Correspondence

The prognostic value of finding schizonts of Plasmodium falciparum in the peripheral blood

The 2 editions of 'Severe and Complicated Malaria' (*Transactions*, 1986, **80**, supplement and 1990, **84**, supplement 2) contain much information of great value. However, there is one concept on p. 14 in the first edition and p. 24 in the second that is overstated: 'The presence of *P. falciparum* schizonts in peripheral blood is also a sign of severity'. This seems to refer to Field & Niven (1937: *Transactions*, **30**, 569–574).

In Field & Shute's informative book (1956: *The Microscopic Diagnosis of Human Malaria - II. A Morphological Study of the Erythrocytic Parasites*. Kuala Lumpur; Studies of the Institute for Medical Research of the Federation of Malaya, no. 24) it was stated that, in Malaysian infections, 'the presence of schizonts in blood films is *usually* [my italics] an indication of clinical severity' (p. 24, paragraph 1). The following section points out that schizonts in blood films from mild infections are by no means rare and, in some parts of the world, are fairly common.

In my article 'Schizogonic forms of *Plasmodium falciparum* in the peripheral blood (1961: *Progress in Protozoology: Proceedings of the First International Congress on Protozoology, Prague, August 22–31, 1961*, pp. 456–463), which is a compilation of reports from 5 continents, it is shown that schizogonic forms in peripheral blood are not rare, occurring in more than 50% of cases.

The use of induced malaria for the treatment of neurosyphilis enabled us to follow, on a daily basis, patients infected with strains of malaria from the USA, Panama, South America and Africa. Schizogonic forms appeared in the peripheral blood early in the primary attack, usually before the maximum parasitaemias when the parasitaemia was very low, and usually persisted for only a few days. They also appeared in relapses.

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Female genital schistosomiasis: a neglected risk factor for the transmission of HIV?

We have recently reported that female genital schistosomiasis (FGS), a frequent manifestation of infection with *Schistosoma haematobium*, could be an important risk factor for the bi-directional transmission of HIV (Feldmeier *et al.*, 1994: *International Journal of STD and AIDS*, 5, 368–372). Our assumption was based on the unique clinical and immunological features that characterize the egg granuloma: chronic lesions frequently located in the vulva, vagina and cervix of afflicted women. As the AIDS epidemic is on the verge of spreading from urban to rural areas (Godfrey-Fausset *et al.*, 1994; *Nature*, **368**, 183–184) and schistosomiasis has entered metropolitan areas (WHO, 1993; *WHO Technical Report Series*, no. 830), the possible association between AIDS and urogenital schistosomiasis should be a matter of concern for those 44 African countries where both diseases coexist.

In an assessment of the epidemiological situation of schistosomiasis haematobium in East Africa we came across data that seem to support an association between FGS and HIV. The highest increase in sero-prevalence of HIV during the last 10 years has been in Uganda, Kenya, Malawi and the Central African Republic, countries where the prevalence of schistosomiasis haematobium is up to 70%. On the Kenyan side of Lake Victoria, an area highly endemic for *S. haematobium*, HIV prevalence is 30%, twice as high as in Nairobi (14%, Valadez, 1993: *IX Conference on* AIDS, *Berlin*, poster C11-2865). In the Mwanza area of Tanzania at the southern end of Lake Victoria (also an endemic area of schistosomiasis haematobium) HIV prevalence in women is 1·2–1·7 times greater than in men; a fact not observed in areas free of schistosomiasis haematobium (Barongo *et al.*, 1992: *AIDS*, **6**, 1521–1528) or other countries (Rwandan HIV Seroprevalence Study Group, 1989: *Lancet*, **8644**, 941–943). In addition, in rural Tanzania and South Africa the highest prevalence of female HIV carriers is found in the 15-24 years age group, decreasing thereafter (Abdool-Karim *et al.*, 1992: *AIDS*, **6**, 1535–1539; Barongo *et al.*, loc. cit.). The distribution of HIV seroprevalence is thus synchronized with the characteristic age-dependent prevalence of schistosomiasis haematobium. For women living in towns and rural settlements, the prevalence of HIV peaks later (25–34 years) (Barongo *et al.*, *loc. cit.*). Finally, in contrast to common sexually transmitted diseases (STDs), clinically apparent lesions due to schistosomiasis are already present when a girl reaches sexual maturity and impair the barrier function of the vaginal epithelium before her first sexual intercourse, whereas lesions due to STDs almost by definition appear later in life (Feldmeier *et al.*, 1993: *Acta Tropica*, **55**, 139–169).

If genital lesions due to schistosomiasis haematobium are indeed a cofactor for the transmission of HIV, it should contribute significantly to the spread of AIDS in those countries where the parasitic disease is endemic. Schistosomiasis, unlike AIDS or STDs, does not carry a social stigma, is curable, and can be controlled at the community level. Thus thorough epidemiological studies are urgently required, not least because confirmation of FGS as a risk-factor for transmission of HIV might help to stem the tide of despair in AIDS-ridden African countries. Moreover, interventions aimed at high risk groups—as long as the prevalence of HIV in the general population remains rather low—may delay, or even prevent, widespread dissemination of HIV infection in the rest of the population (Potts *et al.*, 1991: *Lancet*, **338**, 608–613).

