Review Article

Metabolic bone disease in preterm infants

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Summary

Small preterm infants often develop osteopenia with or without rickets and with or without fractures. Whether these bone abnormalities all form part of the same disease process with a wide spectrum of presentation or whether each abnormality represents a different disease is as yet unclear. Bone mineralization depends largely on adequate supplies of calcium and phosphate. The normal intra-uterine accretion of these minerals is higher than can be achieved by feeding preterm babies postnatally with breastmilk or conventional formulas. Supplementation with calcium, phosphorus and vitamin D is needed to prevent the development of 'neonatal osteopathy'. The main action of vitamin D in the preterm baby is probably to increase the intestinal absorption of calcium and phosphorus, although it may, together with other calciotrophic hormones, have a more specific effect on bone growth.

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Von Sydow[1] noticed rickets in preterm infants as early as 1946. Since that time several authors[2-9] have reported rickets in neonates, especially in very low-birth-weight (VLBW) babies. A wide variety of metabolic bone diseases in neonates have recently been reported, ranging from metaphyseal demineralization to generalized skeletal demineralization, periosteal reactions and fractures.

It is not known whether rickets, bone demineralization, fractures and periosteal reactions all form part of the same disease process with the same pathogenesis but with a wide spectrum of clinicoradiological presentation, or whether each of these entities represents a different disease with its own pathogenesis, pathology and outcome. Koo *et al.*[10] and Masel *et al.*,[8] proposed a method for the radiological classification of skeletal disease in preterm infants, based on the presumption that the different radiological features represent a single disease process. The possible evolution of this 'neonatal osteopathy', starting as demineralization at the one end of the spectrum and manifesting as frank rickets with fractures at the other end of the spectrum, raises problems of diagnosis, therapy and prognosis at each stage. Several reports of rickets in small babies have been published[11-13] where a decreased bone density was noted *before* the development of frank rickets. Moreover, most cases of rickets in babies are associated with a decreased bone mass.[6,8,10,14-17] Rickets in small babies is often associated with fractures which tend to be multiple and/or occur in unusual sites.[2,4,6,8,13,18-20] However, infants may present with fractures *without* the radiological signs of rickets and with normal or only very slight abnormalities in serum biochemistry.[2,21]

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Prevalence

The prevalence of these different metabolic bone diseases vary in different studies, depending on the type of infant studied and the diagnostic methods used.

The true incidence of *skeletal demineralization* in neonates is unknown, partly because it usually remains subclinical until frank rickets or fractures develop. Moreover, accurate routine methods have not yet been established for the *in vivo* evaluation of bone mass or bone mineral content (BMC) in babies. Poznanski *et al.*[22] used conventional chest radiography for measuring humeral cortical thickness as an indicator of cortical bone mass. Another group of workers[23-25] recently described the use of photon absorptiometry for quantitating bone mineral content in neonates. They documented a direct correlation between gestational age and BMC[23,25,26] as well as cortical bone mass[22] in appropriate-for-gestational-age (AGA) infants. BMC did not differ in AGA and small-for-gestational-age (SGA) babies.[27] A direct correlation was also found between BMC and bone width,[23] as well as between BMC (or cortical bone mass) and birth weight in AGA babies.[22,25] No sex[22,23,26] or race[26] differences in BMC or cortical bone mass at birth have been demonstrated. Postnatally the increase in BMC of preterm babies is significantly less than the expected *in utero* BMC,[25,27] implying that postnatal bone mineralization lags behind *in utero* bone mineralization.

Although the prevalence of metabolic bone disease in babies varies in different series, it is clear that it is very common, especially in VLBW and extremely low-birth-weight (ELBW) infants, approximating 100% in some studies (Table I).

