References

1 Fine PR, Kuhlemeier KV, De Vivo MJ, Stover SL. Spinal cord injury: an epidemiological perspective. *Paraplegia* 1980;**17**:237-50.
2 Castanier MM, Garard R, Berard E, Deider C, Bourret J. Epidemiology of spinal cord injury in Rhode-Alpes region, France 1970-75. *Paraplegia* 1979;**16**:76-87.
3 Kurtzke JF. Epidemiology of spinal cord injury. *Exp Neurol* 1975;**48**: 163-236.
4 Krans JF, Franti CE, Riggins RS, Richards D, Bormani NO. Incidence of traumatic spinal cord lesion. *J Chronic Dis* 1975;**28**:471-92.
5 Krans JF. A comparison of recent studies on the extent of the head and spinal cord injury problem in the United States. *J Neurosurg* 1980;**53**: S35-S43.
6 Shrosbree RD. Spinal cord injury as a result of motor cycle accidents. *Paraplegia* 1979;**16**:102-12.
7 Silver JR, Morris WR, Otfinowski JS. Associated injuries in patients with spinal injury. *Injury* 1980;**12**:219-24.
8 Huelke DF, O'Day J, Mendelsohn RA. Cervical injuries suffered in automobile crashes. *J Nuerosurg* 1981;**54**:316-22.
9 Bedbrook GM. Spinal cord injury with tetraplegia and paraplegia. *J Bone Joint Surg (Br)* 1979;**61**:267-84.
10 Ohry A, Brooks ME, Rozin R. Misdiagnosis of spinal cord injuries—the physiatrist's point of view. *Paraplegia* 1980;**18**:15-20.
11 Ravichandran G. Traumatic single facet subluxation without neurological damage of the cervical spine—a new clinical sign. *Archives of Orthopaedic and Traumatic Surgery* 1978;**92**:221-4.
12 Morpurgo CV. *The treatment of patients with multiple injuries associated with damage to the spinal cord. Recent progress in anaesthiology and resuscitation.* Amsterdam: Excerpta Medica, 1975:471-5.
13 Scher AT. Cervical spine injuries without evidence of fracture or dislocation. *S Afr Med J* 1976;**50**:962-5.
14 Scher AT. Unilateral locked facet in cervical spine injuries. *AJR* 1977; **129**:45-8.
15 Scher AT. Unrecognised fractures and dislocations of the cervical spine. *Paraplegia* 1981;**19**:25-30.
16 Penning L. Diagnostic clues by x-rays—injuries of the lower cervical spine. *Acta Neurochir (Wien)* 1970;**22**:234-44.
17 Wholey MH, Bruwer AJ, Baker HL. The lateral roentgenogram of the neck; with comments on atlanto odontoid basin relationship. *Radiology* 1958;**71**:350-6.
18 Whalen JP, Woodruff CL. The cervical prevertebral fat stripe. A new aid in evaluating the cervical prevertebral soft tissue space. *AJR* 1970;**109**: 445-51.
19 Taylor AR, Blackwood W. Paraplegia in cervical injuries with normal radiographic appearance. *J Bone Joint Surg* 1948;**30B**:245-8.
20 Snyder RG. Quoted by Cullen SA in: Mason JK, ed. *The pathology of violent injury.* London: Edward Arnold, 1978.
21 Bothwell PW. The problem of motorcycle accidents. *Practitioner* 1962; **188**:475.
22 Arnold E. In: Mason JK, ed. *The pathology of violent injury.* London: Edward Arnold, 1978:1-30.

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Lesson of the Week

Plasmodium falciparum malaria in Nigerians who live in Britain

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Plasmodium falciparum malaria affected a Nigerian who was studying in Glasgow and returned home for a short holiday. Malaria caused by *P falciparum* is life-threatening and is not often seen in Glasgow; malaria caused by the more benign *P vivax* is ten times more common.[1] In London, however, such a case would not be unusual.[2] Many residents of Glasgow have relatives whom they visit in India and Pakistan, where *P vivax* predominates. In London much of the malaria originates in west-central Africa, where *P falciparum* is widespread.

