur in the setting of an appropriate psychiatric history with an identifiable precipitant to the attack. Migraine has been described[4 5] in association with transient global amnesia. This association points to an ischaemic aetiology for the syndrome, and increasingly the consensus has been that it is due to vascular insufficiency affecting bilaterally the hippocampus or hypothalamus.

Several features in transient global amnesia remain unexplained. How can an ischaemic aetiology fit with the attacks usually being solitary and rarely exceeding three or four episodes? Why is the syndrome unassociated with other abnormal physical signs and symptoms and followed by complete recovery? As yet no pathological findings have been reported. One recent paper has reported a familial incidence,[6] which would be difficult to explain on an ischaemic vascular basis.

Where a patient presents during an amnesic attack, hypoglycaemia should be excluded and, if in doubt, the doctor should give intravenous glucose. On clinical examination signs of head injury should be sought, as should features to suggest alcoholism. Particular note should be made of confabulation, classically associated with hypothalamic disturbance. Loss of insight or automatic behaviour should raise suspicion of an alternative diagnosis such as encephalitis or epilepsy. When, however, a diagnosis of transient global amnesia can be made from its typical clinical features and by exclusion, sedation and a good prognosis may be given with confidence.

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Give cyclists room to move

A whole new group of people discovered some of the joys of cycling during last week's tube and rail strike when they borrowed or resurrected old bikes in desperate attempts to get to work. They discovered only some of the joys because instead of pedalling along cheerfully in the sun they had to spend much of their time squeezing through rows of stationary cars. If local authorities and the Government had seen fit to provide cycle routes, lanes, and crossing places, then these tiro cyclists could well have reached work more quickly than usual, feeling brighter, and with saved pounds in their pockets.

Fear of being injured is the main factor stopping more people cycling to work, and they are right to be worried by this. The latest figures show that all accidents to cyclists were up by 6% in the third quarter of 1981 (compared with the third quarter of 1980) and deaths were up by 10% to 85. These figures deceive to a large extent as there is no good denominator (though the Department of Transport's rough figures suggest that cycle traffic was up by 7% in the same time) and many of the accidents are to those under 15 (100 of the 303 cyclists killed in 1980 were 15 or under). But cycling to work through Central London is still much more dangerous than flying to Los Angeles.

Although advertising campaigns urging motorists to "steer clear of bikes" and telling cyclists to ensure that they can be seen may help, the only way to cut cycling casualties down to almost nothing is to keep cyclists apart from motorists. Milton Keynes has 60 miles of cycle lanes (soon to be 200) and Peterborough has 20 miles, but most local authorities in Britain are doing very little to help cyclists. The Government is encouraging them to do more, but thousands of miles of cycle lanes are what we need. Alternatively, as an article in *New Society* several years ago showed,[1] by closing minor back streets to motor-cars, it is possible to provide through routes even in cities such as London.

Traditionally, a government has always had two main duties towards its citizens: to safeguard their health and to maintain the law. To these, a third should now be added: ensuring the freedom to travel reasonable distances for work or play. The recurrent transport disputes have shown how much the average inhabitant of our cities is at the mercy of any disaffected group of workers. Cycling has the benefits of a personal form of transport that is cheap, non-polluting, non-selfish, and efficient for distances of up to eight miles. The Government should stop its delaying tactics, with its stream of vapid consultative documents, and act to ensure that its citizens can travel safely and freely without hindrance by others.

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