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

L S Taitz

Children's Hospital,
Sheffield S10 2TH
L S Taitz, MD, senior lecturer
in paediatrics
(Dr Taitz died on
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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*), copper deficiency,^{3,5} 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 de-

ficiency, 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, McAllison 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

J D M Douglas, A H Milne

Tweeddale Medical
Practice, Fort William
PH33 6EU
J D M Douglas, MRCP,
general practitioner

Robert Gordon's Institute
of Technology, Aberdeen
AB2 3BJ
A H Milne, MRCP, senior
medical adviser, survival
centre

Correspondence to:
Dr Douglas.

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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

hours cleared the other three. His total time in the water for the nine dives was 44 minutes (figure). His subsequent neurological and musculoskeletal decompression sickness was successfully treated with hyperbaric oxygen according to the US Navy table No 6.

Dive profile, case 1. Total time in water (A + B) was 44 minutes (US Navy no stops time at 18 metres = 60 mins)

Case 2—Five descents and ascents to and from 15 metres took diver 2 a total of 48 minutes and were completed without decompression stops. Subsequent musculoskeletal decompression sickness was successfully treated with reference to US Navy table 6.

Case 3—Over two weeks diver 3 made 14 dives a day in rapid succession with no decompression stops. The first six dives were to 18 metres with an average time spent on the bottom of 10 minutes, followed by eight dives to 9 metres with times spent on the bottom ranging from five to 10 minutes. He continued to dive to this pattern despite developing neurological decompression sickness. When his illness was brought to medical attention he was successfully treated, 56 hours after surfacing from his last dive, according to an extended US Navy table 6 and had follow up hyperbaric oxygen treatments.

Comment

Three professionally trained divers followed widely used decompression tables but sustained decompression sickness from depths considered shallow and from

total times at depth within safe limits for a single descent and ascent. Repeated descending and ascending along the series of cages was the factor that caused decompression sickness.

The absorption, distribution, and release of nitrogen from the body during a dive with normal air is a complex event. Doppler studies have shown that "symptomless" bubbles are formed even after shallow dives, which do not cause decompression sickness.² In "yo-yo" diving, on subsequent descents bubbles may not be completely returned to solution, and they may become coated by clotting proteins.³ These rheological seeds may grow more quickly with subsequent dives in a sequence and cause tissue damage through embolisation of blood vessels. Symptomless patent foramen ovale causing arterialisation of bubbles has been postulated as a mechanism of bubble distribution,⁴ and clinical studies suggest permanent neurological deficits may remain after treatment for decompression sickness.⁵

The long term health hazards of diving are not completely known; we believe that the diving techniques used in fish farming are dangerous. A dive conducted while the increased nitrogen from a preceding dive is still being offloaded introduces a more complex pattern into decompression than is catered for in current decompression procedures.

Fish farm diving carries risks from contaminated water and from net entrapment, which has already resulted in the death of a diver. These problems can be eliminated by fish husbandry methods that use special nets rather than divers to clear dead fish.

- 1 Douglas JDM. Medical problems of sport diving. *BMJ* 1985;291:1224-6.
- 2 Spencer MP. Decompression limits for compressed air determined by ultrasonically detected blood bubbles. *J Appl Physiol* 1976;40:229-35.
- 3 Hallenbeck JM, Anderson JC. Pathogenesis of decompression sickness. In: Bennett PB, Elliott DH, eds. *Physiology and medicine of diving*. 3rd ed. London: Baillière Tindall, 1982:435-60.
- 4 Wilmshurst PT, Byrne JC, Webb-Peploe MM. Patent foramen ovale and decompression sickness in divers. *Lancet* 1989;ii:513-4.
- 5 Adkisson GH, MacLeod MA, Hodgson M, et al. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989;ii:119-22.

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Care of women with puerperal psychiatric disorders in England and Wales

R J Prettyman, T Friedman

Leicester General Hospital, Leicester LE5 4PW
R J Prettyman, MRCP, senior house officer
T Friedman, MRCPsych, consultant liaison psychiatrist

Correspondence to: Dr Friedman.

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Women are known to have a high incidence of mental disorder in the puerperium.¹ Mothers are admitted to hospital with serious mental illness including puerperal psychosis after childbirth at a rate of about 1 in 450 pregnancies.² A population of 500 000 might be expected to produce 10 cases of puerperal psychosis needing admission each year.

Over the past 40 years specialist units for the care of mentally ill mothers and their babies have emerged in Britain. We present a survey of the current provision of psychiatric mother and baby inpatient facilities among the health authorities of England and Wales.

Methods

A brief questionnaire was circulated to the unit general managers for psychiatric services in 201 health authorities in England and Wales. Questions were asked about the current facilities for care of women with puerperal psychiatric disorders; the number of

consultants caring for such women; plans for changing or upgrading the facilities; and perceived priority of providing a specialised service.

Results

We received 194 completed questionnaires, a response rate of 97%. Thirty eight districts (19%) reported having dedicated facilities for mentally ill mothers and their babies. Ninety four (48%) reported that mothers with babies were admitted to acute general psychiatric wards, using existing facilities, and 50 (27%) used facilities in another district. Twenty one districts (10%) reported that there was no provision for any sort of joint admission. These categories are not mutually exclusive. The table shows the distribution of these types of facility by health region. Almost half the dedicated facilities (18) were stated to consist of one or two beds, with a few units having over six beds.

Of the 38 districts with dedicated mother and baby facilities, 21 (55%) indicated that mothers were admitted under the care of consultant(s) with a special interest in puerperal psychiatry. Only 58 (35%) districts with no dedicated mother and baby facilities stated that admissions were under the care of consultant(s) with a special interest ($\chi^2=6.46$, $df=1$; $p=0.01$).

Thirty districts indicated that they planned to set up a dedicated mother and baby inpatient facility and 63