Correspondence

Sudden death due to myocardial tuberculosis

Sir,—A great number of patients with tuberculosis have been treated in our clinic since its foundation in 1954, and before the period of antituberculous chemotherapy numerous patients died at the advanced stage of their disease. In the past few years six patients have died in our clinic of miliary tuberculosis. Our necropsy reports include the microscopic findings in the heart. In only one case did the microscopic examination reveal myocardial involvement by granulomas with central caseation. All six patients were cachectic and seriously ill, and died of toxic cardiac failure three to five days after being admitted to the clinic.

For several years we have been treating the problem of cardiac sarcoidosis. In this context cases of sudden death in apparently healthy young people whose necropsies show sarcoidosis are always of interest. With this in mind we should like to comment on the contribution by Dr PJW Wallis and others (February 1984;39:155–6). This report deals with the sudden death of a 31 year old well nourished man during absolute wellbeing at work. Acid fast bacilli were identified only in the caseating material of one enlarged mediastinal lymph node. No mention was made of cultural identification. The microscopic examination of the myocardium revealed granulomas without typical central caseation. In our opinion the clinical course and the localisation of the granulomas (lung, liver, kidneys, heart) indicate systemic sarcoidosis with myocardial involvement. The tuberculoid findings in the mediastinal lymph node could be related to an additional infection due to contact with a sister with tuberculosis.

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The cause of death was miliary tuberculosis with myocardial involvement.

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Comparative trial of two non-sedative H1, antihistamines, terfenadine and astemizole, for hay fever

Sir,—Dr BJ Freedman (May 1985;40:399) suggests that the favourable response to astemizole compared with that to terfenadine in the maintenance therapy of hay fever reported by Drs PH Howarth and ST Holgate (September 1984;39:668–72) may be an effect of dose.

A more likely explanation relates to study duration. Tachyphylaxis to competitive H1 antagonists is well known and organ specific. It is clear, for example, that tolerance to the sedative effects of classical antihistamines often develops within a few days and the same may well be true of nasal histamine blockade. Clinical tolerance to antihistamines has been well described and long recognised.1

More recently Krause and Shuster in a clinical urticaria study2 showed that "The displacement of the weak response curve was maximal at 2 weeks with chlorphenamine and somewhat less at 4 weeks. This is similar to that previously found with terfenadine and suggests tolerance. By contrast astemizole showed an even greater effect at 4 than at 2 weeks."

It is important when we are discussing antihistamine therapy to distinguish between short term symptomatic therapy, where a non-sedative competitive antagonist may have an important role, and longer term maintenance therapy. In the latter situation astemizole has not so far been shown to cause tachyphylaxis. Resistance to tachyphylaxis is almost certainly due in some way to its extremely slow dissociation from H1 receptors.3

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