Indeed, there have now been more trials reported with these drugs than with any other antimalarial therapy. In contrast, reversible neurological side effects, including extrapyramidal and psychiatric syndromes and tremor, have been very well documented after treatment with chloroquine [3, 8–14]. Because chloroquine is known to cause adverse neurological events in humans and artesunate is not, we suggest that the patient described by Franco-Paredes et al. [1] was most likely suffering from the adverse effects of excessive chloroquine.

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Reply to Newton et al.

Sir—We thank Drs. Newton, Day, and White [1] for the stimulating and valid criticisms of our recently published letter [2]. We believe that the main contribution of our letter is that it adds to the growing literature on the misdiagnosis of malaria, something we frequently encounter in our practices. We described a patient who presented to our clinic with a neuropsychiatric syndrome. His signs and symptoms were not totally compatible with an extrapyramidal syndrome or a cerebellar syndrome, and we attributed them to neurotoxicity due to antimalarial therapy. He had been misdiagnosed with malaria multiple times [2]. We recognize that chloroquine may rarely produce a neuropsychiatric syndrome or cerebellar dysfunction during the treatment of acute malaria [3–5]. Nevertheless, it should be noted that most of the neurological symptoms described by our patient (inability to concentrate, severe anxiety, and lack of coordination) occurred while the patient was receiving monotherapy with artesunate only, and he clearly worsened during the last course of dual therapy with artesunate and chloroquine. Therefore, chloroquine neurotoxicity may have contributed to the expression of some of the signs and symptoms, and we acknowledged this possibility in our report [2].

We are familiar with the important value of artemisinin-based combination therapies in the global control and treatment of malaria; and, more importantly, we are familiar with their overall safety profile, because several million patients have been treated with these compounds during the past decades. However, it would be hasty to attribute the patient’s problems just to chloroquine, given the multiple, prolonged, and unnecessary courses of oral artesunate he received, compared with the currently recommended regimens [3, 6]. Furthermore, in support of our suggestion that orally administered artesunate may be associated with clinical neurotoxicity in humans, a previous report by Miller and Panosian [7] described a syndrome of cerebellar dysfunction that was temporally associated with the oral administration of artesunate in a patient who had returned from West Africa. In this particular case, the patient apparently took oral artesunate for only 4 days, while our patient took 5 complete 10-day courses of artesunate [7]. Other reports have also suggested the potential neurotoxicity of artesunate [5, 8]. Sodium artesunate and dihydroartemisin, metabolites common to all artemisin analogues, can induce neurotoxicity in different neuronal culture types in vitro [9].

In summary, based on previous evidence that suggests a potential neurotoxic effect of artesunate in humans [5, 7, 8] and the fact that some of the patient’s neurological symptoms began prior to receiving chloroquine, we remain concerned about—and cannot rule out—the possibility of the contribution of repetitive and
prolonged courses of oral artesunate to our patient’s neurological syndrome.

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Medical Treatment of Fish Bone–Related Liver Abscess

Sir—Cases of hepatic abscess due to fish bone penetration are rare and may be fatal. Nisbet et al. [1] reported a case of a fish bone penetrating from the pylorus of the stomach to the liver and causing persistent Streptococcus anginosus infection. Surviving patients described in previous reports were all surgically treated. In this report, we describe a case of liver abscess caused by fish bone penetration in which medical treatment was successful.

A 40-year-old man with a history of hypertension and hyperlipidemia, which had been controlled with medical treatment for 3 years, was admitted to the hospital because of intermittent chills and concurrent fever. Symptoms had progressively worsened. At admission, the patient reported vague epigastralgia, and he was febrile (temperature, 41.9°C) and had tachycardia (heart rate, 139 beats/min) and hypertension (blood pressure, 195/71 mm Hg). The patient’s abdomen was soft, but it was tender over...