Diagnosis

Severe rickets and multiple fractures in small babies may be completely subclinical.[15,16,21] In preterm babies fed standard formulas, demineralization at the growing ends of long bones seems to start almost immediately after birth. Rickets, fractures and generalized bone demineralization in preterm babies always occur before the age of 6 months,[3,5,6,9,31] and usually before the age of 3 months.[3,6,8,10,13,14,21,30]

The diagnosis of metabolic bone disease in infants can be suspected on the grounds of abnormal biochemical data, especially raised serum alkaline phosphatase (ALP) levels. The precise value of hyperphosphatasaemia as an indicator of bone disease in the neonate is, however, still controversial. High

TABLE I. PREVALENCE OF METABOLIC BONE DISEASE IN LBW, VLBW AND ELBW INFANTS

Bone disease	Condition of infant and study ref.	Prevalence	Diagnostic method
Metaphyseal demineralization	**ELBW**[8]	**100%**	**Ordinary radiography**
Generalized demineralization	**ELBW**[8]	**67%**	**Ordinary radiography**
	VLBW[6,10,28]	**15 - 31%**	**Ordinary radiography**
	VLBW[29]	**100%**	**Fine-grain radiography**
Rickets	**ELBW**[8,30]	**44 - 61%**	**Ordinary radiography**
	VLBW[3,6,7]	**13 - 32%**	**Ordinary radiography**
	LBW[9]	**37 - 55%**	**Ordinary radiography**
	VLBW[10]	**1/19**	**Ordinary radiography**
Fractures	**ELBW**[8]	**38%**	**Ordinary radiography**
	VLBW[6]	**20%**	**Ordinary radiography**
	Preterm with rickets	**5/9**	**Ordinary radiography**
	VLBW[10]	**1/19**	**Ordinary radiography**

serum ALP values have been reported in infants with bone demineralization, rickets, periosteal reactions and fractures.[10,13,20,30,32] Serum ALP has been shown to have an inverse correlation with gestational age,[32] growth velocity in the preterm baby[33] and calcium intake,[28] but no correlation between ALP and BMC or vitamin D supplementation has been reported.[34] The high serum ALP levels in preterm infants with or without rickets may even be a normal response to a physiological increase in osteoblastic activity and bone mineralization.[19]

The diagnosis of metabolic bone disease, based on abnormal biochemical findings, must be confirmed radiologically. Bone demineralization is usually present in infants with rickets.[6,8,10,14–17] The typical radiological signs of rickets, namely loss of the dense white metaphyseal line, increased submetaphyseal lucency, fraying, splaying and cupping of the metaphysis, as well as fractures may be present.

Only a few publications are available on the histology and bone histomorphometry of 'neonatal osteopathy'. The postmortem histological findings in 1 preterm baby suffering from rickets showed excessive proliferation of subperiosteal bone associated with moderate amounts of osteoid proliferative changes in the epiphyseal cartilage, mildly increased but poorly ossified osteoid on endochondral bone, and mild osteoporosis.[35] The undecalcified bone of 2 preterm babies with florid radiological and biochemical evidence of rickets, revealed the expected loss of normal epiphyseal architecture, thick osteoid seams, increased numbers of osteoblasts and osteoclasts, and peritrabecular marrow fibrosis.[13] A major problem with the interpretation of less striking histological abnormalities involves the lack of published data on normal histomorphometric findings in healthy infants, as well as the fact that the histology may indeed change dramatically even with mild and short-term disease.[36] Much more work is required in this field before bone histology assumes the importance and standing it enjoys at present in studies of metabolic bone disease in adults.

Pathogenesis

The pathogenesis of metabolic bone disease in infants, and especially that of osteopenia in the preterm baby, is largely unknown. It is difficult to compare the data from different reports, because of numerous variables in the different studies, e.g. the type of baby (ELBW, VLBW, preterm, term, sick, healthy, AGA, SGA, etc.), the feeding regimen (ordinary formula, formula adapted for preterm babies, pooled breastmilk, mother's own breastmilk, additional varying dosages of calcium, phosphorus, vitamin D or vitamin D metabolites), the age of the baby and the duration of follow-up, as well as the method used to detect bone disease (photon absorptiometry, conventional radiography, fine-grain radiography). Furthermore, the cause of this 'neonatal osteopathy' is likely to be multifactorial, making interpretation and comparison of data from the different studies even more confusing.

