absorptiometry (DEXA) in corticosteroid associated osteoporosis. We reported to the summer 1991 meeting of the British Thoracic Society[1] our experience of 19 patients (13 female) with corticosteroid dependent asthma. Twelve of these patients had sustained at least one vertebral compression fracture. The mean corticosteroid dose and treatment duration for this fracture group was 10·1 mg prednisolone/day for 13·1 years and 13·9 mg prednisolone/day for 9·6 years in those patients without fracture (NS). The mean L2-L4 density was significantly lower in those patients with fractures (0·797 g/cm^2 versus 1·143 g/cm^2, $p < 0·0001$). The mean neck of femur density was also significantly lower in those patients with fractures (0·718 g/cm^2 versus 0·901 g/cm^2, $p < 0·005$). The study by Dr Luengo and colleagues used dual photon densitometry, which is known to be a less precise measure than DEXA,[2] and this may explain why in their study, though spinal density was higher in asthmatic patients without fractures, the difference was not statistically significant. It would appear therefore that a single measure of L2-L4 density by DEXA is a useful screening test for corticosteroid associated osteoporosis.

Finally, in their discussion the authors ignore the effect of the menopause on the changes described. There is significant evidence that premenopausal women, who (though it is not stated) probably make up most of "group 1," are protected from corticosteroid associated osteoporosis, presumably by the effects of oestrogen.[3]

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2 Holbrook TL, Barrett-Connor E, Klauber M, Sartoris D. A population-based comparison of quantitative dual-energy *x*-ray absorptiometry with dual-photon absorptiometry of the spine and hip. *Calc Tiss Int* 1991;**49**:305–7.
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AUTHORS' REPLY We thank Dr Gallacher and colleagues for his interest in our article comparing bone mineral mass as a predictor for vertebral fractures in steroid dependent asthma. In our study we found that the mean L2-4 density was lower in steroid dependent patients with vertebral fractures than in those without fractures. The difference, however, did not reach statistical significance. In contrast, they found that the values obtained by bone densitometry were significantly lower in steroid dependent asthmatic patients with fractures than in patients without fractures.

Unfortunately, neither their letter nor the abstract of their work offers information about important aspects such as the age of subjects and the selection of patients for their study. We studied 99 consecutive steroid dependent asthmatic patients and detected vertebral fractures in 32 (prevalence 32%) while they investigated 19 patients, 63% of whom suffered fractures. This very high prevalence of vertebral fractures suggests that selection bias operated in the inclusion of patients in their study. We also suspect that in this study patients with fractures were much older than subjects without fractures and thus comparison of absolute values of bone density between the two groups could be misleading.

We agree that the menopause should be considered as an independent factor in patients having continuous steroid treatment. But the suggestion that the bone of premenopausal women is "protected" against the deleterious effect of steroids cannot be substantiated by the study of Boyce *et al* (their reference 3) because this did not include a control group of asthmatic patients not having steroid treatment. On the other hand, it has been found that steroid treatment decreases osteocalcin concentrations in both premenopausal and menopausal women. As osteocalcin is a marker of bone formation this finding also argues against the hypothesis of Dr Gallacher and his colleagues.

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Bronchial compression as a result of lung herniation after pneumonectomy

We read with interest the paper of Dr K F Whyte and others (November 1991;**46**:855–7) concerning a 40 year old housewife with a right pneumonectomy syndrome. A thoracotomy to attempt a corrective procedure was unsuccessful because of right apical pleural adhesions and the patient remained severely disabled.

We have seen five patients with the right pneumonectomy syndrome in the past two years, seven months to 13 years after right pneumonectomy. All have been treated with the implantation of an expandable mammary prosthesis into the right hemithorax to correct the position of the mediastinum and the left lung. Two patients are the subject of a report that will be published.[1]

All five patients experienced an immediate and important reduction of dyspnoea. Pulmonary function improved and bronchoscopy showed a considerable decrease of bronchial compression in all patients. The improvement was sustained in four patients; in the other patient, who had the pneumonectomy 13 years previously, a cicatricial hernia occurred at the site of the implantation. A Marlex Mesh patch has been attached to the thoracic wall, and a new expandable prosthesis will be implanted.

We believe that implantation of an expandable mammary prosthesis is the treatment of choice in the right pneumonectomy syndrome.

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1 Janssen JP, Brutel de la Rivière A, Carpentier Alting MP, Westermann CJJ, Bergstein PGM, Duurkens VAM. Post-pneumonectomy syndrome in adulthood: surgical correction using an expandable prosthesis. *Chest* (in press).

School microepidemic of tuberculosis

We read with interest the recent paper by Dr Bredin and colleagues (December 1991;**46**: 922–3). A recent outbreak of tuberculosis in Leeds also infected principally school age children.[1]

In 1988 a 23 year old woman from a poor, predominantly white community within the city, designated by the council as an area of deprivation, presented with smear positive pulmonary tuberculosis. Screening of contacts revealed 10 with pulmonary tuberculosis, eight of whom were children (age range 4–8 years). All but two of the children and one adult were asymptomatic; in six cases the place of contact was the school or community centre. A further 10 children had unexplained grade 2 or 3 positive Heaf test responses and received chemoprophylaxis with isoniazid and rifampicin. None of those infected had received neonatal BCG immunisation. This outbreak demonstrates that, despite the declining incidence of tuberculosis, poor indigenous communities in England remain at risk of tuberculosis. Poor nutrition and housing probably contributed to this outbreak; socioeconomic details were not provided by Dr Bredin and colleagues but such factors may have played a part in the size of their microepidemic. None of our subjects treated with chemoprophylaxis have developed tuberculosis so far; this contrasts with the Cork epidemic, where the brother of the probable index patient developed tuberculosis following chemoprophylaxis, possibly because of his heavy exposure.

Dr Bredin and colleagues also noted that, of 324 children who had received neonatal BCG, 63% had a grade 1 or 2 Heaf response, compared with 85% of 262 children who had not received neonatal BCG; but they were unable to determine the protective effect of neonatal BCG because the number of active tuberculosis cases was too small for statistical comparison. We have recently examined the effects of our neonatal BCG immunisation programme, which is offered to all Asian babies, on the Heaf status of schoolchildren routinely tested at the age of 12–13 years.[2] Of 5013 non-Asian children tested, 8% had a Heaf test response of grade 2 or more, compared with 47% of 366 Asian children tested. On the basis of current Department of Health guidelines, 8% of non-Asian and 75% of Asian children had appropriate immunity, avoiding the need for repeat BCG immunisation. The high prevalence of grade 1 and 2 Heaf responses in Cork, and the surprising observation that rates were higher in children who had not received infant BCG immunisation, presumably reflects a higher prevalence of tuberculosis in the Irish community. We believe that, as suggested in a recent editorial in the *British Medical Journal*,[3] neonatal BCG immunisation should be offered routinely in areas of social deprivation in the United Kingdom.

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