

by haemagglutination inhibition was not obviously greater in the 17-→28 weeks group than that found in the 6-16 weeks group (Table I). It is well known that the excretion rate of H.C.G. in urine falls to a low level late in pregnancy. However, it has been observed by Wide and Gemzell (1962) that H.C.G. can exist in a physiologically inactive form in which it still combines with antibody, and that the apparent excretion rate of H.C.G., titrated by immunoassay, late in pregnancy is much greater than would be expected from past work carried out by bioassay. This could well explain the unexpected success of immunodiagnosis at late stages of pregnancy. It is of interest in this connexion that in those tests where a positive result was obtained by haemagglutination inhibition, and a negative result by the toad test method, there was apparently no reason to suppose that the levels of H.C.G. were unusually low (Tables V and VI). It may be that in these cases the proportion of physiologically active H.C.G. in the samples was low. In the light of Wide and Gemzell's observations with heated H.C.G. it is interesting that potassium periodate, which has been used to destroy non-specific inhibitors of agglutination in serum samples before assay for H.C.G., is also known to destroy the physiological activity of the hormone; it did not, however, affect the hormone's antibody-combining power in these tests.

Summary

A one-stage haemagglutination inhibition test for pregnancy has been described. Of 126 clinically confirmed pregnancies 124 were positive by this test, and there was no evidence of loss of accuracy even at a late stage of pregnancy. Non-pregnant controls totalling 211 individual samples from persons aged 17 to 59 were all negative by this test. Tests were carried out on 388 samples submitted for pregnancy diagnosis by the male-toad test and the results compared at a later date when clinical confirmation could be obtained. In 350 out of the 388 samples tested the results by haemagglutination inhibition and by the toad test were in agreement. The 38 discrepant results consisted of 12 tests where the toad test was positive and the haemagglutination inhibition test negative; of these subjects, seven were pregnant and five not pregnant on subsequent clinical examination. A further 26 tests were positive by the haemagglutination inhibition test and negative by the toad test, and clinical examination confirmed that 23 of these patients were in fact pregnant; three patients could not be traced. If it is assumed that those results in which both the toad test and the haemagglutination inhibition test were positive were correct, the overall accuracy of the haemagglutination inhibition method was 98.2% and the accuracy of the male-toad test 92.8%.

Although the accuracy of this test for the diagnosis of pregnancy was of a high order, quantitative assay of urine samples in parallel with bioassay was unsatisfactory. This could be due either to the presence of differing proportions of hormonally inactive but immunologically active H.C.G. in the different samples or to interference by non-specific antigens and antibodies.

It has been found that a biologically inactive form of H.C.G. produced by treatment of H.C.G. in solution with potassium periodate retained its original capacity to combine with antibody.

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RICKETS IN IMMIGRANT CHILDREN IN LONDON

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We wish to draw attention to the prevalence of rickets among the children of certain immigrant communities living in London. All the cases of active nutritional rickets seen during the last five years in the paediatric departments of Guy's and King's College Hospitals are reported. There were 16 cases, and it is of great interest that only one was English; six were West Indian, five Greek Cypriot, one Nigerian, one Maltese, one Irish, and one Spanish. All the children were born in England except two, both of whom had lived in London for at least a year before rickets was diagnosed. Ten were girls and six boys. Their ages when first seen ranged from 9 to 38 months. All had clinical evidence of rickets, including skeletal deformity. Seven presented with bowing of the legs (Fig. 1), two with fractures, and seven with unrelated conditions.

Though no normal standards of body weight are available for non-indigenous children in London, a low level of nutrition in some of our cases was suggested by a body weight below the average for English children. Eight out of 14 whose weights were recorded were below the tenth percentile (Tanner and Whitehouse, 1959). A poor nutritional status was also suggested by the low haemoglobin levels, which in 11 out of 16 cases were between 4.5 and 10.5 g./100 ml.

All the children had biochemical and radiological evidence of active rickets. Serum calcium concentration

(see Table) was normal (9–11 mg./100 ml.) in 11 cases and low (less than 9 mg./100 ml.) in four. Serum inorganic phosphate concentration was normal (4–7 mg./100 ml.) in only five cases and low (less than 4 mg./100 ml.) in 11. Serum alkaline phosphatase concentration was considerably elevated (above 20 K-A units/100 ml.) in all cases except one, in which long delay occurred between taking the sample of blood and performing the estimation.