It is clear that BMC and cortical mass at birth is lower in preterm babies than in term babies.[22,26] The postnatal increase in BMC in preterm babies also lags significantly behind the expected intra-uterine BMC.[25,27] The BMC at birth as well as postnatal bone mineralization do not seem to differ in AGA and SGA preterm babies.[27]

Some of the factors that may be involved in the pathogenesis of 'neonatal osteopathy' are listed in Table II and discussed below.

TABLE II. FACTORS POSSIBLY INVOLVED IN THE PATHOGENESIS OF 'NEONATAL OSTEOPATHY'

- **Vitamin D**
- **Minerals**
  - **Calcium**
  - **Phosphate**
- **Other calciotrophic hormones**
  - **Parathyroid hormone**
  - **Immunoreactive calcitonin**
- **Systemic disease and acidosis**
- **Protein and trace elements**
- **Miscellaneous**
  - **Immmobilization and weightlessness**
  - **Sex hormones**
  - **Prostaglandins**
  - **Growth-regulating hormones and factors**

Vitamin D

Maternal nutrition is important for the vitamin D status of the fetus and may play a role in fetal and postnatal bone mineralization and calcium homeostasis. There is a direct relationship between maternal and cord blood 25-hydroxyvitamin D (25-OHD) concentrations.[37–45] The majority of studies report no correlation between maternal and cord blood 1,25-dihydroxyvitamin D ($1,25(OH)_2D$) levels,[37,38,43,44] but a

direct correlation has been reported in one study.[45] Data on the correlation between maternal and cord blood 24,25-dihydroxyvitamin D ($24,25(OH)_2D$) are conflicting.[37,44–46] Although vitamin D deficiency in pregnancy, a seemingly common occurrence,[39,47,48] does not appear to affect fetal growth,[47] it may cause congenital rickets,[49,50] affect future dentition,[51] or put the infant at risk for developing rickets later in life.[7] Low maternal and neonatal serum 25-OHD levels have been recorded in babies with hypocalcaemia,[52] but the effect of maternal vitamin D status on bone mineralization is less clear and no relationship in BMC just after birth and cord blood 25-OHD could be documented.[53]

Although most proprietary infant formulas in the RSA are enriched with vitamin D, usually to a level of about 400 IU/l vitamin D, preterm babies consume only small amounts of milk initially, thus diminishing their vitamin D intake, often to as little as 50 - 100 IU daily. The exact vitamin D requirements of preterm babies are still unknown. Rickets has been reported in ELBW and VLBW babies receiving no additional vitamin D[5] or up to vitamin D 400 IU daily.[11,19]

Intestinal absorption of vitamin D in the preterm infant seems adequate,[54–56] although not necessarily so in the VLBW baby.[57] Medium-chain triglycerides (MCT), which increase fat absorption in preterm infants,[58,59] have no effect on vitamin D absorption.[57] In preterm babies maximal calcium absorption and calcium balance similar to fetal accretion have been found with vitamin D dosages ranging from 600 to 2 000 IU daily.[57,60]

The concentration of the different vitamin D metabolites may be influenced by the amount of vitamin D binding protein (DBP), which has been found to be normal in the cord blood of term babies, but decreased in preterm cord blood.[61,62] Although initial studies showed that preterm infants were unable to maintain adequate 25-OHD serum levels,[63] more recent studies clearly demonstrated that the hepatic as well as the renal hydroxylation of vitamin D is adequate in the fairly healthy preterm infant.[55–57,60,64–67] However, in 45% of sick preterm babies 25-OHD levels remained low until 12 weeks of age.[28] The hydroxylation reactions may also be impaired in some babies under 2 000 g who may then develop bone demineralization and/or rickets.[9–11,13,68] In contrast, serum 25-OHD values have also been reported as normal,[19,30,31,69] or even increased[4,20,30] in preterm infants with rickets.

