LETTERS TO THE EDITOR

Sir,

We would like to comment on the letter by Davis, Diggory and Seward[1] as we feel that their results are biased and misleading. Acetazolamide is a drug with potentially fatal metabolic consequences.[2,3] The flaw in their study is the exclusion of patients who may already have sustained a fatal arrhythmia, or who have previously been hospitalised with side-effects requiring cessation of therapy.

Biochemical monitoring is used to screen for two serious consequences of acetazolamide therapy: renal failure[4,5] and metabolic acidosis.[5] Exacerbation of mild renal failure is indicated by an increase in creatinine (or even less sensitively urea). Metabolic acidosis is indicated by a reduction in bicarbonate. It has previously been shown that serum sodium and potassium are not significantly altered by acetazolamide therapy.[6] Acetazolamide produces a hyperchloraemic acidosis with an impairment in H^+ ion secretion and a consequent loss of K^+ ions. Hypokalaemia is a late manifestation of acetazolamide toxicity which should not occur if patients have their metabolic state regularly assessed, with a view to cessation of their medication should they become significantly acidotic. Some patients benefit from having concurrent administration of supplemental alkaline therapy without a reduction in drug efficacy.[7]

In approximately 40% of 2000 patients given a trial of long-term carbonic anhydrase inhibitor therapy the drug had to be discontinued because of side effects.[7] In patients with commonly encountered problems, such as mild renal impairment, chronic respiratory disease or diabetes, or in those taking aspirin, the metabolic acidosis induced may have serious[4,5,8] or fatal consequences.[2,3]

We recommend that patients on long-term acetazolamide continue to have regular measurements of creatinine and electrolytes (in particular bicarbonate), with a view to discontinuing therapy should there be any suggestion of incipient renal failure or significant metabolic acidosis.

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Sir,

We are sorry that Mr Barnes finds our study results biased and misleading. We were not looking at short-term electrolyte disturbance, but were interested only in elderly patients who had been receiving long-term acetazolamide (i.e. for >6 months). Few ophthalmology departments routinely measure such patients' electrolytes when they attend clinic and our study was aimed at answering the question as to whether or not this was acceptable practice. Patients who had sustained a fatal arrhythmia or had had their therapy withdrawn because of earlier side effects were not included. We found no significant difference in potassium, sodium and urea between the patients using acetazolamide and an age- and sex-matched control group who were not using acetazolamide.

Mr Barnes' argument, based mainly on case reports, would seem to suggest that we should routinely measure bicarbonate to screen for the development of acidosis. Most hospitals, including the Chelsea and Westminster, no longer offer routine measurement of venous bicarbonate. This is because

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