The third child, a 2 year old who had had recurrent chest symptoms for several months, was referred for surgery for a possible bronchogenic or duplication cyst diagnosed on computed tomography. Surgery was cancelled when a preoperative Mantoux test (1 U) yielded a positive result. None of the patients had a history of contact with someone with tuberculosis. All were given standard antituberculous treatment and progressed well.

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- 1 Clark JE, Cant AJ. Pitfalls in contact tracing and early diagnosis of childhood tuberculosis. *BMJ* 1996;313:221-2. (27 July.)

Failing regimens should be modified with multiple drugs instituted simultaneously

EDITOR,—Julia E Clark and Andrew J Cant emphasise the importance of pyrazinamide for mycobacterial killing.¹ Though this is pertinent, they do the drug a disservice by adding it alone to a failing treatment regimen.

The cornerstone of the treatment of *Mycobacterium tuberculosis* is the use of multiple agents to decrease the number of viable bacteria rapidly and to prevent the emergence of drug resistance.² Although the child in the authors' case 2 was probably infected with a fully sensitive organism, her regimen seemed to be failing after one month. The chances of the organism developing resistance to both agents in the absence of poor compliance with treatment is admittedly low, but the worsening clinical situation should have raised the suspicion of drug resistance. The addition of a single agent in the face of suspected drug resistance and extensive disease is equivalent to treating the patient with only one antibiotic and may rapidly lead to further resistance. This is the commonest prescribing error contributing to the acquisition of drug resistance in *M tuberculosis*.³

Guidelines on the management of *M tuberculosis* in both Britain⁴ and the United States⁵ emphasise the need to treat proved drug resistant *M tuberculosis*, or *M tuberculosis* that has been contracted in areas of high drug resistance, with three or four active agents. The guidelines do not, however, mention the need to modify failing regimens with multiple drugs instituted simultaneously, to prevent the emergence of drug resistance. Although drug resistant *M tuberculosis* is uncommon in Britain, it will become a greater public health threat unless there is a greater awareness both of the problem and of how to combat its spread.

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- 1 Clarke JE, Cant AJ. Pitfalls in contact tracing and early diagnosis of childhood tuberculosis. *BMJ* 1996;313:221-2. (27 July.)
- 2 Van Scoy RE, Wilkowske CJ. Antituberculous agents. *Mayo Clin Proc* 1992;67:179-87.
- 3 Mahmoudi A, Isaman MD. Pitfalls in the care of patients with tuberculosis. *JAMA* 1993;270:65-8.
- 4 Chemotherapy and management of tuberculosis in the United Kingdom: recommendations of the Joint Tuberculosis Committee of the British Thoracic Society. *Thorax* 1990;45:403-8.
- 5 Initial therapy for tuberculosis in the era of multidrug resistance: recommendations of the Advisory Council for the Elimination of Tuberculosis. *MMWR* 1993;42:No RR7.

Authors' reply

EDITOR,—We agree with J R Greig and colleagues that, when drug resistance is suspected, more than one antituberculous drug should usually be added to treatment. This has been our practice when we have met this problem. We considered drug resistance to be most unlikely in our case 2 as the index case was known to be infected with fully sensitive *Mycobacterium tuberculosis* and there had been no concerns about compliance. Furthermore as Greig and colleagues admit, resistance to two antituberculous drugs after only a few weeks' treatment would be most unusual. Clinical examination, radiology, and bronchoscopy showed that enlarged hilar lymph nodes obstructing bronchi were responsible for the lobar collapse. It is well recognised that nodes may increase in size after the start of antituberculous treatment² as dead and dying tuberculous bacilli provoke a greater inflammatory response. This leads to increased bronchial compression and lobar collapse and can be reversed by the administration of steroids, which were probably the most important change in treatment. Pyrazinamide was added because it kills organisms rapidly, making the treatment regimen shorter and more effective.

This case illustrates the importance of clinical evaluation of children with tuberculosis and the use of concomitant anti-inflammatory treatment when tuberculous nodes are compressing important structures.