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Abdominal tuberculosis

Hibbs *et al.*, in their article on abdominal tuberculosis in Cairo, described 22 abdominal tuberculosis patients (1994: *Transactions*, **88**, 317–318). In 2 patients the diagnosis of tuberculosis was made by the demonstration of acid-fast bacilli in stools. We do not agree with this as a diagnostic criterion for intestinal tuberculosis. The positive predictive value of acid-fast bacilli in a stool smear for a positive mycobacterial stool culture is low. Moreover, most mycobacteria isolated from stools are non-tuberculous (Colon *et al.*, 1989: *AIDS*, **3**, 539–541; Colebunders *et al.*, 1990: *Annales de la Société Belge de Médecine Tropicale*, **70**, 303–309; Morris *et al.*, 1993: *Journal of Clinical Microbiology*, **31**, 1385–1387). Only a stool culture positive for *Mycobacterium tuberculosis* is indicative of an *M. tuberculosis* infection. However, such infection is not necessarily localized in the abdomen; the

patient may simply have pulmonary tuberculosis with acid-fast bacilli in the stools because he or she swallowed expectorations.

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Abdominal tuberculosis: a reply

In our article on abdominal tuberculosis in Cairo (Hibbs *et al.*, 1994: *Transactions*, **88**, 317–318) diagnosis of tuberculosis was made by demonstration of acid-fast bacilli in stools. Colebunders *et al.*, in the preceding letter, are absolutely correct that this is a poor criterion for diagnosing intestinal tuberculosis. Stool cultures from both of these patients grew *Mycobacterium tuberculosis* and we should have indicated this. I agree that these organisms could have come from swallowed expectorations from pulmonary infection, but we found no evidence of pulmonary infection in these 2 patients. Gastrointestinal abnormalities were found by barium enema, computerized tomography scan and laparotomy in one patient, and barium enema in the other. We thank the authors for pointing out our error.

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Thiacetazone reactions

We read with interest the recent articles on cutaneous reactions to thiacetazone (Nunn *et al.*, 1993: *Transactions*, **87**, 578; Kelly *et al.*, 1994: *Transactions*, **88**, 113).

As part of our studies of leprosy and tuberculosis in the continuing Lepra Evaluation Project/Karonga Prevention Study in northern Malawi, we have been monitoring drug reactions in tuberculosis patients. All tuberculosis patients start their treatment in hospital, with a short course regimen for smear-positive patients and a standard 12 months regimen for smear-negative patients. Both regimens contain thiacetazone, started immediately in the standard regimen and in the out-patient phase in the short course regimen. The re-treatment regimen also includes thiacetazone at the out-patient stage.

In 1993, 244 patients were started on antituberculosis treatment in Karonga District Hospital. Nine smear-positive patients died while in hospital or absconded, so never received thiacetazone. Another 11 patients were transferred from elsewhere to continue out-patient therapy in Karonga district. 246 patients were thus treated wtih thiacetazone in the district during the period under study. Of 216 patients tested, 119 (55%) were HIV positive.

Twenty-six of the 246 patients developed skin rashes. Thiacetazone was thought to be the most likely cause in all but 2 cases. In 5 of these patients, 2 on the short course, 2 on the standard, and one on the re-treatment regimen, the medical officer diagnosed Stevens–Johnson syndrome, defined as a severe bullous form of erythema exudativum multiforme with mucosal involvement. Onset was between 8 and 12 d after starting thiacetazone. Four of the cases were HIV positive adults and one was the one-year-old child of an HIV positive mother; the child and 3 adults died as a direct result, despite stopping all antituberculous drugs and giving intensive supportive therapy (systemic corticosteroid treatment was not used). The other adult died 2 months later, after an initial recovery.

The mortality rate due to Stevens–Johnson syndrome among HIV positives of approximately 3% (4 of $246 \times 0{\cdot}55$) is consistent with that found in other African studies (Nunn *et al.*, 1991: *Lancet*, **337**, 627; Pozniak *et al.*, 1992; *AIDS*, **6**, 809; Kelly *et al.*, *loc. cit.*). Our results strengthen the arguments for finding alternative treatment regimens in areas where a large proportion of tuberculosis patients is HIV positive.

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A lens even farther

In my letter entitled 'A lens too far?' (1993: *Transactions*, **87**, 496), I suggested that the reliability of microscopical diagnosis of malaria, and other tropical diseases, could be improved by the substitution of 7× Huygenian eyepieces for the now standard 10× wide field eyepiece.

Since the letter appeared, I have learned that the major manufacturer of microscopes used in developing countries—Olympus Optical Co., Tokyo, Japan—ceased production of 7× Huygenian eyepieces in 1993.

I am pleased to be able to report that, after strenuous representations, Olympus Optical Co. have agreed to recommence production of the lens in 1995 and will supply it against specific requests. The lens will not be listed in standard sales literature, so it must be specified as a special item when orders are placed with the agents or directly with the manufacturer.

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