Child abuse and metabolic bone disease: are they often confused?

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It has been claimed that children with fractures (usually multiple) attributed to child abuse may in fact have metabolic bone diseases, such as osteogenesis imperfecta.[1,2] I report a series of court cases brought between 1981 and 1990 in which I was asked for a further opinion on this question.

Subjects, methods, and results

I assessed 22 infants and children with fractures (mean age 6 months) who were the subjects of proceedings for child protection (19) or whose parents had been charged with causing their injuries (three). I scrutinised the medical notes, court papers, and *x* ray films for evidence of metabolic bone disease, particularly osteogenesis imperfecta (using the classification of Sillence *et al*[3]), copper deficiency,[4,5] metabolic disease of prematurity, and "temporary brittle bones."

Fourteen children had at least one rib fracture, which was associated with limb fractures in 10 and with limb and skull fractures in one. Sixteen children had rib fractures or metaphyseal fractures, or both, which are characteristic of non-accidental injury. Absence of bruising at the site of the fracture in 10 children was claimed to be evidence for metabolic bone disease, but nine had clear additional evidence of non-accidental injury. The diagnoses suggested as alternatives to non-accidental injury were osteogenesis imperfecta in nine children, prematurity in two, and copper deficiency in two. In eight children osteogenesis imperfecta, copper deficiency, and temporary brittle bone disease were all offered as alternative diagnoses.

In two patients, who were siblings, type I osteogenesis imperfecta had been diagnosed by the paediatrician. No other child had any first order features of osteogenesis imperfecta—namely, recurrent unexplained fractures while in foster care, blue sclerae in both the child and a parent, a history of recurrent fractures in one parent, multiple wormian bones, or dentinogenesis imperfecta. Second order features of osteogenesis imperfecta, such as joint hypermobility, or discoloured sclerae in a child under 6 months, were reported in several cases but were either unconfirmed or not diagnostic in the absence of first order features. Apart from the fractures, the bones were radiologically normal in all except the two children with type I osteogenesis imperfecta. No evidence of copper deficiency, such as deficient copper intake associated with extreme prematurity or malnutrition, or of osteopenia, delayed bone age, refractory hypochromic anaemia, or neutropenia was found in any of the 10 children in whom it had been suggested. There were two premature infants. Neither had been fed parenterally and neither showed evidence of osteopenia or rickets. Temporary brittle bone disease was proposed as a diagnosis for seven children. The fractures ceased immediately the children were placed in foster care. All had additional evidence of abuse.

Additional evidence of abuse was present in 17 cases (table). In 20 cases the courts decided that the fractures were due to child abuse; in the remaining two, in which the fractures were due to proved osteogenesis imperfecta type I, there was other evidence of abuse. The children were placed in foster care and only one further fracture occurred, four years after placement, when the child fell off a wall.

Evidence of child abuse in children and infants with unexplained fractures

Brain damage	1
Multiple evidence of abuse and neglect	2
Subdural haemorrhage	2
Facial bruises and torn frenulum	2
Multiple bruises and oral injury	1
Multiple bruises	5
Previous bruising suggestive of non-accidental injury	1
Neglect	2*
Confession by perpetrator	1
Fractures only	5

*Fractures proved to be due to type 1A osteogenesis imperfecta.

Comment

In cases that have reached the stage of court proceedings the likelihood that a form of brittle bone disease has been missed is small, and with a careful history, complete examination, and appropriate investigations there is usually no difficulty in excluding these very rare conditions. Absence of bruising at the site of fracture is irrelevant to deciding whether a fracture is traumatic or not. In none of the children in this series was there convincing evidence of osteogenesis imperfecta, copper deficiency, or any metabolic bone disease.

1 Paterson CR, McAllion S. Osteogenesis imperfecta in the differential diagnosis of child abuse. *BMJ* 1989;299:1451-4.
2 Taitz LS. Child abuse and osteogenesis imperfecta. *BMJ* 1988;296:292.
3 Sillence DO, Senn A, Danks DM. Genetic heterogeneity in osteogenesis imperfecta. *J Med Genet* 1979;16:101-16.
4 Paterson CR. Child abuse or copper deficiency? *BMJ* 1989;295:213.
5 Taitz LS. Copper deficiency and unexplained fractures in infants. *Child Abuse Review* 1987;1:6.

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Decompression sickness in fish farm workers: a new occupational hazard

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Fish farming is a new industry that needs divers to maintain the cages in which the fish are kept. Dead fish in a cage provide a focus of infection for healthy fish and must be cleared out regularly. Some fish farmers think that the most effective method of clearing dead fish is with divers, but this new diving practice also has its problems. We report three anomalous cases of decompression sickness.

The medical problems of compressed air diving are reviewed elsewhere.[1] Decompression sickness is caused by the release of dissolved nitrogen from tissues during and after the ascent phase of the dive. The principal manifestations are joint pain or impairment of central nervous system function shortly after surfacing. Delay in starting recompression treatment may cause spinal paraplegia or cerebral damage. Using decompression tables reduces the likelihood of decompression sickness by predicting ascent rates and stoppages for given times and depths.

Case reports

Case 1—In a series of nine descents and ascents to and from 18 metres without decompression stops diver 1 cleared six cages and after a surface interval of three