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Letters

Measurements for haemoglobin

During the meeting of the International Committee for Standardisation in Haematology (ICSH) secretariat it was noted that an increasing number of papers published in both monographs and journals are now using the World Health Organisation International Committee for Standardisation in Haematology/International Society of Haematology recommendation to express haemoglobin as gram/litre (g/l). Furthermore, haemoglobinometers, which use this scale, are being introduced.

The ICSH secretariat would like to point out the need for a rationalisation of the expression of haemoglobin results that would conform with the principles of SI units, and which should be encouraged as quickly as possible.

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(Will authors please note. Editor)

Convulsions and encephalopathy in a patient with leukaemia after treatment with metronidazole

Metronidazole is an antimicrobial agent commonly used to treat anaerobic bacterial infections. We consider that the side effect of convulsions resulting from treatment with metronidazole¹ may often be unrecognised, particularly as a consequence of cumulative exposure^{2,3} in a patient receiving many other drugs and suffering many complications of treatment. We describe a patient with acute myeloid leukaemia, who developed epileptiform seizures, encephalopathy, and sensory neuropathy after treatment with metronidazole.

A 43 year old woman presented with acute myeloid leukaemia (FAB classification M4) in December 1984. She was treated with Cytarabine 100 mg/m² intravenously for seven days and daunorubicin 50 mg/m² for three days.

During the course of chemotherapy she developed a fever, but no causative organism was identified. Metronidazole (a total of 18.5 g) was given intravenously over the next 12 days in combination with a penicillin and gentamicin. The induction treatment was repeated on 17 December for a further week. Ten days later she became feverish again and *Staphylococcus epidermidis* was isolated from blood cultures. Treatment with fucidin and netilmicin was started and there was an initial improvement, although the temperature never settled. Metronidazole (a total of 10 g) was added with the onset of diarrhoea because pseudomembranous colitis was suspected. A few days later the patient became acutely unwell, had rigors, and the parenteral antibiotics were changed to erythromycin, rifampicin, and ceftazidime. There was dramatic improvement and after five days all antibiotics were stopped.

The patient was clinically well, without fever, and ready for discharge when she was found on the floor having a grand mal fit. Over the next 24 hours she had several more fits despite treatment with chlor-methiazole and phenytoin. A computed tomographic scan showed no focal lesion. An electroencephalogram showed encephalopathy. The urea and electrolyte concentrations sampled 30 minutes before the first fit were normal. The platelet count had not fallen below $60 \times 10^9/l$.

The patient made a recovery to normal within 48 hours, but during this period she suffered some retention of urine. A repeat electroencephalogram four weeks later showed moderate abnormality. At this time the patient was in complete remission awaiting consolidation of treatment.

The patient received a total of 29 g metronidazole and developed epilepsy, encephalopathy, and, probably, sensory neuropathy manifested by retention of urine. Acute encephalopathy and epilepsy secondary to metronidazole probably resulted from a high cumulative dose.³ It is known that the drug crosses the blood brain barrier; in dogs it has been shown that high doses of metronidazole induce Purkinje cell lesions.⁴ It is especially important to be aware of this side effect in the leukaemic patient to avoid unnecessary invasive investigations in the search for

hidden leukaemic deposits or foci of infection.

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Enzyme markers in acute non-lymphoid leukaemia

We read with interest the recent review in the journal by Dr Drexler *et al.*¹ It is stated that "the main group AML did not show one distinct enzyme marker phenotype". While this may be true for the purine salvage enzymes, we would like to draw attention to the use of certain lysosomal enzymes in the diagnosis and classification of acute myeloid leukaemias in both adults and children.² We have now studied 50 patients with acute non-lymphoid leukaemia and have measured the activities and isoenzyme profiles for the enzymes β hexosaminidase and α mannosidase. A summary of our results is shown in the table. Patients with AML, AMOL, and AMOL showed significant increases in these enzyme activities when compared with peripheral blood granulocytes of leukaemic cells of lymphoid origin. Moreover, in patients with AML in particular there was a reduction in β hexosaminidase B isoenzyme peak when compared with the A component.