absence of a family history and the presence of stigmata classically characteristic of non-accidental injury: complex skull fractures, metaphyseal fractures, rib fractures, and fractures at different stages of healing. Fractures sustained by children with mild osteogenesis imperfecta are the same as accidental fractures sustained by normal children: typically fractures of the shafts of long bones. Astley states that metaphyseal fractures are found only in severe osteogenesis imperfecta and not in mild cases.¹ Rib and skull fractures occur in mild osteogenesis imperfecta with the same frequency as they do in normal children. The only series describing metaphyseal, skull, and rib fractures as a prominent feature of mild osteogenesis imperfecta is the series of Paterson quoted by Dr Blumenthal. I am sure that Dr Blumenthal has read the ensuing correspondence from Taitz² who points out that not all courts have accepted Paterson's evidence. Taitz has calculated, with support of several authoritative references,³ that the chance of a child having a mild form of osteogenesis imperfecta without a family history is between 1:1 000 000 and 1:3 000 000.

The third statement (about metaphyseal fractures) in Dr Blumenthal's letter is quoted directly from Paterson who in turn claims authority from Astley¹ but Astley's full quotation is: 'There is evidence of obvious generalised bone disease of osteogenesis imperfecta so that confusion with non-accidental injury does not occur'.

For these reasons I cannot accept Dr Blumenthal's claim that courts will be misled by what I have written.

References

- ¹ Astley, R. Metaphyseal fractures in osteogenesis imperfecta. *Br J Radiol* 1979;52:441-3.
- ² Taitz LS. Child abuse and osteogenesis imperfecta. *Br Med J* 1988;296:292.
- ³ Taitz LS. Child abuse and osteogenesis imperfecta. *Br Med J* 1987;295:1082-3.

Non-accidental injury and copper deficiency

Sir,

Recent excellent articles by Dr Helen Carty¹ and Dr J C L Shaw² on non-accidental injury and copper deficiency correctly and clearly state that the court is not the right place to decide on the child's welfare. I tend to agree with them. It appears that on both prosecution and defence sides winning a case is often of more importance than the truth. In achieving this each side tries to exploit the subtle nature of the medical evidence presented by the so called expert witnesses; this evidence has to be grasped and decided upon by the non-medical judges and juries—an almost impossible task. In my opinion the medical experts are as much to blame as the non-medical law profession in compromising the child's welfare. Surely society and the medical profession, as part of society, should not accept this unsatisfactory situation and should campaign for a system, perhaps away from the courts, which seeks the real truth and ultimately does justice to the child.

References

- ¹ Carty H. Brittle or battered. *Arch Dis Child* 1988;63:350-2.
- ² Shaw JCL. Copper deficiency and non-accidental injury. *Arch Dis Child* 1988;63:448-55.

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Good tolerance of pyrazinamide in children with pulmonary tuberculosis

Sir,

By adding pyrazinamide to the usual triple treatment consisting of rifampicin, isoniazid, and ethambutol, it is possible to reduce the duration of antituberculous treatment with equal efficacy.¹ Despite recommendations² paediatricians seem reluctant to prescribe pyrazinamide, probably because of fear of hepatotoxicity and effects on uric acid metabolism.

We have assessed the tolerance of pyrazinamide in 43 children with primary tuberculosis and abnormalities on chest radiography (principally hilar lymphadenopathy with or without parenchymal infiltration). The diagnosis of tuberculosis was confirmed by a positive Mantoux test (>10 mm) and the finding of *Mycobacterium tuberculosis* in the children or their parents, or both. Mean age at diagnosis was 6 years (range 2 months to 15 years). Nineteen children were less than 3 years old.

Each morning, one hour before breakfast, during the initial phase all the children received rifampicin, isoniazid, and ethambutol at recommended paediatric doses³ and pyrazinamide (mean dose 23.3 mg/kg, range 20 to 37 mg/kg).

This regimen of four drugs was prescribed for two months in 38 children, three months in two, and four months in three children (because of initial resistance to isoniazid). The second phase of treatment consisted of daily rifampicin and isoniazid.

Plasma uric acid and plasma glutamic oxaloacetic and glutamic pyruvate transaminases were measured before initiation of treatment, one month later, and at the end of treatment with pyrazinamide. Plasma uric acid concentrations were normal before treatment (150-360 µmol/l). They increased slightly (360-450 µmol/l) in nine of the children (21%) and returned to normal when pyrazinamide was stopped. There were no signs of gout nor arthralgias.

Liver enzymes were normal before treatment in 42 children and remained normal in 40. In two children transaminases increased (to three times normal values) at one month but returned to normal without modification of treatment. In one child the four antituberculous drugs were started just after viral A hepatitis; this did not prevent the normalisation of liver functions.

This paediatric study is in agreement with those carried out in adults and shows that pyrazinamide is well tolerated when used in doses <35 mg/kg/day even in infants. Furthermore, except in those five children with initial resistance to isoniazid, the duration of treatment was reduced to six months, which compares favourably with