

2. STRASSER EJ, DAVIS RM, MENCHEY JM: Lightning injuries. *J Trauma* 1977; 17: 315-319
3. COLEMAN TH: Deaths by lightning. *Penn Med* 1969; 72: 56-58
4. Death by lightning (E). *Lancet* 1977; 1: 230
5. YOST JW, HOLMES FF: Myoglobinuria following lightning stroke. *JAMA* 1974; 228: 1147-1148
6. HANSON GC, MCILWRAITH GR: Lightning injury: two case histories and a review of management. *Br Med J* 1973; 4: 271-274
7. BURDA CD: Electrocardiographic changes in lightning stroke. *Am Heart J* 1966; 72: 521-524
8. CURRENS JH: Arterial spasm and transient paralysis resulting from lightning striking an airplane. *J Aviat Med* 1945; 16: 275-277
9. KLEINER JP, WILKIN JH: Cardiac effects of lightning stroke. *JAMA* 1978; 240: 2757-2759
10. KLEINOT S, KLACHKO DM, KEELEY KJ: The cardiac effects of lightning injury. *S Afr Med J* 1966; 40: 1141-1143
11. CRITCHLEY M: Neurological effects of lightning and of electricity. *Lancet* 1934; 1: 68-72
12. CHIA BL: Electrocardiographic abnormalities of congestive cardiac failure due to lightning stroke. *Cardiology* 1981; 68: 49-53
13. REES WD: Pregnant women struck by lightning. *Br Med J* 1965; 1: 103-104
14. SHARMA M, SMITH A: Paraplegia as a result of lightning injury. *Br Med J* 1978; 2: 1464-1465
15. MORGAN ZV JR, HEADLEY RN, ALEXANDER EA, SAWYER CG: Atrial fibrillation and epidural hematoma associated with lightning stroke; report of a case. *N Engl J Med* 1958; 259: 956-959
16. TAUSSIG HB: "Death" from lightning — and the possibility of living again. *Ann Intern Med* 1968; 68: 1345-1353

## Nutritional rickets in vegetarian children

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Vitamin-D-deficiency rickets, once the most common metabolic bone disease, is now rare in North America.<sup>1</sup> However, this decade has seen a resurgence of the problem.

For many years much of the reported nutritional rickets has occurred in infants who did not receive vitamin D supplementation.<sup>2</sup> Whether human breast milk contains sufficient vitamin D for normal term infants has recently been a controversial issue.<sup>3-6</sup>

Vegetarian children are susceptible to vitamin D deficiency. In its extreme form vegetarianism is often motivated by cultural convention or religious dictates, and vegetarian parents frequently apply their dietary rules to their children. The omission of vitamin-D-fortified milk from the infant's diet, coupled with failure to provide vitamin D supplements, has resulted in an increase in the number of reported cases of nutritional rickets in infants and children.<sup>7-10</sup> We describe three children in whom rickets developed after prolonged periods of strict vegetarian diets. One of the children also had anemia due to vitamin B<sub>12</sub> deficiency.

### Case reports

#### Case 1

A 3½-year-old black boy of Ja-

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maican parents was referred to the outpatient department of our hospital because of genu valgum, first noted by his mother 6 months previously. She had delayed seeking medical advice until the child's gait became awkward and he began to fall frequently.

The boy had been born at term after an uneventful gestation, and the developmental milestones had been normal. Tooth eruption had started at 9 months of age. His mother, who was a strict vegetarian, breast-fed him for 1 year and then started giving him a purely vegetarian diet,\* excluding milk and milk products, meat, fish and eggs. He had never been given vitamin supplements.

The child's height was 98.5 cm (50th percentile), weight 15.5 kg

(50th percentile) and head circumference 53.5 cm (98th percentile). He had normal teeth with no enamel hypoplasia. The fontanelles were closed and there was frontal bossing. The child had enlarged costochondral junctions and wrists, and marked genu valgum, with an 11-cm separation of the medial tibial malleoli. Pertinent laboratory and roentgenologic data are given in Table I.

#### Case 2

A 1½-year-old black girl of Jamaican origin was seen in the outpatient department of our hospital because of vomiting. She was admitted for investigation when pallor of the mucous membranes was noted and a blood count confirmed the presence of anemia.

The patient had been born at term after a normal gestation, and the developmental milestones were within normal limits. She had started walking at 13 months of age. In

\*A typical daily diet was as follows: *breakfast*, bitter or mint tea, orange or fruit juice, and rice or vegetable soup; *lunch*, potatoes or plantain, and coconut "milk"; *dinner*, green leafy vegetables, green beans or carrots or cabbage, and rice or potatoes or yams.