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- 1 Miller FJW, Seal RME, Taylor MD. *Tuberculosis in children*. London: Churchill, 1963:246-7.

Ethnic differences in aortic aneurysm and peripheral vascular disease

Authors' data are inadequate

EDITOR,—Racial differences in risk factors for vascular diseases are well known. It would be no surprise if the prevalences of different vascular diseases and the incidences with which they present showed racial variations, but it is difficult to accept that the data presented by Gregory Y H Lip and D Gareth Beevers provide evidence to support this.¹ Indeed, the second paragraph of their letter suggests that they are using incidence data to provide information about prevalence in different races while maintaining that the mode of presentation may also vary among races, which means that incidence would not reflect prevalence in different racial groups.

The authors examined the admission diagnoses of patients in their hospital with "aortic aneurysm" and "peripheral vascular disease" between 1976 and 1986. Why did they examine such dated records? And surely discharge diagnoses would be more accurate than admission diagnoses. The disease categories are not clearly defined. Does "aortic aneurysm" include both thoracic and abdominal aneurysms, symptomatic cases and asymptomatic incidental findings, dissections and ruptures, medically and surgically treated cases, and elective and emergency surgery? If all groups are included then the total number of patients with aortic aneurysms admitted in the nine, 10, or 11 year period (depending on the unspecified cut offs they used) is similar to the number of cases admitted in a single year to many smaller district general hospitals in Britain. Does this indicate

a problem with ascertainment? What is meant by "peripheral vascular disease"? This could vary from asymptomatic absence of pulses to peripheral gangrene. Were definitions of disease constant throughout the study period?

The authors also seem to have had some difficulty in defining the race of those admitted, since the number of patients whose race was unspecified or unknown is greater than the combined totals of patients in the black and Asian groups. Even if the data were accepted as representative, would they provide us with insight into racial differences in the prevalences or incidences of these diseases? We know that vascular diseases are related to age and that the age distributions of white, black, and Asian populations differ. The authors' failure to allow for the confounding effects of different age distributions in the racial groups may have affected the analysis.

There are so many problems with the data presented in this letter that one must ask whether it is time that letters purporting to present original observations should be peer reviewed in the same way as full papers are.

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- 1 Lip GYH, Beevers DG. Epidemiology of aortic aneurysm and peripheral vascular disease may show ethnic differences. *BMJ* 1996;313:173. (20 July.)

Authors' reply

EDITOR,—Peter Wilmshurst fails to appreciate that the aim of our letter was simply to highlight the ethnic differences in vascular disease, which David Coggon and colleagues did not consider in their analysis of the contrasting epidemiology of aortic aneurysm and peripheral vascular disease in England and Wales.¹ We chose to illustrate this point by describing briefly the ethnic distribution of our hospital patients admitted with abdominal aortic aneurysms and peripheral vascular disease. We accept that such data are crude and open to many biases and confounders, but our intention was not to provide a complete treatise on the epidemiology of the disease but merely to make a point.

We chose to examine the computerised hospital admission diagnoses between 1976 and 1986 because those dates approximated to the period studied by Coggon and colleagues.¹ The diagnoses were based on the hospital activity analysis recordings and are the same as the diagnostic categories of the *International Classification of Diseases* that Coggon and colleagues used. There are limitations to the use of hospital activity analysis, which in most hospitals does not record ethnicity.

Detailed studies of ethnicity are difficult to conduct and would require precise definitions, especially for the different subgroups of Asians from the Indian subcontinent (Punjabi, Gujarati, Hindu, Bengali, etc) in Britain. Only a detailed prospective longitudinal study of a defined population would provide precise data on ethnic differences in the prevalence or incidence of diseases. Our letter was merely intended to reinforce Coggon and colleagues' statement that "there are other causes for aortic aneurysm beside the well established risk factors for atherosclerosis" and to suggest that the influence of ethnic factors should be included in any future analyses.

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- 1 Coggon D, Winter P, Martyn C, Inskip H. Contrasting epidemiology of aortic aneurysm and peripheral vascular disease in England and Wales. *BMJ* 1996;312:948. (13 April.)