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 tuberculous 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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**This letter was sent to the authors, who reply below.

Thank you for your letter enclosing the comment by Drs Kirsten and Schaedel. We, like Drs Kirsten and Schaedel, initially thought that this young man had died as a result of either sarcoid heart disease or giant cell myocarditis. However, neither of these conditions can be confidently diagnosed until other granulomatous diseases are excluded. The finding of typical tuberculous disease elsewhere strongly suggested that the granulomatous infiltration of the heart was also tuberculous in origin. The absence of caseation and our failure to demonstrate tubercle bacilli within the myocardium does not preclude a diagnosis of tuberculous myocarditis. Indeed, previous reports of this disease have often failed to identify tubercle bacilli in spite of typical histological changes in the myocardium (1, 2). Furthermore, central caseation is often absent in the early stages of development of miliary tubercles (3).

Although definitive proof, provided by the culture of tubercle bacilli, is lacking in our case we contend nevertheless that the cause of death was miliary tuberculosis with myocardial involvement.

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1 Wilbur EL. Myocardial Tuberculosis: A Case of Congestive Cardiac Failure. *Am Rev Tuberc* 1938;**38**:769–76.
2 Schnitzer R. Myocardial Tuberculosis with Paroxysmal Ventricular Tachycardia. *Br Heart J* 1947;**9**:213–19.
3 Payling Wright G, Heard BE. In: Systemic Pathology, 2nd Edition, Vol 1. Symmers W. St C, ed. London: Churchill Livingstone, 1976:336.

Comparative trial of two non-sedative H_1 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 H_1 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 study[2] showed that "The displacement of the weal response curve was maximal at 2 weeks with chlorpheniramine 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 H_1 receptors.[3]

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1 Dannenberg TB, Feinberg SM. The development of tolerance to antihistamines. *J Allergy* 1951;**22**:330–9.
2 Krause LB, Shuster S. A comparison of astemizole and chlorpheniramine in dermographic urticaria. *Br J Dermatol* 1985;**112**:447–53.
3 Laduron ??, *et al.* In vitro and in vivo binding characteristics of a new long-acting histamine H_1 antagonist, astemizole. *Mol Pharmacol* 1982;**21**:294–300.