ourselves,[1] as have others.[2 3] We are uncertain of the reasons but the withdrawal of sympathetic activity and concomitant redistribution of potassium would seem the most likely possibility. We have also consistently observed a lengthening of the QS2 interval with rest, consistent with sympathetic withdrawal.

As discussed in our paper a positive chronotropic response may be associated with $beta_2$ and $beta_1$ stimulation, but a positive inotropic response with an increase in systolic blood pressure is due to $beta_1$ stimulation.[4] We observed a significantly greater positive inotropic response and increase in systolic blood pressure with fenoterol than with salbutamol.

We agree with the authors that at recommended doses there is no difference between the beta selectivity of fenoterol and salbutamol. As we discussed in our paper, however, patients may not adhere to the recommended doses during severe attacks of asthma and we feel justified in examining the effects of higher doses of these agents.

The pharmacokinetics have not been discussed because they are not relevant to this study. We wished to examine the extrapulmonary effects of these agents in normal volunteers, in doses that might be used in the clinical setting by patients suffering acute attacks of asthma. Our interest was with the pharmacodynamics, not kinetics. We agree with the authors that isoprenaline will be rapidly metabolised locally and that fenoterol will accumulate—indeed, we specifically pointed this out in our paper.

We have not attempted to extrapolate directly to chronic asthma but have simply observed significant differences in normal volunteers that may have relevance to asthmatic patients.

The authors do, however, raise an extremely important point regarding the lower recommended doses of fenoterol compared with salbutamol in Sweden. The recommended doses are similar in New Zealand for salbutamol and fenoterol. Fenoterol is dispensed by metered dose inhaler as 200 μg/puff compared with salbutamol 100 μg/puff; the common use of two puffs at each treatment means that in practice patients regularly use a dose of fenoterol that is twice that of salbutamol. Furthermore, there are other international differences in the concentrations of fenoterol nebuliser solutions. The standard solution in New Zealand contains 5 mg/ml of fenoterol while in Canada the solution contains 1 mg/ml. The reasons for these international differences require closer examination and explanation.

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Adrenal function in patients with active tuberculosis

SIR,—I read with interest the study of metabolic changes in tuberculosis, reported by Dr DJ Barnes and colleagues (May 1989;44:422–4), but their findings in respect to serum sodium changes in a Melanesian population differ from observations on English patients.

These workers found that 41% of their patients with tuberculosis were hyponatraemic. In a series of 125 patients with tuberculosis I found that only 24% had abnormally low serum sodium concentrations, with a lowest value of 126 mmol/l. The entire series tended to have a low sodium level with a mean value of 135·9 (SD 4·34) mmol/l.

Dr Barnes and colleagues failed to identify adrenal dysfunction as a common problem in patients with active tuberculosis in the tropics, and postulated that salt depletion or vasopressin excess might be alternative explanations for hyponatraemia. The discrepancy between these two populations points to salt depletion in the tropics as the additional factor.

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AUTHORS' REPLY We would like to thank Dr Mustchin for his interest in our paper. His English patients certainly had a lower incidence of hyponatraemia than our Melanesian population (24% versus 41%; $p < 0{\cdot}02$, χ^2 test). We would agree that the most likely cause for our higher prevalence of hyponatraemia is salt depletion. Mild hyponatraemia is a common finding on presentation in febrile illness in the tropics. It is our impression that it is just as common in pneumonia, malaria, etc, as it is in tuberculosis; but this has not been formally studied. Ideally, we would have liked to have measured the serum and urine osmolalities in our patients, but this was not logistically possible.

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