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of a *ovale* in sub-Saharan Africa takes place. Thus in the 2 years 1989-90, 69% of all cases of *P. ovale* were imported from West African countries.²

The absolute requirement for visitors to malaria-endemic regions is that appropriate advice should be given about protection against mosquito bites and malaria chemoprophylaxis. Travellers should be made aware of the symptoms of malaria, and general practitioners the possibility of malaria in such a traveller.

We thank Dr D. Warhurst for the DNA analysis.

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Reduction of coma by quinine loading dose in falciparum cerebral malaria

SIR,—Increasing *Plasmodium falciparum* resistance to chloroquine and severity of cerebral malaria have become a major problem in Yaoundé, Cameroon.¹ On the basis of pharmacokinetic indices in cerebral malaria,² a quinine infusion regimen (8 mg of quinine base/kg body weight over 1 h followed by 8 mg of base/kg over 7 h, then 8 mg/kg over 8 h three times daily for 3 days) was randomly allocated to 10 patients; 10 others received a standard regimen used in Cameroon (8 mg of base/kg over 8 h, three times daily for 3 days). Thick blood films were obtained every 3 h for the first 12 h and every 12 h thereafter. They were stained with Giemsa and examined for parasites.

All patients were African. They all had cerebral malaria and fulfilled the definition proposed by Warrell et al³—unrousable coma according to the Glasgow coma scale that was not attributable to other causes of encephalopathy, and *P. falciparum* monospecific

COMPARISON BETWEEN TREATMENT GROUPS ON ADMISSION AND AFTER START OF TREATMENT

	iv quinine		p*
	Loading dose	No loading dose	
Mean age	24.2	22.1	NS
Rectal temperature on admission (°C)	39.8	39.8	NS
Glasgow score on admission	8.6	8.8	NS
Duration of coma before admission (h)	10.0	10.2	NS
Duration of coma after start of treatment (h)	6.8	13.0	0.003
Parasite clearance time (h)	40.8	52.2	0.05

*Non-parametric Mann-Whitney U test corrected for ties.
iv = intravenous, NS = not significant.

parasitaemia (>2000 parasites/ μ l of whole blood).⁴ Both treatment groups were similar with respect to age, body temperature, severity, and duration of coma on admission (table). All patients recovered and were discharged 3 days after admission. No obvious harmful effect was observed. In the loading-dose group, there was a significant decrease in the duration of coma, and the parasite clearance times were reduced by an average of 25% (table).

Davis et al⁵ recommended the use of an intravenous quinine loading dose as being a safe and effective treatment. We are unaware of other reports of controlled trials of the clinical efficacy of a quinine loading dose, and our preliminary findings show its effectiveness and good tolerance. Our results need to be confirmed but they indicate the usefulness of this simple regimen as a treatment for severe malaria, even in places where intensive-care facilities are not available.

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Malaria, bednets, and mortality

SIR,—Dr Velema (Sept 7, p 642) suggests alternative explanations for the large impact on childhood mortality associated with the introduction of insecticide-treated bednets reported by us (June 22, p 1499). He questions the importance of malaria as a cause of death in The Gambia.

Velema accepts that insecticide-treated bednets may have prevented deaths caused by malaria. However, there was a further non-specific reduction in mortality, which we attributed to malaria acting as a risk factor for mortality due to other causes. This can be termed indirect mortality. Velema suggests that, rather than malaria being an indirect cause of death, the non-specific reduction in mortality might have resulted from weekly contact of children with village health workers (VHWs). In support of this he cites a study from Benin¹ in which children seen by a VHW had a lower risk of dying in the next 6 months than children not seen. This non-specific protective effect of contact with a VHW could not be related to any particular curative or preventive measure. It may be that unprompted contact with health services is a marker of health awareness and of a protective health-conscious attitude, rather than a protective measure per se.

In The Gambia, VHWs have few resources to exercise any direct curative action: their role in preventive medicine and health education is negligible. As we reported in our paper, the comparison of mortality rates in villages with and without the presence of a simple, village-based primary health care programme suggests that this scheme does not significantly reduce child mortality in The Gambia. A similar conclusion was drawn in a previous study.² We feel that the most likely explanation for the large reduction in

M 18