SIR,—In a letter sent to all doctors and dentists in the United Kingdom on 3 January 1974 the medical assessor for the Committee on Safety of Medicines draws attention to 130 reports concerning jaundice following the use of halothane for anaesthesia. These reports had been volunteered to the committee under its usual reporting procedure for adverse reactions to drugs. He states that the committee accepts that the findings of the analysis of these reports published by Dr. W. H. W. Inman and Professor W. W. Mushin (5 January, p. 5) "add support to the previous published evidence that jaundice may on occasion follow the administration of halothane." He goes on to point out that the report by Dr. Inman and Professor Mushin "shows that . . . multiple exposure to halothane carries a relatively greater risk than single exposure," the risk being apparently dependent on the interval between administrations of halothane.

This belief may well prove to be correct, but before it is accepted and current clinical anaesthetic practice changed—an action of enormous consequence for anaesthesia and for health—the grounds upon which it is based need full examination. We are of the opinion that the evidence reported so far does not support the belief.

Dr. Inman and Professor Mushin note that the reports to the committee do not attribute a causal connexion with any anaesthetic agents other than halothane and that the reports are highly selected. Moreover they state that there is fairly general agreement that out of several possible factors multiple exposure to halothane has been a common one. In support of their conclusions they cite the paper by Professor Mushin and his colleagues¹ in which the risk of jaundice was estimated to be between 1 in 6,000 and 1 in 20,000 among patients exposed to halothane more than once within a four-week period. This incidence was calculated from a survey of patients anaesthetized in Cardiff. Out of 123,516 patients given a general anaesthetic, two came to postmortem examination with hepatic necrosis attributed to anaesthesia, one of these having had two halothane anaesthetics and one having had two non-halothane anaesthetics. On the basis of the fact that 70% of patients had halothane and an estimate that 7% of these had two exposures within 28 days (based on a local survey of 558 consecutive patients coming to surgery), from the occurrence of this single case of hepatic necrosis they calculated the incidence to be 1 in 6,000 (70% of 123,516=86,461; 7% of 86,461=6,052). Calculating from their figures the incidence of hepatic necrosis from two non-halothane anaesthetics on exactly the same basis we find that the risk is 1 in 2,594. One would have to conclude that patients would be almost three times less likely to develop hepatic necrosis with repeated administration of halothane within 28 days than of other anaesthetics. This estimate may well be correct and we contend that it is just as probable because there is no statistical sense in either case.

In view of the bias inherent in the reports to the Committee on Safety of Medicines we propose that to obtain a valid conclusion requires a very large prospective study. With an estimated incidence of jaundice following halothane anaesthesia repeated within

four weeks of between 1 in 6,000 and 1 in 20,000 this would require the prospective study of 2,000,000 randomly allocated anaesthetics to satisfy the null hypothesis, a formidable undertaking. This is of the order of studying all patients anaesthetized in one year in N.H.S. hospitals. Until this has been done we respectfully suggest that the assessor's letter is needlessly alarmist.—We are, etc.,

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¹ Mushin, W. W., Rosen, M., and Jones, E. V.,
British Medical Journal, 1971, 3, 18.

Chronic Brucellosis

SIR,—Your interesting leading article (23 February, p. 299) correctly says that treatment of this condition may be difficult. In my part of the world the disease is not rare and creates much disability and suffering. Accordingly I sought advice on treatment from a former colleague in the Public Health Laboratory Service whose experience of the problem was much greater than the usual. He advised two 170-mg capsules of clomocycline four times a day for not less than six weeks. In a single, long-suffering patient the benefit was dramatic, including relief of signs and symptoms and a fall in antibody titre.

One successful case is little enough evidence, I agree; but so few doctors seem to realize the need for substantial dosage and a relatively long course of treatment that you may think it worth presenting the details.—I am, etc.,

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Colour Television Hazard

SIR,—A recent tragedy in this town has prompted me to write in the hope of publicizing a potentially lethal hazard of the colour television set, which is becoming part of the family in Britain.

Four children were admitted to this hospital late one evening from a home in which a colour television set had gone up in smoke. The youngest two were brought in dead (ages 1 and 2 years). The third (aged 4 years) was in acute respiratory distress and the fourth (aged 7 years) was moribund. This last child died after two days in the intensive care unit on a respirator (she had fixed dilated pupils and apnoea on admission). All had been sleeping soundly in bed. The father was working a night shift; the mother had gone into the neighbouring house for a while, leaving the television set switched on. She was later summoned to her house to find it filled with copious noxious fumes.

A few evenings after this terrible event a colour television set in the residents' mess of this hospital burst into smoke, when a doctor had sufficient presence of mind to disconnect the set from the mains supply. It would seem that there may be some defect in the design or construction of colour television sets as similar episodes have been reported from other areas, though I have not heard of loss of life occurring elsewhere.

It seems an urgent situation which de-

mands that all parents be warned about the danger of leaving a colour (or black and white?) television set connected to the mains supply and then leaving children unattended in the home.—I am, etc.,

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Death during Dental Anaesthesia

SIR,—In your medicolegal column (2 February, p. 207) you report the inquest on a recent death during dental anaesthesia at which the cardiac arrest was attributed to asphyxia. May I put on record my reasons for doubting the validity of this explanation?

Four days after the cardiac arrest, when the patient was still alive though decerebrate (he died next day), the dentist gave me verbally a detailed account of the case. He was quite definite that the patient, who was "nervous and palish," was seated semi-upright at a tilt of 45°. While the extraction was being attempted, after induction with methohexitone, there was movement of the head, but not of the limbs. He gave six breaths of nitrous oxide (without oxygen) and then withdrew the mask, after which breathing continued normally and there was no further movement. Nothing amiss was noticed until suddenly, within three minutes of needling the vein for induction, the patient collapsed with cardiac arrest.

A week after this talk with the dentist and two weeks before the inquest Professor Sir Robert Macintosh and I visited him in his surgery; we also talked to his chairside assistant. The dentist confirmed the sequence of events he had already described to me and was certain that the cardiac arrest occurred within three minutes of needling the vein. He also confirmed that during this interval nothing amiss was noticed, either by himself or by his chairside assistant. The latter knew the patient, who was formerly her employer, and was struck by his pallor as he entered the surgery. Demonstrating with a subject in the chair, she estimated the patient's tilt as a good deal more upright than 45°.

I attended the inquest and have a transcript of the proceedings in front of me. Referring to the patient's colour, the dentist said that the face was pale throughout. He gave the position of the chair as between 50° and 55° from the vertical, but later put it at "something in the order of 50°", indicating with his arm a tilt of about 45°.

Reviewing the case, I think it unlikely that so brief an administration of nitrous oxide would deepen anaesthesia appreciably or produce much anoxia; and both would be quickly reversed by the succeeding breaths of air. In a young healthy person, such as the patient, asphyxia does not progress to the point of cardiac arrest either insidiously or with such speed; it does so only when it is severe and prolonged, with signs so gross—indeed so terrifying—that not even a child could fail to notice them and appreciate their seriousness.

Sir Robert and I concluded that, of the two possible causes of the cardiac arrest, asphyxia was the less likely one. We thought it probable that the patient fainted after the prick of the needle and then continued to be kept head up, the faint remaining un-