Table I—Laboratory and roentgenologic data for three patients with rickets due to vegetarian diets

Finding	Normal values	Case 1	Case 2	Case 3
<b>Plasma level</b>				
Calcium (mg/dl)	9.5-10.5	7.0	8.8	7.5
Inorganic phosphate (mg/dl)	3.7-6.5	2.1	1.1	2.2
Alkaline phosphatase (IU/l)	154-341	852	1408	1018
Immunoreactive parathyroid hormone (ng/dl)	< 0.30	0.75	0.70	0.67
25-hydroxycholecalciferol (ng/dl)	8.1-33.2	< 3.0	< 3.0	< 3.0
Generalized aminoaciduria		Present	Absent	Present
Roentgenographic		Normal	Signs of rickets	Signs of rickets

recent months, however, she had become progressively weaker, and by the time of her admission she was no longer making any effort to walk.

The child had been breast-fed from birth. In addition, from the age of 9 months she had been receiving a restricted vegetarian diet (generally similar to that in case 1), with wheat, milk and milk products, meat, fish and eggs excluded. She had never been given vitamin supplements. Both parents were strict vegetarians.

She was pale and lethargic. Her length was 73 cm (3rd percentile), weight 8 kg (3rd percentile) and head circumference 45 cm (25th percentile). The conjunctivas were pale. None of her seven erupted teeth had enamel hypoplasia. The anterior fontanelle was closed and there was frontal bossing. The costochondral junctions, wrists, knees and ankles were enlarged, and she had genu valgum, with a 6-cm separation of the medial tibial malleoli. The abdomen was protuberant and the liver edge soft and smooth, extending 4 cm below the right costal margin. The spleen was enlarged 3 cm below the left costal margin. The hemoglobin level was 6.3 g/dl. The erythrocytes in a smear of peripheral blood showed an abnormal variation in shape and size, some being unusually large, and a bone marrow aspirate contained megaloblasts. The serum vitamin B<sub>12</sub> level was 248 (normally 200 to 900) pg/ml; the serum folate level was normal. Other pertinent laboratory and roentgenologic data are shown in Table I.

### Case 3

A 2<sup>3</sup>/<sub>4</sub>-year-old girl, the only sibling of the patient in case 2, was seen because of genu valgum within a few days of her sister's admission. Her parents indicated that she had had an episode of tetany within the previous week.

The child has been born at term after a normal gestation. She had been breast-fed and given vitamin supplements for 1 year, and then had begun receiving a strictly vegetarian diet (generally similar to that in case 1), consisting of legumes, nuts and fruit juices and excluding wheat, milk and milk products, meat, fish and eggs. The diet was

not supplemented with vitamin preparations.

She was thin and had severe genu valgum. Her height was 91 cm (25th percentile), weight 12.3 kg (10th percentile) and head circumference 48.5 cm (50th percentile). There was no enamel hypoplasia. The anterior fontanelle was closed. The costochondral junctions and wrists were enlarged, and the distance between the medial tibial malleoli was 11 cm. Pertinent laboratory and roentgenologic data are reported in Table I.

### Treatment and outcome

All the children responded to therapy with vitamin D<sub>3</sub> (2000 or 3000 IU/d for 3 months) and the addition of milk to their diet: the plasma levels of calcium, inorganic phosphate, alkaline phosphatase, immunoreactive parathyroid hormone and 25-hydroxycholecalciferol became normal, and the aminoaciduria and rachitic lesions disappeared. In case 2 vitamin B<sub>12</sub> therapy (500 µg of the vitamin given intramuscularly) corrected the megaloblastic anemia.

### Discussion

A balanced diet denotes the intake of a variety of food containing proteins, minerals and vitamins in sufficient quantity to meet daily nutrient needs for growth and metabolism. The minimum intake of vitamin D necessary to prevent rickets may be about 100 IU/d.<sup>11</sup> Some studies have suggested that breast milk contains more than 400 IU/l of vitamin D in water-soluble form.<sup>12,13</sup> However, more recent reports refute claims that significant amounts of biologically active vitamin D are present.<sup>14-18</sup> Indeed, bioassays of the lipid extract of human milk show that it contains only about 20 IU/l of antirachitic activity.<sup>14-19</sup> We consider it important to give vitamin D supplements to all breast-fed infants during the first year of life, during which exposure to sunlight is unreliable in temperate regions of the world such as Canada.

Infants of vegetarian parents are at even greater risk of rickets. Although plant foods in appropriate combinations may be a good source of protein, individual vegetables do not provide an adequate blend of

amino acids for long-term nutrition in the human.<sup>7</sup> Pure vegetable diets — those excluding legumes, nuts and grains — are nutritionally inadequate in vitamins B<sub>12</sub> and D, calcium and iron,<sup>8,20</sup> and sometimes in protein.<sup>7</sup> Newborn infants of vegetarian mothers may be poorly endowed with vitamin D, and by the time of weaning they may already have covert vitamin D deficiency. The institution at that stage of a purely vegetarian diet, itself deficient in vitamin D and calcium, will exacerbate the condition and lead rapidly to overt rickets.