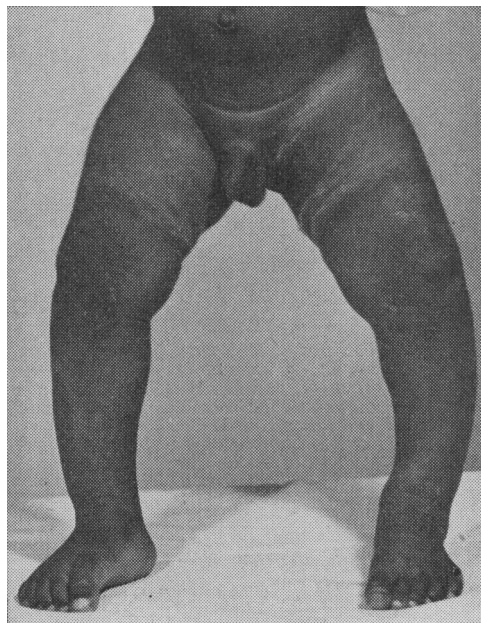


FIG. 1.—Bowling of legs.

Biochemical Investigations in 16 Cases of Rickets

Case No.	Nationality	Sex	Age at Onset (Months)	Serum Calcium (mg./100 ml.)	Serum Phosphate (mg./100 ml.)	Serum Alkaline Phosphatase (K-A Units/100 ml.)	Haemoglobin (g./100 ml.)
1	Greek Cypriot	F	19	10.8	4.5	46	6.4
2	"	F	13	7.6	6.8	47	9.5
3	"	F	29	10.9	2.5	84	4.7
4	"	F	15	—	3.4	37	12.9
5	"	M	27	9.7	3.2	19	11.3
6	Maltese	F	20	9.3	3.4	104	9.3
7	West Indian	F	19	8.6	1.9	184	10.2
8	"	F	21	9.0	1.7	125	9.5
9	Nigerian	F	14	8.4	1.8	155	11.2
10	Spanish	F	16	8.8	2.7	58	10.2
11	West Indian	M	9	9.1	3.6	55	9.0
12	English	M	30	9.2	3.2	75	9.5
13	West Indian	F	10	9.4	4.1	70	10.3
14	Irish	M	26	9.2	3.7	71	14.3
15	West Indian	M	38	9.3	4.2	78	9.5
16	"	M	24	9.3	5.2	71	12.3

Fractures occurred in four cases. In one there were complete fractures of both ulnae and one radius (Fig. 2). In the other three there were greenstick fractures of radius, ulna, and femur respectively.

The prompt healing on administration of vitamin D, which in the majority was given orally in a dosage of 3,000 units daily, confirmed the nutritional aetiology. In the remainder, who were given an initial dose of vitamin D by injection, failure to relapse on a normal diet excluded any vitamin D resistance.

In the dietary history of these children there were two outstanding features: (1) prolonged feeding with breast or cows' milk, and (2) failure to give either vitamin supplements or dried cows' milk fortified with vitamin D.

The social background of the cases was difficult to determine from available records, but it was of interest that in the cases of the Irish and the English child home conditions and standard of care were very poor. The English family had come under the attention of the National Society for the Prevention of Cruelty to Children.

Discussion

Nutritional rickets, once a common disease in this country, had virtually disappeared. From personal experience the few cases seen had either a poor social background or inadequate parents. However, a recent survey of the Pakistani community in Glasgow by Dunnigan *et al.* (1962) uncovered 35 subjects with active osteomalacia or rickets among 74 adults and children examined. Aetiological factors in the Glaswegian cases were similar to those present in our series although there were certain interesting differences.

Pigmented Skin

When sunlight is limited and vitamin D intake marginal, dark-skinned children are more likely to develop rickets than light-skinned children. This is probably due to the absorption of ultra-violet light by melanin pigment in the skin, with consequently less production of vitamin D. It has been suggested (Jonxis, 1961) that this disadvantage led to the disappearance of dark-skinned races from temperate zones in bygone years. Even where sunlight is plentiful, as in Cyprus, a general paediatrician may still see about 10 to 15 cases of nutritional rickets in a year (C. Joannides, personal communication, 1962). These cases occur either during the winter or when sunlight is partially excluded, as, for example, in mountainous districts where children are well wrapped up and stay indoors. On the other hand, nutritional rickets is very rare in the West Indies, where the favourable climate encourages an open-air life for

