in those dying of cardiac causes compared with those who survived (0.11 (0.07) and 0.05 (0.02) $ms^{1/2}$ respectively, P<0.001). However, heart rates were similar in the two groups (66 (12) beats/minute in survivors and 66 (13) beats/minute in those who died).

Although there are methodological problems with qualitative measurement of QTc dispersion, previous work has shown that variation in interlead measurements far outweigh inaccuracies introduced by any other factor, including interobserver and intraobserver variation.[2] In our study the intraobserver and interobserver variability expressed in absolute values was of the order of 11 $ms^{1/2}$ and 16 $ms^{1/2}$ respectively, suggesting that the differences in QTc dispersion between patient groups cannot be explained by variability in measurement. In fact, we reported coefficients of variation on page 875 of our article.

The study by Agha W Haider and Saba Naz showing reduced QTc dispersion in patients with preinfarction angina warrants further investigation. Although preinfarction angina might confer some early benefit in hospital, it tends to be associated with more recurrent ischaemia and a worse longer term outcome.[3] A recent study showing increased QT dispersion in diabetic patients with autonomic dysfunction[4] suggests that measurement of QT dispersion in this group of patients may be a cheap and valuable tool for detecting patients at increased risk of sudden cardiac death, as suggested by György Jermendy.

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Introduction of the computer assisted prescribing scheme Prodigy was premature

EDITOR,—Jacqui Wise writes about claims that have been made since an interim report was published on the computer assisted prescribing project Prodigy.[1 2] We are disappointed by the lack of statistical evidence in the report and thus by its lack of scientific integrity.

Prodigy, which was used by 137 practices, was evaluated over the period December 1995 to February 1996.[2] In response to the question "How much would you want to continue with Prodigy?" 14 of the 86 respondents indicated that they would continue to use it, 32 indicated that they would continue if it was improved somewhat, 33 indicated that they would continue only if improvements were considerable, five selected the vague option "some time in the future," and two chose "never again." As opinions were solicited during the expected honeymoon period of the new system, these responses are downbeat.

Surprisingly, the results are reported as "confirming desirability" of the system to general practitioners. The author of the report also claims that the system's "effectiveness" is confirmed by the fact that prescribing costs (adjudged from the net ingredient cost per prescribing unit) for Prodigy sites rose by 4.8%, compared with 5.9% for all other practices. This is reported as a "relative reduction in the rise of expenditure of 1.1%." Firstly, this is an absolute reduction and not a relative one. Secondly, there is no indication of the variance of this prescribing indicator for the groups compared; indeed, no statistical analysis is reported. The author lists a large number of evaluation methods, including a 10% poll of general practitioners by questionnaire, so considerable resources have been spent. If no reliable and statistically robust conclusions can be drawn then these resources have been wasted.

Perhaps it was misinformation that led the health minister, Gerald Malone, to make the specious claim that "Prodigy research has broken through frontiers in computer based support for general practice."[1]

Decision support in therapeutics is one of the most important areas for research into, and development of, clinical knowledge systems. In our opinion, the Prodigy software was thrust prematurely into general practice systems as an active appendage to support decision making. This can alienate users by operating for some of the time as an "uninvited guest" in the clinical decision making process. No computer can reliably predict what each user does not know; thus complementary active and passive systems to support general practitioners' knowledge should be developed. In the case of Prodigy, we believe that political initiatives have been misguided and that medicine has lost an opportunity to gather reliable evidence in computer assisted prescribing.

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Shortage of psychiatric beds exists in Staffordshire too

EDITOR,—Doris Hollander and colleagues highlight the considerable overoccupancy of acute psychiatric beds in Greater London and suggest that similar studies be made in other parts of Britain.[1] Our clinical experience in North Staffordshire suggested a similar problem. To assess the effect of changes in local services and, specifically, the effect of provision of non-acute beds[2] on the use of acute beds we retrospectively investigated bed occupancy between 1987 and 1993. North Staffordshire's population of 460 000 is served by 94 acute psychiatric beds (0.2 beds per 1000 population), and the Jarman underprivileged area scores range from −33.26 to 38.37. Bed occupancy for 1987-93 was derived from Körner returns. The reliability of this information source for identifying admissions was ascertained by comparison of the data with manual ward registers, case notes, and registers required under the Mental Health Act. Körner data were found to record at least 99.5% of all admissions.

Bed occupancy was less than 100% in only one year (1991) and reached 130.5% in 1988 (table 1). No significant trend could be shown for either bed occupancy or length of stay. The methodology used is more likely to have underestimated than overestimated occupancy. In addition, the number of extracontractual referrals could not be reliably assessed.

Table 1—*Bed occupancy and mean length of stay for acute psychiatric beds in North Staffordshire. Figures in parentheses are 95% confidence intervals*

Year	Bed occupancy (%)	Mean length of stay (days)
1987	115.2 (85.0 to 145.3)	50.0 (44.4 to 55.6)
1988	130.5 (127.0 to 134.0)	38.3 (34.7 to 42.0)
1989	101.0 (90.5 to 111.2)	35.2 (31.7 to 38.7)
1990	103.7 (100.0 to 107.6)	33.1 (30.3 to 35.8)
1991	99.5 (95.8 to 103.2)	37.0 (33.3 to 40.7)
1992	114.7 (110.3 to 119.2)	39.0 (35.6 to 41.9)
1993	103.2 (96.5 to 109.9)	31.3 (28.9 to 33.7)

These figures suggest that North Staffordshire has a longstanding shortage of acute psychiatric beds. Whether recent innovations in the provision of services will have an impact on bed occupancy remains to be seen.

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1 Hollander D, Powell R, Tobiansky R. Bed occupancy in psychiatric units in Greater London is 113%. *BMJ* 1996;313:166. (20 July.)
2 Audit Commission. *Finding a place. A review of mental health services for adults*. London: HMSO, 1995.

Early diagnosis of childhood tuberculosis

High index of suspicion is needed

EDITOR,—In addition to highlighting the pitfalls of contact tracing in childhood tuberculosis, Julia E Clark and Andrew J Cant raise the important issue of diagnostic difficulty in this age group.[1] With the rising incidence of tuberculosis in Britain, a high index of suspicion must be maintained, especially in patients with unusual clinical features. In the past six months three white patients presenting to our department have been investigated extensively before the diagnosis of tuberculosis was confirmed.

The first patient was a 4 year old girl who presented with a seven week history of cough and left upper lobe consolidation in a chest radiograph, which persisted despite treatment with oral antibiotics. She underwent bronchoscopy three months later, when an endobronchial tumour was found in the left main bronchus; computed tomography showed this to be entirely intraluminal. Bronchoscopic biopsy of the tumour showed caseating granulomas and acid fast bacilli. A Mantoux test (10 U) subsequently yielded a strongly positive result.

The second patient was a 3 year old boy referred from his local hospital with a three week history of increasing stridor. A chest radiograph and computed tomogram showed a large paramediastinal mass suggestive of a bronchogenic cyst. At thoracotomy the mass was found to be an enlarged lymph node containing acid fast bacilli. A preoperative Mantoux test (10 U) had yielded a negative result.

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 Britain[4] and the United States[5] 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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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 treatment[2] 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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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.)