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. 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 Taitz who points out that not all courts have accepted Paterson’s evidence. Taitz has calculated, with support of several authoritative references, 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 Astley 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

Non-accidental injury and copper deficiency

Sir,

Recent excellent articles by Dr Helen Carty and Dr J C L Shaw 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 if 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.

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. Despite recommendations 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 doses 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
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the classical nine month regimen containing rifampicin, isoniazid, and ethambutol.

References

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Accidental poisoning

Sir,

Dr Craft’s annotation on accidental poisoning raises some interesting points.1 The Republic of Ireland has no regulations concerning use of child resistant containers or closures. Despite considerable effort and input into public education (return used medicines campaigns, ‘play it safe’ series, posters, and television campaigns by the Health Education Bureau and others, etc) the number of children attending our hospital after accidental drug ingestion has not altered (table).

Table No of children attending hospital after accidental drug ingestion

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</thead>
<tbody>
<tr>
<td>Total No of children</td>
<td>322</td>
<td>237</td>
<td>323</td>
<td>230</td>
</tr>
<tr>
<td>Salicylates</td>
<td>67</td>
<td>34</td>
<td>30</td>
<td>11</td>
</tr>
<tr>
<td>Paracetamol</td>
<td>19</td>
<td>24</td>
<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

In 1987 a working party convened by the Faculty of Paediatrics of the Royal College of Physicians of Ireland concluded that application of child resistant containers to all medications on a regulatory or voluntary basis could be counter productive, especially where the drug contained therein was not toxic (vitamins, antibiotics, etc). It tried to select out those drugs which are most commonly ingested or most potentially toxic and recommended that they be dispensed in child resistant containers. The following groups of drugs were included:

- Anticonvulsants
- Digoxin
- Theophylline and derivatives
- Sedatives, hypnotics, and tranquillisers
- Tranquilizers and other antidepressants
- Opiate derivatives, including preparations containing diphenoxylate
- Appetite suppressants

It was estimated that the above groups of drugs account for about 60–70% of accidental ingestions presenting to hospital. It was suggested that ordinary containers could be provided on the request of patients, to old or disabled people, or at the discretion of the prescribing doctor or dispensing pharmacist.

The secret of success in accident prevention is access prevention. Child resistant containers constitute the most consistent method of prevention. Every consequence of accidental poisoning (induced emesis, gastric lavage, hospital admission, intravenous fluids) is extremely unpleasant to that most vulnerable group—toddlers and preschool children.

It never ceases to amaze me how little apparent consumer resistance there is to some everyday individually wrapped items (milk, marmalade, mayonnaise, peanuts) which can be extremely difficult to open. Therefore on with child resistant containers in a wider range of medications and household chemicals!

Finally, there is one commercial closure for use with liquids—‘Squeeze ‘N Turn’ (United Closures and Plastics).

References

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Isolated pulmonary histiocytosis

Sir,

Isolated pulmonary histiocytosis is indeed a rare disorder, but it is not as rare as reported by McDowell et al in their article.1 In world literature, 11 cases of isolated pulmonary histiocytosis in the paediatric age group have been reported.2

The prognosis of isolated pulmonary histiocytosis is grave. Of 11 reported cases, six died within one year of diagnosis.2 Most of these patients were treated with a combination of steroids and alkylating agents. Nondahl et al reported one case who improved with steroids alone.2

- Aspirin, paracetamol, and other analgesics
- Non-steroidal anti-inflammatory agents
- Iron (haematinics)
- Antihistamines, including antitussives and decongestants