Microorganisms accumulated in these cracks and might have contaminated cleaning equipment even when it had been disinfected. A plastic hose helped reduce the problem. Flushing the hose for several minutes before filling cleaning equipment also controlled the risk of dissemination. Both *P aeruginosa* and *Enterobacter cloacae* were recovered from the inner areas of the rubber hose.

Hospital infection-control procedures may have to take into account sources of microorganisms that are often overlooked.

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False chloroquine resistance in Africa

SIR—Chloroquine is still the drug of choice for the treatment of uncomplicated falciparum malaria in most of sub-Saharan Africa. But the increasing number of therapeutic failures with chloroquine has led to concern about whether this drug can remain effective for long in Africa. Before deciding to change the drug policy on the first-line antimalarial treatment, the extent of true drug resistance related to drug quality must be investigated. In fact, the quantity of counterfeit or underdosed antimalarial drugs in Africa is unknown. Only a few representatives of health authorities are willing to acknowledge this problem, despite the concerns raised by previous correspondents.[1,2]

Two personal observations suggest the reality of counterfeit or underdosed chloroquine. First, during the screening of patients for enrolment in a clinical study, we have encountered patients with symptoms and positive blood smears who claimed to have self-medicated with chloroquine a few days before the consultation, and who had a negative urine test.[3] These patients invariably purchased chloroquine from unofficial sources, such as street vendors and grocery stores. The most common reasons given for this practice include lower cost, accessibility even at odd hours, and the choice to buy a few tablets instead of the entire packed tablets in a pharmacy. Second, we were asked to investigate the quality of the tablets because a batch of chloroquine tablets ordered through a local supplier suggested tampering with the bottle label. Analysis by high-performance liquid chromatography at Rhône-Poulenc-Rorer in France showed that, despite the characteristic N (for Nivaquine) engraved on the tablets, each tablet contained only 2·5–18 mg chloroquine phosphate. The "real" Nivaquine contains 136 mg chloroquine sulphate per tablet, equivalent to 100 mg chloroquine base.

The high cost of specialist drugs imported from well-known European pharmaceutical companies and the economic difficulties of most Africans have encouraged the importation of cheaper drugs, some of which are underdosed. Because prescription and provision of drugs are not strictly regulated in many African countries, these cheaper drugs are often sold by unofficial vendors. Poor-quality chloroquine not only leads to severe and complicated malaria and death, but also can select resistant parasites in underdosed patients,[3] and lead to an increase in false-resistant cases. These effects tend to discredit chloroquine, despite its high cost-benefit ratio, in the eyes of local consumers. National and international regulations to regulate and control the unacceptable practice of producing, exporting, and importing low-quality or false drugs are urgently needed. Western pharmaceutical manufacturers should reconsider their questionable marketing practice to overprice their drugs in less developed countries.

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Tuberculosis in elephants

SIR—David Frankel's June 7 news item (p 1675)[1] on the discovery of tuberculosis in two captive Asian elephants in the USA is not, in fact, surprising. Before this report, eight elephants had died from tuberculosis in the USA, four of them before 1941.[2] There has also been one such death in the UK, in 1875.[3] These animals were all Asian elephants, except for one case in an African elephant.[4]

In the past few years, US zoos and circuses have tested captive animals for tuberculosis. 171 animals in 40 institutions were tested with avian Old tuberculin, avian purified protein derivative (PPD), bovine PPD, mammalian old tuberculin, and the serum ELISA test.[2] About 33% of animals reacted to one or more of the skin tests and 11% were positive with the ELISA test procedure. Unfortunately, none of these results can be regarded as accurate, because there is no standard international procedure for testing these animals, or indeed for the interpretation of the results. We do not even know if they are one of the mammalian species, like the orang utan, that requires special procedures for tuberculosis screening. Some of these elephants, when given serial skin tests, changed from positive to negative, then back to positive. There are no confirmed reports of human beings becoming infected as a result of elephant contact.

The situation in Myanmar (formerly, Burma) is potentially much more serious for both species. This country still employs about 4000–6000 trained elephants, each with its own rider, or mahout, for timber extraction. One imagines that a similar number of people have direct contact with these elephants, and that possibly several tens of thousands of people have indirect contact with them on a daily basis.

Tuberculosis in Burmese elephants is difficult to diagnose because of limited resources, equipment, and trained veterinary personnel. The prevalence of tuberculosis in these animals is unknown.[4] The prevalance must be high compared with other elephant populations because of their constant exposure to human beings, and domestic farm animals. Affected elephants tend to suffer chronic weight loss, but that alone is a poor guide to diagnosis since the elephants can also suffer heavily from parasitism.

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DEPARTMENT OF ERROR

Safety guidelines for use of nitric oxide—In this letter by L Foubert and colleagues (*Lancet* 1992; **339**: 1615–16) the rate constant ($1{\cdot}93\times10^{-18}$ cm^6 mol^{-2} s^{-1} at 300°K) was incorrect. It should have read $1{\cdot}93\times10^{-38}$ cm^6 molecule^{-2} s^{-1} at 300°K.

Defective homocysteine metabolism as a risk factor for diabetic retinopathy—In this Research letter by S Neugebauer and colleagues (Feb 15, p 473), there were two errors in the table. The serum creatinine values are in µmol/L and the SD value of the HbA_{1c} in diabetic patients with retinopathy is 1·3, not 13.

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