

Measuring changes on just one axis will considerably underestimate the effect of an intervention on the burden of illness. Future research should collect serial data on severity to estimate changes in "total illness."<sup>2</sup>

Secondly, for trials of a new intervention the control is usually nothing (placebo). But for trials of antibiotics for acute respiratory infections the established treatment is already antibiotics. These then are trials of "no antibiotics" (the "new" intervention) against "antibiotics." Therefore patients recruited are likely to be the least ill. Re-examining the trials in a Cochrane review<sup>3</sup> we could extract this "non-recruitment because the child was too ill" out of the total recruited from only two of the seven trials (52/232 and 27/240). We need a greater understanding of how selection of patients for trials may affect the interpretation and application of results.

We also have surprisingly little information about alternative treatments. With spontaneously remitting

illnesses such as acute otitis media, killing bacteria ("cure") has no advantage over palliating the symptoms.<sup>2</sup> We know that antihistamines and decongestants contribute modest, if any, benefit.<sup>4</sup> But which is the best analgesic? Is anything else helpful? Innovative emerging treatments, such as using benign commensals to overwhelm pathological bacteria, may ultimately prove the most effective treatment for acute otitis media<sup>5</sup> and make the current debate over antibiotic use redundant.

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## Prescribing of lipid lowering drugs to South Asian patients: ecological study

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Coronary heart disease is the major cause of morbidity and mortality in the South Asian population in the United Kingdom, and its incidence is higher than in the white population.<sup>1</sup> This excess risk seems to be determined by a combination of metabolic factors leading to the insulin resistance syndrome, psychosocial factors, and established risk factors.<sup>2</sup> Ten out of 15 coronary risk factors measured were reported to be higher in South Asian patients than in their European counterparts, and several of these factors are believed to reflect relative deprivation. South Asian people are also at risk owing to high triglyceride concentrations and low concentrations of high density lipoprotein cholesterol. Although substantial evidence shows the value of lowering cholesterol in people at risk, studies have shown that many patients are not receiving appropriate treatment.<sup>3</sup> We investigated the relation between ethnicity and prescribing of lipid lowering drugs.

### Methods and results

We approached all general practices in one health authority to obtain consent to use their prescribing analyses and cost data for 1996-7. Sixty two (63.9%) of 97 practices gave consent. We obtained the following information for each practice from the health authority: proportion of South Asian patients in the nested age bands 35-69, 40-69, 45-69, 50-69, and 55-69, identified by using name based analysis software (Nam Pehchan)<sup>4</sup>; whether single handed or group practice; proportion of general practitioners of South Asian origin; fundholding status (particularly relevant at the time); Jarman index (surrogate measure for practice workload) for the practice's council ward; and Townsend score (measure of deprivation) for the ward. Comparative analyses of these

demographic factors for each practice showed that consenting and non-consenting practices did not differ significantly (table).

We determined the number of defined daily doses of all lipid lowering drugs prescribed per 1000 South Asian patients in each nested age band for each consenting practice. We used multiple regression analysis (backward and forward selection techniques) to explore the relation between the number of defined daily doses prescribed per 1000 patients (aged 35 to 69) and the practice characteristics. Because of non-linearity and heteroscedasticity of the residual errors, we reanalysed the data after logarithmic transformation of the response variable. We identified two practices as extreme cases (as defined by SPSS) and excluded them from the analysis.

The median number of defined daily doses per 1000 patients was 4775 (interquartile range 2592 to 7336). Owing to strong correlation, we analysed Townsend score and Jarman index separately. The table shows the factors ranked in order of importance for predicting volume of prescribing, with Townsend score included. The parsimonious model includes only the percentage of South Asian patients and deprivation of the practice ward. The negative regression coefficients indicate reduction of prescribing levels with increasing numbers of South Asian patients and levels of deprivation. The results were not significantly changed by use of the various nested age bands or by replacement of Townsend score with Jarman index.