After making a survey among travellers to the Indian subcontinent,[3] attitudes to malaria prophylaxis were studied among Nigerians resident in Britain. It emerged that this group does not take the precautions that are normally recommended to travellers overseas.[4]

Immunity against malaria declines when constant reinfection stops: re-exposure may then result in serious illness

Case report

A 27-year-old Nigerian student first came to Scotland in 1978. She had had malaria as a child but had had no attacks after the age of 14 years. She returned home for holidays totalling six months over three years, and most recently for three weeks in December 1981. She had taken no antimalarial prophylaxis. Five days after returning to Scotland from her latest holiday she developed headache, anorexia, and fever with shivering. These symptoms persisted for four days and she then had hallucinations and repeated vomiting, which caused her admission to hospital. On examination she had a temperature of 39·5°C and was drowsy. The tip of the spleen could be felt and the edge of her liver was palpable and tender. Blood films showed ring trophozoites of *P falciparum* in about 5% of her red blood cells. Her haemoglobin concentration fell below 10 g/dl, but after treatment with chloroquine she recovered steadily.

General attitude to prophylaxis

Fifty-three Nigerians were interviewed in the Nigerian Airways offices in London. All had been resident in Britain for at least two years, and 45 had had malaria before coming to Britain. They were asked about their most recent trip to Nigeria in the past five years: 36 had taken no antimalarial tablets; 16 had taken tablets, but irregularly, or had discontinued them immediately on returning to Britain. Only one person had taken tablets regularly, including the month after return.

The usual reason given for not taking any tablets was that they preferred to treat attacks when they occurred, and this was linked

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to a belief that they had immunity from previous infection. Several thought that vaccinations given to them had included malaria. Other reasons given included pregnancy, allergy to drugs, "no malaria present," and "precautions not necessary on a short trip." When a general practitioner had been consulted he had advised prophylaxis in all but one instance.

Discussion

Falciparum malaria may cause death rapidly. Prevention is therefore important, usually by taking regular and appropriate antimalarial drugs for the duration of exposure and for one month afterwards.[4] Similar advice is given to those who are likely to be exposed to *P vivax*, *P ovale*, and *P malariae*. This is in part to try to prevent an unpleasant illness and in part because the risk of falciparum malaria cannot be excluded even in parts of the world where it is unlikely to occur. Few people, knowing that it is possible to prevent a potentially lethal illness, would not choose to do so. But many fail to take adequate precautions because they forget, fail to take tablets after leaving a malarious area, or believe that tablets are not necessary for a short trip or after several years of exposure to malaria. Frequently they do not consider the problem at all, and may not get advice either during preparations for travel or on the journey.

People who were born and grew up in malarious areas present particular problems. Children suffering repeated infections usually develop some immunity, though they may die in the process. Adults who are immune rarely become ill after reinfection and consider the disease as a minor inconvenience.[5] As the case history illustrates, however, immunity declines when reinfection stops, leaving a person vulnerable again to serious illness after as little as a year.

Most of the people who were interviewed about prophylaxis planned eventually to return to live permanently in malarious areas, and since they did not receive regular antimalarial prophylaxis before coming to Britain they would not expect to do so in the future. Although immunity does wane when constant re-exposure stops, some immunity may be retained, and malaria contracted on holidays at home may not be as dangerous as when it affects people who have never been exposed. What then should the advice be? The choice appears to lie between taking regular prophylaxis, as advised to people who are not immune, or treating attacks when they occur. Prophylactic drugs have different effects on the various forms of the malarial parasite,[6] and some components of immunity may be boosted when reinfection occurs despite prophylaxis or when clinical attacks only are treated. One policy for people who return to live in Nigeria permanently is to take regular prophylaxis for three months and thereafter only to treat attacks. It is necessary to remember that attacks of this potentially lethal disease must be treated quickly, and the longer the gaps between reinfection the greater the urgency.

