patients with renal disease and hypertension, not the incidence of renal disease in subjects with raised countertransport (which is not known).[8,9] It cannot be concluded, therefore, that patients with essential hypertension and raised countertransport are at increased risk of nephropathy.

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AUTHORS' REPLY,—We appreciate the comments made by Dr P A Rutherford and colleagues concerning our study. In the penultimate paragraph of our discussion we commented on the question of arterial blood pressure. Although actual blood pressure was similar in the two groups of parents, more parents of patients with proteinuria were being treated for hypertension. We believe that this does not contradict earlier reports but we recognise that the relation between blood pressure and sodium-lithium countertransport is complex.

The overactivity of sodium-lithium countertransport is believed to represent the expression of a susceptibility rather than a disease gene[1]; thus arterial hypertension must result from the interaction of different factors, both environmental and genetic, with abnormalities of countertransport. Our original finding of higher blood pressure in the parents of diabetic patients with proteinuria was obtained in a population studied in the early 1950s, when treatment of hypertension was not readily available. The introduction of pharmacological or other interventions in recent years (for example, diet, exercise, increased awareness of risk, etc) may also have contributed to blur this relation.

We showed a difference in sodium-lithium countertransport activity not only when mid-parental values were used but also when all individual parental values were analysed. Moreover, we used all values to calculate proportions below and above the median value and to postulate modes of inheritance. Maximum velocity in our paper relates to velocity measured at physiological external sodium concentration, which, we are aware, is not saturating.

The p value for the correlation of countertransport between parents and patients was 0·033 by Pearson correlation and 0·006 by Spearman's. We apologise for the typing error. This correlation remained significant even when the outlier value was removed (Pearson's $r=0·36$, $p<0·024$; Spearman's $r=0·40$, $p<0·012$). The association between hyperlipidaemia and countertransport does not imply a cause and effect relation, and we thought that excluding hyperlipidaemia by history was adequate in this study.

We agree that in the general population roughly half of sodium-lithium countertransport is attributable to genetic factors and simply stated that in certain subgroups this could be as high as 80-90%. We accept that the higher prevalence of raised countertransport in patients with renal disease and hypertension does not imply that hypertensive patients with high countertransport are at greater risk of renal and cardiac complications. Strong evidence for this is emerging, however, from several recent studies that show an increased renal and cardiovascular risk in hypertensive patients with high countertransport.[2,3]

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## Early HIV infection

SIR,—The peripheral blood CD4+ cell counts quoted in the editorial by Dr Ann Marie Swart and colleagues as risk markers of progressive HIV disease are wrong by a factor of 10.[1] The critical CD4+ cell counts quoted by Volberding *et al* are 200/mm³ and 500/mm³,[2] which convert to $0·2\times10^9$/l and $0·5\times10^9$/l respectively (not $2\times10^9$/l and $5\times10^9$/l as stated in the editorial).

This confusion arises presumably because the authors, as do most doctors, use counts per cubic millimetre in everyday medical language, whereas the SI convention adopted by the *BMJ* and many automated cell counters refers to cells in thousands of millions per litre. The attempt to standardise cell counts in this cumbersome way has been sustained for long enough. Perhaps "$\times10^9$/l" should be given the same treatment as the SI unit for blood pressure, the kilopascal.

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**Sorry—ED, *BMJ*.

## *Drug Points*

### Colitis associated with ibuprofen

Drs D CLEMENTS and G T WILLIAMS, and Professor J RHODES (University Hospital of Wales, Cardiff CF4 4XW) write: Proctitis due to indomethacin suppositories is well recognised.[1] Oral non-steroidal anti-inflammatory drugs have been implicated in exacerbations of colitis, but they have been thought to be the cause in surprisingly few cases.[2,7] The fenamates, mefenamic acid and flufenamic acid, are well recognised as causing diarrhoea, and colitis associated with their usage has been reported.[3-6] Patients who develop colitis when taking mefenamic acid may tolerate other nonsteroidal anti-inflammatory drugs without a recurrence of diarrhoea or colitis. Two cases of proctitis associated with ibuprofen and naproxen have been reported.[3] We report on a patient who developed colitis after taking ibuprofen.

A 63 year old man was admitted as an emergency with diarrhoea, urgency, and rectal bleeding. He had had no gastrointestinal symptoms until eight weeks before admission, when he had started taking ibuprofen (400 mg three times a day) for back pain. Shortly after starting this his diarrhoea began and was associated with urgency. He was passing watery stools with blood 15-20 times a day. He had some left iliac fossa pain before opening his bowels. He had no family history of inflammatory bowel disease and had had no contact with anyone else with diarrhoea. He had given up smoking seven years previously, his general health was otherwise good, and he had not lost weight.

On examination he was well with no fever or tachycardia. Abdominal examination gave normal results. There was no perianal disease, and sigmoidoscopy confirmed a symmetrical proctitis. A rectal biopsy specimen showed diffuse moderate acute-on-chronic inflammation of the large bowel mucosa. Neutrophil polymorphs invaded the crypt epithelium, and there were occasional crypt abscesses. No granulomas, lymphoid aggregates, or parasites were present, and there was no appreciable architectural disturbance. A full blood count and biochemical profile gave normal results, and the plain abdominal film showed faecal loading of the right and proximal transverse colon, while the descending colon appeared oedematous. Three stool cultures were obtained but no pathogens were isolated.

Ibuprofen was withdrawn, and because of his severe symptoms he was started on both oral and topical steroids, with mesalazine 400 mg three times a day. Within two days his motions were normal and he was discharged home. Three weeks after discharge he had maintained his improvement and steroids were withdrawn. Three months after presentation sigmoidoscopy and rectal biopsy gave normal results. He remained well and asymptomatic after 12 months without receiving all treatment. Barium enema examination, sigmoidoscopy, and rectal biopsy gave normal results. He has not been rechallenged with ibuprofen.

This case shows that non-steroidal anti-inflammatory drugs other than the fenamates can cause colitis. Ibuprofen is now available without prescription, and patients may not volunteer its usage; the association could therefore pass unrecognised. In the only other report of colitis associated with ibuprofen histological examination was unavailable; in our case there was complete histological resolution. There were no clinical or histological features suggestive of Crohn's disease or specific infection. Non-steroidal anti-inflammatory drugs should be considered in all patients presenting with colitis.[8]

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