South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 16 June 1962 Volume 36 No. 24 Deel 36 Kaapstad, 16 Junie 1962

EDITORIAL : VAN DIE REDAKSIE

RICKETS IN SOUTH AFRICA

Two recent papers[1a,2] have indicated that infantile rickets is still common in South Africa, at least in the Bantu children around Johannesburg and in the mixed races in the Cape Peninsula. Dancaster and Jackson[1] found that the prevalence of rickets in a Coloured outpatient population between the ages of 3 and 12 months was certainly over 30% and possibly as high as 80%, depending upon the diagnostic criteria selected. The same authors give reasons for considering that the most reliable criterion for the diagnosis of rickets, and for following its progress, is radiological. Thus, some patients with obvious radiological rickets had normal inorganic phosphorus and alkaline-phosphatase levels in their plasma. In other infants with normal radiographic appearances, the alkaline-phosphatase levels were high, but follow-up studies on these showed no further evidence of rickets, even when no prophylactic measures had been taken. Nevertheless, in a more recent paper, Dancaster and Jackson[1d] indicated that a raised serum-alkaline-phosphatase level was found to be the best biochemical index of activity, and this is in agreement with Wayburne and Dean.[2] A depression of the serum-phosphorus level was less constant, and this did not always rise after radiographic healing of the rickets, while the serum-calcium level was depressed in only half the cases of active rickets. Even the calcium times phosphorus product was a poor indication of activity of the rachitic process.

Why should rickets occur so frequently in such a sunny country? Dancaster and Jackson[1b] concluded that, among the various possible aetiological features, the most significant difference between a rickety and a normal group of children was the actual exposure to sunlight, which was very significantly less in the rachitic group. This finding corresponded well with the seasonal incidence of rickets and the fact that most of the affected children were born in late summer and autumn. Another factor which appeared to be of some importance was the period of breast feeding. Although some completely breast-fed infants developed rickets, there was a probably significant increased likelihood of this disease in children who had been on the breast for less than three months. It is curious that this should be so, since both the calcium and the phosphorus content of breast milk is less than that of cow's milk, and breast milk contains very little vitamin D.

Although it is generally accepted that premature infants are more liable to rickets, this was not observed in the series of Dancaster and Jackson. Nor did the actual intake of calcium appear to have any relation to the development of rickets. In this series there were nine patients in whom all the factors considered were favourable, and yet rickets had developed — these cases appear to constitute a mystery and suggest that yet other factors may be operating. One of these might be the level of the phosphate intake, although this is unlikely.

One further factor to consider is the hereditary one, or the possibility of considerable individual variations in requirement of and susceptibility to vitamin D. There would, in fact, appear to be a wide range of variation in otherwise normal children. Thus, at one end of the spectrum we find the hypercalcaemic syndrome, produced by quite small additions of calciferol to staple foodstuffs, occurring particularly in Britain. Next we see children who need little vitamin D to prevent rickets; then those who will develop rickets unless they receive considerable amounts of the vitamin; and at the further end of the spectrum are those children with hereditary vitamin-D-resistant rickets, who need colossal quantities of calciferol (e.g. 100,000 units daily) for healing of their bony lesions. The degree of susceptibility to vitamin D might generally be a hereditary factor, and Dancaster and Jackson noted the frequent occurrence of rickets in several members of single families. While it is difficult to rule out purely environmental factors as the cause of this observation, the importance of heredity in the aetiology of rickets has been strongly supported by Jonxis.[3] However, the discovery of multiple cases of rickets in a family, the appearance of rickets over the age of 3 years, or the lack of healing on usually adequate doses of vitamin D, all indicate that careful consideration should be given to the possibility of true vitamin-D resistance, possibly in combination with the Fanconi syndrome or renal tubular acidosis.[1c]

Although the incidence of overt rickets in kwashiorkor does not seem high, Dancaster and Jackson found no support for the popular idea that undernutrition protects against active rickets. In fact, both their evidence and that of Wayburne and Dean clearly indicate that lack of growth affords no protection against rickets whatever.

Two points of radiological interest are worth mentioning. In several cases, including some of the most severe with no other indication of healing, a doubling of the outline of the cortex of long bones and of metacarpals was seen[1d,2] — presumably representing partially calcified subperiosteal osteoid tissue. This phenomenon should probably be included as a part of the characteristic radiographic findings in active rickets and should no longer be considered to represent either scurvy or healing of the rickets. Secondly, the considerable retardation of bony development in active rickets was frequently shown by non-vizualization of carpal and other ossific centres. It may be debated whether this indicates actual delay in formation of the centres or simply their almost total lack of calcification.[1d,2] That the latter might be the case is suggested by the speed with which quite large centres appear after treatment with vitamin D in some instances.

The response to therapy in Dancaster and Jackson's patients (reported in the final part of their series on 'Studies in Rickets in the Cape Peninsula' on p. 479 of

this issue of the *Journal*) strongly supported the idea of individual variation in susceptibility to vitamin D. Three children who did not have 'resistant rickets' in the true sense, failed to show any healing after a single intramuscular dose of 600,000 units. A practical point of importance was that the hake liver oil used did not appear to be as potent as would have been expected from its reputed vitamin-D content, so that it seems that much larger doses must be given than generally recommended if this is used as therapy.

Finally, as with so many other diseases, we may rather plaintively ask (with Queen Victoria) 'if preventable, why not prevented?' The prevention of rickets could be largely achieved (though not entirely) even without fortification of foodstuffs with vitamin D and without additional vitamin D being supplied to infants. The proper exposure of young children to sunlight or sunshine would appear to be the most important prophylactic measure, which surely indicates the need to educate the mothers in this respect.

1. (*a*) Dancaster, C. P. and Jackson, W. P. U. (1960): S. Afr. Med. J., **34**, 776.
(*b*) *Idem* (1961): *Ibid.*, **35**, 890.
(*c*) *Idem* (1961): *Ibid.*, **35**, 1057.
(*d*) *Idem* (1962): *Ibid.*, **36**, 364.
2. Wayburne, S. and Dean, R. F. A. (1960): S. Afr. J. Lab. Clin. Med., **6**, 21.
3. Jonxis, J. H. P. (1955): Glasg. Med. J., **36**, 227.