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References

1 Walker E, Qayyum A. Malaria in Scotland and its presentation in a Glasgow hospital. *Health Bulletin of the Scottish Home and Health Department* 1981;**39**:281-6.
2 Ellis CJ, Eykyn SJ, Watkins P, Bell M, Geddes AM. Malaria in Birmingham and a London teaching hospital. *Br Med J* 1979;i:385-8.
3 Walker E, Qayyum A. Attitudes to malarial prophylaxis. *Br Med J* 1981; **283**:411-2.
4 Malaria prevention in travellers from the United Kingdom. Reports of meeting convened by the Ross Institute. *Br Med J* 1981;**283**:214-8.
5 Maegraith B. Continued problems of malaria in the tropics. In: Hendrickse RG. *Paediatrics in the tropics*. Chapter 21. London: Oxford University Press, 1981.
6 Bruce-Chwatt LJ. The role of drugs in a malaria program. *Am J Trop Med Hyg* 1972;**21**:731-5.

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What is the latest view about the hazards of taking cannabis?

An Expert Group of the Advisory Council on the Misuse of Drugs has recently reviewed the effects of using cannabis.[1] They stated that it was not possible to reach any incontestable conclusions as to the effects on the human body of the use of cannabis. Much of the research done so far had failed to demonstrate positive and important harmful effects in man attributable solely to using cannabis. In several areas there was evidence to suggest that deleterious effects might result in certain circumstances. There is a continuing need for further research, particularly on the epidemiological characteristics of using cannabis and on the effects of its long-term use by man. There was also evidence to suggest that the therapeutic use of cannabis or of substances derived from it for treating certain medical conditions might, after further research, prove to be beneficial. Anyone concerned about the effects of cannabis on man should read this careful review of the scientific evidence.—THOMAS BEWLEY, consultant psychiatrist, London.

1 Home Office. *Report of the expert group of the advisory council on the misuse of drugs on the effects of cannabis use*. London: Home Office, 1982.

A patient who has had laser treatment for a detached retina asks whether there would be any risk to his sight if he was to play squash. Can he be categorically advised that such activity would not result in a further detachment?

Laser treatment is not commonly used alone in the treatment of retinal detachment. It is more usually used for sealing flat retinal holes or strengthening weak areas of the peripheral retina by promoting adhesion between the neuroretina and the underlying retinal pigment epithelium and choroid, in a process analogous to spot-welding. This is done to prevent detachment rather than to cure it. At operation pieces of silicone sponge are usually sutured to the external surface of the sclera overlying the holes in the retina that have caused the detachment, closing them permanently by indentation of the sclera and application of intense cold, or laser light, to the areas around the holes, again in order to make the retinal and subretinal layers adhere together. At the same time subretinal fluid may need to be drained from the eye, though this potentially hazardous manoeuvre should be avoided whenever possible. Once the retina has become satisfactorily flat and the deliberate scarring around the retinal holes has occurred, it is unusual for redetachment to take place by the same retinal holes reopening. Nevertheless, a further retinal detachment may occur in the same eye if fresh retinal holes develop, and also in the opposite eye, which commonly shares in the same, often familial, peripheral retinal degenerative process. Retinal detachment due to such degeneration is nearly always a potentially bilateral disorder, and not only in highly myopic patients. For that reason, long-term postoperative follow-up with prophylactic treatment of any further degenerative areas that develop is essential. Even when the original detachment resulted from an injury, such as a retinal dialysis after blunt trauma in a young person, there is sometimes evidence of pre-existing retinal weakness which may also be found in the other eye.

In summary, if there is a pre-existing weakness in the peripheral retina, not only the direct injury that a squash ball can cause to the eye (which may be avoided to a great extent by eye-protecting guards) but also the traction forces that the rapid linear and angular accelerations of the game of squash place upon any abnormal adhesions between the peripheral retina and the vitreous body (which cannot be avoided) will tend to lead to retinal tears and detachment. Although after successful retinal detachment surgery the sealed original retinal break is unlikely to reopen, even under these stresses, any other weaknesses present in the same or the other eye may well be worsened, causing a second fresh retinal detachment.—B S HARCOURT, consultant ophthalmic surgeon, Leeds.