resistance in Zimbabwe¹ and quinine infusion for severe malaria cases in areas of resistance to the 4 aminoquinolines has been recommended.²

The pathophysiology of hypoglycaemia complicating cerebral malaria was fully discussed in my paper and nowhere did I implicate chloroquine as a cause or co-factor of the hypoglycaemia in this patient. Indeed, as Dr Ramos Filho and his colleagues point out, chloroquine has not been known to cause hyperinsulinaemia in malaria.³

Finally, I fully agree with the assertion that asexual parasitaemia in a patient with impaired consciousness does not seal the diagnosis of cerebral malaria.⁴ This could not be more true than here in Africa where we have a tremendous degree of experience with this clinical presentation. Other conditions have to be excluded, and this was done in this case. We were even fortunate to have an autopsy (rare in this part of the world) which did not reveal any other cause of coma or death. Obviously, only essential data were given in the case report.

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Myocardial hypertrophy, fibrosis and infarction following exposure of the heart to radiation for Hodgkin's disease.

Sir,
The histopathological illustration accompanying our recent communication on cardiac disease following radiation for Hodgkin's disease (*Postgraduate Medical Journal*, 1986 **62**, 1055–1058), although submitted in the conventional positive form was printed as a negative giving an appearance similar to that of an X-ray film. It is interesting to observe histopathology illustrated in the manner of diagnostic imaging and to recall that the distinction made in optics between object and image has its parallel in the diagnostic sciences where the

pathological disciplines study actual tissue (object) while the radiological sciences study images. This distinction may have, in the past, given rise to the idea that pathology was based on objectivity and radiology on imagination but recent improvements in the quality and variety of diagnostic imaging techniques have blurred the distinction. However, illustrating pathology by the image reversal conventions of radiology does not convey any additional information so that we must take a negative view of this development in histopathological imaging.

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Effects of the opiate antagonist naloxone on learning and memory in patients with multi-infarct dementia.

Sir,

The endogenous opioids have recently been implicated in learning and memory mechanisms in both animals¹ and humans,² and naloxone was reported to improve memory functions in a subgroup of patients with senile dementia of the Alzheimer's type.² We thus tested the effects of acute naloxone administration in 8 drug-free patients (aged 60–81 years) who met DSM-III criteria for multi-infarct dementia (MID) with Hachinski scores greater than 7 and with Mini-Mental States scores of 14–26. In all subjects diagnosis was supported by characteristic head computed tomographic (CT) findings. Subjects were tested twice off and on 1.2 mg i.m. of naloxone, on selected scales of the Wechsler Memory Scale (WMS) and on 12 item list learning task using the Bushke-Fuld Selective Reminding Procedure. The order of the drug and drug-free testing was counterbalanced. No significant ($P < 0.05$) effects were found on the WMS and on the list learning task following 30 and 60 minutes respectively, of naloxone administration.

These findings suggest that administration of opiate receptor antagonists may not be useful in the management of memory and learning functions in MID patients, and are consistent with recent reports demonstrating significant correlation between the severity of dementia and reduced CSF beta-endorphin levels in MID patients.³

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