Serum levels of $1,25(OH)_2D$ have been found to be normal or increased[4,14,18,31,69] in preterm babies with rickets, when compared with formula or breast-fed healthy babies.[31,70] Markestad *et al.*[31] state that the normal serum 25-OHD, the raised $1,25(OH)_2D$ and the low $24,25(OH)_2D$ level found in a VLBW baby with rickets do not differ from those of healthy preterm babies. They speculate that the higher than adult $1,25(OH)_2D$ serum values reported in healthy preterm infants, as well as in babies with rickets, represent a normal compensatory effort to ensure maximal absorption from a mineral-poor diet.[31,66]

Vitamin D metabolism and the hormonally active vitamin D metabolite(s) during fetal and early postnatal life may differ from those in the older child and adult. In animal experiments it has been shown that $24,25(OH)_2D$ is necessary for epiphyseal bone growth, while $1,25(OH)_2D$ is more important for diaphyseal bone growth.[71] It seems fairly certain that $24,25(OH)_2D$ is needed for optimal bone mineralization,[71–74] although some workers dispute this.[75]

Minerals

Calcium deficiency has been cited as a cause for rickets in children over the age of 12 months.[76–78] Maternal malnutrition during pregnancy causes low bone density in the baby,[79] and calcium supplementation in pregnant malnourished women has been shown to result in an increased bone density in their babies.[80]

The fetal skeleton accretes most of its calcium during the last trimester of pregnancy at a rate of elemental calcium 100 mg/kg fetal weight/d.[81,82] It can only be assumed that the calcium requirements of a preterm infant during early postnatal life are similar to that *in utero*. The presumed large need for calcium and phosphorus may thus comprise a major contributing cause to the osteopenia and/or rickets seen in preterm babies. Intra-uterine accretion of calcium cannot be achieved with breastmilk,[83,84] cow's milk,[83] standard infant formulas,[83,85] soy milk products[3,86] or parenteral nutrition solutions.[12] Furthermore calcium absorption is highly variable in preterm infants, ranging from 29 to 80%[83,85,87,88] and is influenced by a multitude of factors. Earlier studies showed that calcium absorption from breastmilk (65%) was higher than from cow's milk (29 - 34%),[83] but more recent work revealed that the type of milk does not influence calcium absorption.[87] Calcium absorption in babies is improved by a high calcium intake,[89–92] adequate vitamin D,[60,92] lactose-containing formulas,[86] and increased gestational age.[83] Data on the effect of increased postnatal age[83,87,90,93] and fat malabsorption[83,93] on calcium absorption are contradictory. Although the fat composition of milk may influence calcium absorption,[58,60,94] supplemental medium chain triglycerides do not seem to improve calcium absorption.[57] The calcium/phosphate ratio in infant feeding may[92] or may not[89] have an effect on calcium absorption. Calcium retention, on the other hand, is enhanced by phosphate supplementation in preterm babies.[60,95]

Osteopenia is more common in VLBW babies fed a low calcium diet.[28] In fact, the growth, length and head circumference of VLBW babies fed breastmilk, approximate intra-uterine growth, but the bone mineralization of these babies is impaired.[88] With higher calcium and phosphate intakes (200 - 250 and 110 - 125 mg/kg bodyweight/d respectively), the BMC of LBW babies paralleled expected intra-uterine mineralization.[26] Calcium supplementation of milk formulas has been shown to decrease the incidence of radiologically identifiable bone disease[17] as well as osteopenia.[26,85]

The estimated oral requirement of phosphate for LBW babies is 110 - 125 mg (4 mmol)/kg bodyweight/d.[86] The intestinal absorption of phosphate in LBW infants fed human milk or modified formulas ranges from 80 to 94%[60,85,88] and seems to be independent of calcium absorption.[60] In preterm babies fed human milk the serum phosphate level is lower,[29,96] the renal excretion of calcium higher,[29,96] and the phosphate retention reduced by 50% when compared with babies fed a modified formula milk.[96] It has been suggested that the preterm infant on breastmilk should have a supplement of elemental phosphate 8 - 9 mg/100 ml milk to avoid hypercalciuria.[60,95]