In our three patients rickets resulted from multifactorial nutritional deprivation, possibly extending back to fetal life. Since the mothers adhered to strict vegetarian regimens they were probably deficient in both vitamin D and calcium during pregnancy, giving birth to offspring with low stores of vitamin D.<sup>21</sup> Evidence of rickets appeared in the children after prolonged feeding of breast milk and foods devoid of vitamin D, unaccompanied by vitamin supplements.

Rickets might still have been prevented in these children with adequate exposure to sunlight. However, the long winters in our area and the patients' dark skin may have reduced their own production of vitamin D.<sup>22</sup>

Another factor that probably contributed to the development of rickets in these children was their low calcium intake.<sup>23</sup> Although some vegetables contain significant amounts of calcium, many purely vegetarian diets fall short of the recommended dietary intake of this element from vegetables alone.<sup>24</sup> Thus, children following a strictly vegetarian diet are at risk of a combined deficiency of vitamin D and calcium.<sup>25</sup>

As in case 2, a vegetarian diet may also lead to clinically manifest vitamin B<sub>12</sub> deficiency. The infant breast-fed by a strict vegetarian who herself may have vitamin B<sub>12</sub> deficiency can show the consequences of such nutritional deprivation at an early age.<sup>26-28</sup>

Vegetarians can ensure a nutritionally adequate diet by using proper combinations of vegetables, cereals and vitamin-D-enriched milk.<sup>29,30</sup> The advocates of strict veg-

etarian regimens expose themselves and their offspring to the risk of multiple nutritional deficiencies. The parents of our patients were amenable to making minor but essential modifications to their children's diet once the necessity was understood. Others have reported similar responses to their advice.<sup>9</sup>

The responsibility for preventing dietary-induced nutritional deficiencies lies with the physicians and other health care personnel who see parents and their infants at well-baby clinics. They should emphasize the need for vitamin D supplementation for any breast-fed baby who is sheltered from sunlight, and they should caution parents against the exclusive use of any vegetable diet that does not contain vitamins D and B<sub>12</sub>, nutrients that are provided by vitamin-D-enriched milk and such fermented dairy products as yogurt and cheese.

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## References

- HARRISON HE: A tribute to the first lady of public health (Martha M. Eliot). V. The disappearance of rickets. *Am J Public Health* 1966; 56: 734-737
- FRASER D, KOOH SW, SCRIVER CR: Hyperparathyroidism as the cause of hyperaminoaciduria and phosphaturia in human vitamin D deficiency. *Pediatr Res* 1967; 1: 425-435
- Committee on nutrition, American Academy of Pediatrics: Vitamin and mineral supplement needs in normal children in the United States. *Pediatrics* 1980; 66: 1015-1021
- BIKBECK JA, SCOTT HF: 25-hydroxycholecalciferol serum levels in breast-fed infants. *Arch Dis Child* 1980; 55: 691-695
- ROBERTS CC, CHAN GM, FOLLAND D, RAYBURN C, JACKSON R: Adequate bone mineralization in breast-fed infants. *J Pediatr* 1981; 99: 192-196
- GREER FR, SEARCY JE, LEVIN RS, STEICHEN JJ, STEICHEN-ASCHE PS, TSANG RC: Bone mineral content and serum 25-hydroxyvitamin D concentrations in breast-fed infants with and without supplemental vitamin D: one year follow-up. *J Pediatr* 1982; 100: 919-922
- FINBERG L: Human choice, vegetable deficiencies, and vegetarian rickets. *Am J Dis Child* 1979; 133: 129
- KRAUSE MV, MAHAN KL (eds): *Food, Nutrition and Diet Therapy*, 6th ed, Saunders, Philadelphia, 1979: 174
- ZMORA E, GORODISCHER R, BAR-ZIV J: Multiple nutritional deficiencies in infants from a strict vegetarian community. *Am J Dis Child* 1979; 133: 141-144
- EDIDIN DV, LEVITSKY LL, SCHEY W, DUMBOVIC N, CAMPOS A: Resurgence of nutritional rickets associated with breast-feeding and special dietary practices. *Pediatrics* 1980; 65: 232-235
- Committee on dietary allowances, National Research Council: *Recommended Dietary Allowances*, 8th rev ed, National Academy of Sciences, Washington, 1974
- SAHASHI Y, SUZUKI T, HIGAKI M, TAKAHASHI M, ASANO T, HASEGAWA T, MIYAZAWA E: Metabolic activities of vitamin D in animals. VI. Physiological activities of vitamin D sulfate. *J Vitam (Kyoto)* 1967; 13: 37-40
- LAKDAWALLA DR, WIDDOWSON EM: Vitamin-D in human milk. *Lancet* 1977; 1: 167-168
- LEERBECK E, SPØNDERGAARD H: The total content of vitamin D in human milk and cow's milk. *Br J Nutr* 1980; 44: 7-12
- HOLLIS BW, ROOS BA, DRAPER HH, LAMBERT PW: Occurrence of vitamin D sulfate in human milk whey. *J Nutr* 1981; 111: 384-390

