of these children are also using sodium cromoglycate spincaps as a preventive agent and therefore require two inhalation devices to be carried with them for administration of their drugs. I would like to point out that it is now possible to simplify their treatment by using the new sodium cromoglycate aerosol. The canister from this device fits the "spacer," and both drugs can be given by the same mechanism.

Children find it very convenient to take both drugs by the same inhaler, and I feel that this improvement might be of benefit to some elderly patients who also have difficulty in co-ordination. Many children prefer the rimiterol "autohaler" which they find easier to use, but, unlike the "spacer," this device has the disadvantage of not being able to accept the sodium cromoglycate canister.

Another practical tip not raised in the article by Dr Jones is the use of slow-release theophylline beads. Slow-release tablets are often unacceptable because the tablets are too large for small children and crushing destroys the sustained-release mechanism. The beads contained in a gyrocap (Rona Laboratoties Ltd) are given on a spoon with jam and provided they are not chewed the sustained-release mechanism remains intact.

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Minor orthopaedic problems in children

SIR,—We were interested to read the timely article on minor orthopaedic problems in children by Mr J A Fixsen and Dr H B Valman (12 September, p 715).

The section on knock knees referred to the common practice of measuring intermalleolar separation. We would like to point out the limited value of this practice. Knock knees are a consequence of the tibiofemoral angle. It is therefore misleading to measure intermalleolar separation, which depends on two variables—the tibiofemoral angle and tibial length. If one ignores tibial growth, serial measurements of intermalleolar separation may give the impression that the situation has deteriorated when in reality the tibiofemoral angle has remained constant.

If it is necessary to measure knock knees we would recommend that the tibiofemoral angle be measured radiologically.[1] Fortunately, in practice such attempted precision is seldom required.

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[1] Salenius P, Vanka E. *J Bone Joint Surg* 1975;**57A**:259.

Reversible renal damage due to glue sniffing

SIR,—I read with interest the report by Drs A M Will and E H McLaren (22 August, p 525) on reversible renal damage due to glue sniffing and would like to raise the following point. A life-threatening renal complication of toluene sniffing, to which the authors did not refer, is the induction of renal tubular acidification defects, which appear to be of the type I distal tubular variety, leading to metabolic acidosis, inappropriately alkaline urine, hypokalaemia, and hyperchloraemia. Most cases reported presented with profound muscle weakness, apparently reversible with abstinence from glue sniffing.[1-4]

The mechanism by which toluene affects the capacity of the renal tubular epithelium in the distal nephron to secrete hydrogen ion is unknown, but it may be due to alteration in membrane permeability or inhibition of intracellular metabolic processes involved in hydrogen ion excretion. Recurrent urinary calculi associated with toluene sniffing were recently described.[5]

It is now clear that control of the sale of products containing toluene is urgently required. Physicians should be aware of the various "syndromes" related to toluene abuse and of the availability of a toluene assay to establish a firm diagnosis.

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[1] Taher SM, Anderson RJ, McCartney R, Popovtzer MM, Schrier RW. *N Engl J Med* 1974;**290**:765-8.
[2] Fischman CM, Oster JR. *JAMA* 1979;**241**:1713-5.
[3] Bennett RH, Forman HR. *Arch Neurol* 1980;**37**:673.
[4] Streicher HZ, Gabolo PA, Moss AH, Kaehny WD. *Ann Intern Med* 1981;**94**:758-62.
[5] Kroeger RM, Moore RJ, Lehman TH, Giesy JD, Skeeters CE. *J Urol* 1980;**123**:89-91.

Audit in renal failure

SIR,—With reference to the paper "Death from chronic renal failure under the age of 50" by the Medical Services Study Group of the Royal College of Physicians (25 July, p 283), as well as the accompanying leading article (p 261) and ensuing correspondence (22 August, p 555 and 12 September, p 726), we wish to point out that treatment facilities have greatly expanded in the Mersey Region since 1979. In the last two years the undersigned members of staff at the regional renal unit have only rarely had to deny dialysis treatment to patients under the age of 60 who would have benefited.

Nevertheless, there have still been times when central facilities have been severely overstretched; and we have grave doubts about whether this improved record can be sustained, unless the number of successful transplant operations increases or funds are made available for further expansion of dialysis. We are already having to provide a proportion of our development from charitable sources, both for capital and for running costs.

Facilities will still be inadequate to treat more than a fraction of patients over the age of 60, many of whom could benefit. However, we are not convinced that *unlimited* availability of dialytic treatment for all age groups, such as exists in certain other developed countries, is desirable.

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Vitamin A toxicity and hypercalcaemia in chronic renal failure

SIR,—In their study on vitamin A metabolism in 38 patients on regular haemodialysis Dr K Farrington and others (20 June, p 1999) found in those taking vitamin A supplements that hypercalcaemic patients had significantly higher vitamin A concentrations than normocalcaemic patients ($p < 0.005$) and that withdrawal of vitamin A supplements caused a significant fall in plasma calcium concentrations ($p < 0.01$) and in plasma alkaline phosphatase concentrations ($p < 0.01$) in seven patients. But serum vitamin A did not correlate with alkaline phosphatase or immunoreactive parathyroid hormone concentrations.

We recently studied 37 patients on regular haemodialysis (results presented at International Congress of Nephrology, 1981),[1] all of whom were receiving daily multivitamin preparations containing 2500 IU of vitamin A a tablet. Using predialysis fasting blood samples elevations of serum vitamin A concentrations were found in all dialysis patients (mean value 8·1 μmol/l (774 IU/100 ml) SD 2·3 μmol/l (223 IU/100 ml)). We found, however, no correlation between serum vitamin A and serum calcium, but there was a positive correlation with both alkaline phosphatase ($r = 0.81$, $p < 0.001$) and parathyroid hormone ($r = 0.83$, $p < 0.001$) for this group of patients.

Unlike Farrington and others we have not observed a hypercalcaemic effect of vitamin A but rather that its action is to cause an increase in parathyroid hormone secretion. Despite these theoretical differences, however, we agree that high serum vitamin A concentrations may be a factor in renal osteodystrophy.

We feel that his lends further support to the view that it is justifiable to discontinue the practice of prescribing multivitamin preparations containing vitamin A to patients with chronic renal failure.

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Treatment of acute mountain sickness

SIR,—Altitude doctors have gossipped about the physiology of man on high mountains for decades and advised various preventive measures. Often they have been wrong, as in their prediction that Everest without oxygen was not possible. Dr M D O'Brien's letter (29 August, p 616) shows beautifully the muddled physiological model we are still playing with. In the first paragraph he states that "hypoxia alone does not cause cerebral oedema," and yet later he says it "does cause marked cerebral vasodilatation and this results in brain swelling due to vascular congestion."

Dr O'Brien is correct, however, to question any future reliance on acetazolamide as a preventive measure to avoid mountain sickness. The evidence that the drug is useful is slowly growing, but I doubt that we have reached the stage of advising all mountaineers to take it. Much more important are the crude changes that the climber or walker can make to avoid the dangers of altitude such as slow ascent and avoiding sleeping high too early. Even a small modification of the planned rate of ascent is likely to be 10 times more important than the acetazolamide. I doubt, too, that the drug will enormously affect the altered respiratory drive, cardiovascular state, fluid shifts, and renal changes that seem to take place in response to hypoxia.

Have you ever driven slightly more recklessly *because* you are strapped into a car by a seat belt? I think probably that you have. Similarly, the swallowing of this insurance policy of acetazolamide may encourage more reckless climbs in a few individuals and focus attention