Phosphate depletion in adults leads to low serum phosphate levels and raised serum $1,25(OH)_2D$ concentrations, resulting in increased intestinal calcium absorption and enhanced bone resorption.[97,98] Phosphate supplementation has been reported to heal rickets associated with low serum phosphate levels and elevated serum vitamin D metabolite levels in ELBW babies.[4,20] In fact, most preterm infants may be phosphate and/or calcium depleted as is evident from their high serum $1,25(OH)_2D$ levels.[66]

Formula feeding with added calcium, phosphate and vitamin D to meet the nutritional needs of growing preterm babies, has led to nutrient retention rates[57,88,99,100] and BMC[101] comparable to expected intra-uterine values. It seems probable that both vitamin D and sufficient minerals are important in preterm infants, and that neither can totally compensate for the other.

Other calciotrophic hormones

Increased serum parathyroid hormone (PTH) levels cause bone resorption and may contribute to the osteopenia of the

neonate. Neonatal hyperparathyroidism results in bone demineralization.[102,103] Although initial studies showed parathyroid function to be impaired in preterm babies,[104] or PTH to be undetectable in 90% of cord blood samples of healthy preterm infants,[105] it is now clear that preterm and LBW babies secrete PTH adequately after birth.[56,105,106] Formula-fed babies have higher serum PTH levels than breast-fed babies, but no effect on bone could be demonstrated.[29] Although no radiological evidence of hyperparathyroidism could be found in LBW babies with rickets,[15] serum PTH level was elevated in all the preterm babies with rickets in whom it was measured.[13,14] At present the correlation between neonatal serum PTH levels and their effect on bone is not clear and needs further investigation.

Immunoreactive calcitonin (iCT) levels in cord serum are higher than in normal adult serum.[44,107] Serum iCT further increases postnatally and this increase is more pronounced in preterm infants than in term babies.[105,106,108] The stimulus for the increase in serum iCT levels is unknown, and calcium administration to preterm babies either had no effect[105,106] or caused a dramatic increase[108] in serum iCT levels. Serum iCT levels seem to remain high for some weeks after birth and may have a protective function against excessive bone resorption.[107,109] The presumed anabolic effect of iCT on growing bone is, however, disputed by others who could not detect iCT in the serum of 1- and 2-month old VLBW babies.[29] Low serum iCT levels have been found in preterm babies with rickets.[14,69] However, at present no data exist whether infants with relatively low iCT levels at birth are more likely to develop osteopenia.

Systemic disease and acidosis

Most series support the clinical impression that preterm babies who develop rickets are more ill than those without bony abnormalities.[2,5,7,10,13,14,20,31,110] In one report the sicker babies had a greater persistence of bony abnormalities and showed less complete recovery.[8] Acidosis is a known cause of bone resorption.[111] The neonate is often acidotic due to systemic disease, infection and immature renal tubular function[112] and this may contribute to the development of 'neonatal osteopathy'.

Protein, vitamin C and trace elements

Protein deficiency decreases bone formation[113] and the hypoalbuminaemia often present in preterm babies may adversely affect bone growth.

In infants, vitamin C deficiency leads to severe osteopenia, but at present no information on the possible effect of vitamin C deficiency in the pathogenesis of neonatal osteopenia is available.

The amount of copper (Cu) in the fetal body increases three- to fourfold in the last trimester of pregnancy and the preterm infant fed breastmilk is in a negative Cu balance until about 5 weeks of age.[114] Severe bone abnormalities due to Cu deficiency, mimicking rickets, have been described[115,116] and it is possible that Cu plays a more important role in neonatal bone development than is generally accepted.

Miscellaneous

Immobilization and weightlessness cause bone loss due to decreased bone formation, unopposed osteoclastic resorption and increased urinary calcium loss.[117] These changes seem to be reversible, although the period of recovery is much longer than the period of loss, and recovery is not always complete.[117] Sick preterm babies are usually very immobile in the immediate postnatal period and this may influence their bone formation. At present no publications in the English-language literature are available on the effects of immobilization and muscle tone on bone mineralization in babies and it can only be speculated whether these factors are of importance in the genesis of neonatal bone disease.