- NAGUBANDI S, LONDOWSKI JM, BOLLMAN S, TIETZ P, KUMAR R: Synthesis and biological activity of vitamin D<sub>3</sub> 3B-sulfate. Role of vitamin D<sub>3</sub> sulfates in calcium homeostasis. *J Biol Chem* 1981; 256: 5536-5539
- REEVE LE, DELUCA HF, SCHNOES HK: Synthesis and biological activity of vitamin D<sub>3</sub>-sulfate. *Ibid*: 823-826
- GREER FR, REEVE LE, CHESNEY RW, DELUCA HF: Water-soluble vitamin D in human milk: a myth. *Pediatrics* 1982; 69: 238
- HARRIS RS, BUNKER JWM: Vitamin D potency of human breast milk. *Am J Public Health* 1939; 29: 744-747
- DWYER JT, DIETZ WH JR, HASS G, SUSKIND R: Risk of nutritional rickets among vegetarian children. *Am J Dis Child* 1979; 133: 134-140
- HILLMAN LS, HADDAD JG: Human perinatal vitamin D metabolism. I: 25-hydroxyvitamin D in maternal and cord blood. *J Pediatr* 1974; 84: 742-749
- HOLICK MF, MACLAUGHLIN JA, DOPPELT SH: Regulation of cutaneous previtamin D<sub>3</sub> photosynthesis in man: skin pigment is not an essential regulator. *Science* 1981; 211: 590-593
- KOOH SW, FRASER D, REILLY BJ, HAMILTON JR, GALL DG, BELL L: Rickets due to calcium deficiency. *N Engl J Med* 1977; 297: 1264-1266
- DWYER JT, DIETZ WH JR, ANDREW EM, SUSKIND RM: Nutritional status of vegetarian children. *Am J Clin Nutr* 1982; 35: 204-216
- FRASER D, KOOH SW, REILLY BJ, BELL L, DUTHIE D: Human rickets due to combined deficiency of calcium and vitamin D. In DELUCA HF, ANAST CS (eds): *Pediatric Diseases Related to Calcium*, Elsevier, New York, 1980: 257-267
- LAMPKIN BC, SAUNDERS EF: Nutritional vitamin B<sub>12</sub> deficiency in an infant. *J Pediatr* 1969; 75: 1053-1055
- HIGGINBOTTOM MC, SWEETMAN L, NYHAN WL: A syndrome of methylmalonic aciduria, homocystinuria, megaloblastic anemia and neurologic abnormalities in a vitamin B<sub>12</sub>-deficient breast-fed infant of a strict vegetarian. *N Engl J Med* 1978; 299: 317-323
- JOHNSON PR JR, ROLOFF JS: Vitamin B<sub>12</sub> deficiency in an infant strictly breast-fed by a mother with latent pernicious anemia. *J Pediatr* 1982; 100: 917-919
- HARDINGE MG, CROOKS H: Non-flesh dietaries. *J Am Diet Assoc* 1964; 45: 537-542
- VYHMEISTER IB, REGISTER UD, SONNENBERG LM: Safe vegetarian diets for children. *Pediatr Clin North Am* 1977; 24: 203-210

## BOOKS

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**HANDBOOK OF AFFECTIVE DISORDERS.** Edited by E.S. Paykel. 457 pp. Illust. The Guilford Press, New York, 1982. \$50 (US). ISBN 0-89862-62206

**HISTOCHEMISTRY.** An Explanatory Outline of Histochemistry and Biophysical Staining. Richard W. Horobin. 311 pp. Illust. Butterworths & Co. (Publishers) Ltd., Woburn, Massachusetts, 1982. \$69.95 (US). ISBN 0-407-00248-0

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**IN HER OWN WORDS.** Oral Histories of Women Physicians. Contributions in Medical History, Number 8. Edited by Regina Markell Morantz, Cynthia Stodola Pomerleau and Carol Hansen Fenichel. 285 pp. Illust. Greenwood Press, Westport, Connecticut, 1982. \$29.95 (US). ISBN 0-313-22686-5

**INTERNATIONAL PERSPECTIVES IN UROLOGY.** Volume 3. Prostate Cancer. Edited by Günther H. Jacobi and Rudolf Hohenfellner. 490 pp. Illust. Williams & Wilkins, Baltimore, Maryland, 1982. \$70 (US). ISBN 0-683-04354-4

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