### Comment

Patients in practices with a greater South Asian population are less likely to be prescribed lipid lowering

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## General practice characteristics and results of regression analysis

Rank order	Factor	Consenting (n=62)	Non-consenting (n=35)	P value	R <sup>2</sup> (n=60)	Regressing log (DDD/1000) against all five factors (forward selection)*	
						Coefficient (95% CI)	Standardised
1	% South Asian patients (median (interquartile range))	4.14 (0.99 to 35.70)	5.39 (0.88 to 55.31)	0.738†	0.496	-0.00490 (-0.00686 to -0.00294)	-0.567
2	Townsend score (median (interquartile range))	1.14 (-0.97 to 4.51)	1.31 (-2.89 to 4.92)	0.910†	0.537	-0.0183 (-0.03398 to -0.00262)	-0.246
3	Fundholding practices (No (%))	28 (45)	12 (34)	0.406‡	0.556	No further factor identified as significant	
4	Single handed general practitioners (No (%))	16 (26)	15 (43)	0.133‡	0.559	—	—
5	% South Asian general practitioners (mean (SD))	31.41 (43.71)	—	—	0.560	—	—

DDD=defined daily dose.

\*Constant=3.778 (95% CI 3.717 to 3.839).

†Mann-Whitney U test.

‡ $\chi^2$  analysis (adjusted using Yates's correction).

drugs. This may be surprising, given the higher cardiovascular morbidity and mortality among South Asian people in the United Kingdom<sup>1</sup> and a possible need for lipid lowering treatment that is equal to, if not greater than, that for the white population.<sup>2</sup> Although this type of analysis does not show a causal link between ethnicity, deprivation, and the prescribing of lipid lowering drugs, the identified trend may demand explanation. Further analysis is needed to ascertain the effects of subsequent prescribing guidelines and recent government strategies promoting the use of lipid lowering drugs.<sup>5</sup> Given the limitations of an ecological study, a standardised assessment is needed to determine the extent of unmet need and risk profiles at the level of the individual patient.

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## The mysterious Weber's test

I had suddenly become deaf in one ear. The first thing to pass through the mind of a neurologist who has suddenly become deaf is the possibility of a nasty tumour at the cerebellopontine angle. Close on the heels of this unwanted thought, however, was the much more reassuring realisation that this was almost certainly a recurrence of an ongoing problem with build-up of wax that I had just let go too long. There was one quick way to make sure. Many neurological tests—such as those assessing power or tone—are essentially impossible to perform on oneself. Eliciting your own reflexes is just about possible, but only if you are ambidextrous with a tendon hammer and supple enough to kneel on a chair while reaching round to tap your Achilles tendon. Simple tests of hearing, however, are easily performed. So when I arrived home, I took a tuning fork out of my bag and performed Rinne's and Weber's tests.

Both are quite simple. Sound is normally conducted to the inner ear more easily through air than bone. In Rinne's test you strike the tuning fork and hold it against the outcrop of bone immediately behind the ear until you can't hear it anymore. Then you put the tuning fork in the air about an inch from the earlobe, at which point, under normal circumstances, the tone can be heard again. I heard nothing, suggesting that there was indeed something (wax was the usual suspect) blocking the free passage of sound waves from the outer to the inner ear.

Then I did Weber's test. For this you strike the tuning fork and put it down on the middle of the forehead. In

patients with nerve deafness on one side, the sound is heard better on the normal side. So far, so sensible. But in patients—and indeed in neurologists—with wax blocking their ears, the sound is heard better in the blocked side. If you have a tuning fork you can demonstrate this simply by blocking one ear with your finger.

Why should this be? There should be no difference between the intensity of the sound waves reaching each cochlea via the bones of the skull. The good ear, in addition, should be getting additional sound via air conduction. So surely the sound should be louder in the normal ear?

I have yet to find anybody—ear, nose, and throat surgeon or neurologist—who can give a convincing answer to my question. If anyone can enlighten me, I would be delighted.

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We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.