Oestrogen and progesterone stimulate *in vivo* bone differentiation and mineralization in rats and are necessary for the initiation of bone formation in plaques and the maintenance of mineralization initiated in fetal life.[118] Postmenopausal osteoporosis and its relation to oestrogen and progesterone is well known. It is tempting to speculate that the shorter period of intra-uterine exposure to these hormones in preterm babies may contribute to their osteopenia.

Bone resorption secondary to prostaglandins have been well documented.[119,120] Periosteal reactions have been described in neonates receiving prostaglandin E_1.[120–122] Preterm infants often receive a multitude of medicines, some of which may, like the prostaglandins, iatrogenically influence skeletal integrity.

Various other systemic growth regulating hormones and circulating growth factors (e.g. growth hormone, somatomedin, insulin and insulin-like growth factor, glucocorticoids, thyroid hormone), as well as a variety of local skeletal growth factors have been shown to influence bone growth *in vivo* and *in vitro*,[123,124] but their role in the development of perinatal bone disease is purely speculative at this stage.

Prognosis, prevention and treatment

The short- and long-term effects of neonatal bone disease are as yet unknown and will probably vary depending on the cause and extent thereof. Rickets may cause an initial decrease in longitudinal growth,[33] but after treatment, growth and skeletal development seemingly normalize.[13,20,69] Whether rickets *per se* has an influence on mortality is still uncertain, but it can cause severe respiratory distress.[35] No long-term follow-up studies of infants with neonatal metabolic bone disease have been reported in order to assess its effect on later childhood and adult bone development. In Sweden[125] 75 preterm babies were examined 4-16 years after birth. It is unknown whether these children had neonatal bone disease, but at the time of the study the BMC was similar in children born SGA and AGA. The boys who were preterm babies, had a lower BMC than controls, but were also shorter and lighter than controls. The BMC of girls who were preterm babies did not differ from that of controls.

Treatment of rickets in the newborn depends on the cause. In the overt case, a cause can usually be found and is most often due to a deficiency of vitamin D and/or calcium and/or phosphorus. A major problem is, however, the prevention of bone disease in the neonate. Vitamin D supplements for pregnant women, result in higher cord 25-OHD and calcium levels, lower ALP concentrations[47] and better growth in the babies than in infants whose mothers did not receive supplements.[48] It may be difficult to diagnose the at-risk child at birth and for this reason Brooke[41] advises supplemental vitamin D (400 IU/d) for all breastfed term babies. Indeed, term breastfed babies receiving supplements of vitamin D 400 IU/d were found to have higher serum 25-OHD levels than infants who did not receive supplements at the age of 12 weeks and 1 year; likewise their BMC was significantly higher than that of controls at 12 weeks, although not at 1 year.[34,84] Rothberg *et al.*[126] found that vitamin D supplementation (500 or 1 000 IU/d) for lactating mothers resulted in higher serum 25-OHD levels in their healthy term babies compared with babies whose mothers received no supplements. However, supplementing the baby's formula (400 IU/d) resulted in even higher serum 25-OHD levels. From these reports it seems

reasonable to recommend additional vitamin D (400 IU/d) to all healthy term babies, especially since the amount of sunlight exposure and the role of solar irradiation in the synthesis of vitamin D in babies are still uncertain.

At present it seems reasonable to advise that preterm babies should receive supplementary vitamin D, calcium and phosphate. The optimal form and dosage of these substances, as well as the time to initiate and terminate supplementation, remain to be defined. In VLBW babies receiving vitamin D 800 IU/d, bone demineralization occurred in 32%,[10] but it was less severe than in those receiving 400 IU/d.[67] Radiological rickets was not detected in preterm infants receiving vitamin D either 400 or 1 000 IU/d.[55] It was detected in 37 - 55% LBW babies without vitamin D supplementation but in only 17 - 36% of those receiving vitamin D 500 IU/d.[9] However, in 2 of 19 VLBW infants and in 8 of 15 ELBW babies rickets developed despite the fact that they had received vitamin D 800 IU and 2 000 IU/d respectively.[10,30]

Whether or not the preterm baby needs one of the metabolites of vitamin D routinely to prevent 'neonatal osteopathy' is still uncertain. A lower incidence of rickets has been reported in LBW babies receiving 1-α-hydroxyvitamin D_3 (1-α-OHD_3) than in babies receiving up to vitamin D 500 IU/d.[9] However, no advantageous effect of 25-OHD therapy, compared with vitamin D supplementation (400 IU or 800 IU) on the bone mineralization of preterm babies could be demonstrated. In fact, compared with treatment with 25-OHD, vitamin D 800 IU/d seemed to result in better bone mineralization at 6 and 9 weeks of age.[67] In another study, 8 ELBW babies who developed rickets while receiving vitamin D 2 000 IU/d were all cured independent of whether they continued to receive the vitamin D 2 000 IU daily or whether the vitamin D was changed to 1-α-OHD_3.[30] As stated previously, preterm babies also need additional calcium and phosphate to maintain expected *in utero* bone mineralization,[26] and to decrease the occurrence of osteopenia and radiologically identifiable bone disease.[17,26,85]

The practical problems regarding the correct prophylactic dosage of vitamin D and minerals for preterm babies therefore still remains. At present the US Academy of Pediatrics advises that the prevention of severe bone disease in the preterm baby appears to rely on at least vitamin D 500 IU/d as well as a high oral intake of minerals.[127] Although there is no evidence at present that vitamin D metabolites should be administered to preterm babies routinely,[127] more research into this important field is required before these vague speculations can be replaced by sound scientific reason.

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Bicycle accident injuries

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Summary

Bicycle accidents in 210 patients are analysed. Ages ranged from 1 to 59 years (mean 14,5 years) with a male predominance. In 52% of patients there was a head or facial injury, 6% being moderate to severe. Of the fractures 64% involved the upper limb, 32% being of the radius and ulna and 22% of the clavicle. The majority of abrasions and soft-tissue injuries involved the limbs.

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Cycling is practised by young and old for exercise, transport and as a formal sporting activity, but the vulnerability of the cyclist is often underestimated.[1,2] Injuries sustained in cycling accidents through error of judgement, environmental circumstances or negligence by other road users are reviewed. The impression gained while on duty in the Trauma Unit of Tygerberg Hospital, Cape Town, that these accidents were common, prompted this investigation.

Patients and methods

The trauma records for the period January 1984 - June 1985 were retrospectively reviewed. All white patients admitted with injuries sustained while cycling were included in the analysis. Patients certified dead on arrival at the hospital were excluded.

Factors assessed were age, sex, whether another vehicle was involved in the accident, the nature of the injuries sustained, the number of radiological investigations required, the number of specialist consultations, and whether in-patient admission was required.

The injuries were classified as: skin lacerations, abrasions, soft tissue contusions, fractures, head injuries (skull and intracranial), dental injuries, and internal organ (other than the brain) injuries. The injuries were graded according to the Abbreviated Injury Scale (AIS) as accepted by the Joint Injuries Scaling Committee of the American Medical Association and the American Association for Automotive Medicine and based on Baker's Injury Severity Scale: grade 1 = minor, 2 = moderate, 3 = serious, 4 = severe, 5 = critical and 6 = non-survivable for each body area injured.[2,3]

Results

In the period reviewed 210 cases were seen, with a maximum of 19 in any one month.

Age and sex

Three infants under 2 years of age were seen; all were passengers on bicycles. Ages ranged from 1 to 58 years (mean 14,5 ± 9,36 years) with 55% of patients being under 13 years of age. Eighty-one per cent were males and 19% were females.

TABLE I. AGE AT TIME OF ACCIDENT

Age (yrs)	No. of cases	% of total
1 - 5	25	11,9
6 - 10	40	19,1
11 - 15	91	43,3
15 - 20	26	12,4
21 - 58	28	13,3
Total	210	100

Type of accident

In 85% of the accidents only the cyclist was involved; in 15% there was a collision with another vehicle. In 13 patients the injuries were sustained by contact between the feet and the wheel spokes.

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