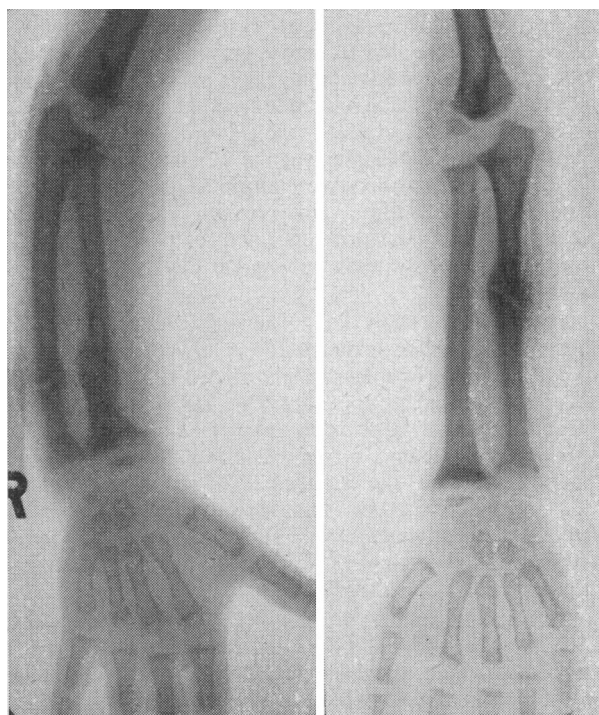


FIG. 2.—Case 15. Radiograph showing active rickets with fractures of radius and ulna.

the children all the year round (B. Symmonds, personal communication, 1962).

Vitamin D is now freely available, and dark-skinned people in temperate areas should no longer develop rickets. In our series 14 of the 16 children had a degree of skin pigmentation well above that of indigenous children.

Prolonged Milk-feeding

In the Cypriot and West Indian children prolonged feeding with cows' milk or breast milk was usual and led to gross dietary deficiency of vitamin D. There is a large variation in the vitamin content of milk. For instance, a child fed on 2 pints of cows' milk a day would receive from 3 to 45 international units of vitamin D, and on 2 pints of breast milk from 4 to 100 units (Macy *et al.*, 1953).

Prolonged breast-feeding is the accepted cultural method of feeding babies in many underdeveloped countries. Indeed, wherever infantile malnutrition is a problem, prolonged breast-feeding should be encouraged (Jelliffe and Dean, 1959), but in this country it may have disadvantages.

Unsatisfactory Welfare Clinic Attendance

It is by attending welfare clinics that the majority of children in this country obtain vitamin D supplements. For the Greek Cypriot mothers attendance at welfare clinics is often unsatisfactory because of language difficulties even in families resident in Britain for many years. Although many West Indian mothers attend welfare clinics regularly and benefit enormously, others go out to work, and their children, who are often looked after by friends or unofficial paid baby-minders, are then not seen at welfare clinics.

There is a striking difference between the age distribution of our cases and those reported by Dunnigan *et al.* (1962). Thus our eldest child was 3 years and 2 months of age, while in the Glasgow series none was below 5 years. However, the Glasgow survey investigated only 14 children below 5 years, and it is possible that a larger sample might have included children with lower vitamin-D intake. Indeed, the Glasgow authors suggest that the absence of rickets in the children under 5 years of age was due to adequate vitamin D provided by foods obtained from welfare clinics which Pakistani families attended. The age difference would probably explain the presence of knock-knees in the Glasgow series and bow-legs in ours.

There was a low incidence of dried-milk feeding among the children reported in this series. Many children in this country do not attend welfare clinics or receive vitamin-D supplements. In such children an important factor in prevention of rickets is the fortification of proprietary forms of dried cows' milk with vitamin D. Thus the regrettable reduction in popularity of breast-feeding in this country may have an unsuspected advantage. The advantages of breast-feeding, however, could still be enjoyed if vitamin-D supplements were administered.

In addition to the problem of vitamin-D deficiency there would appear to be a need for attention to general subclinical undernutrition in the children of our immigrant communities, which was suggested by the low haemoglobin levels and body weights seen in many of our cases.

Summary

All the cases of nutritional rickets seen over the last five years at Guy's and King's College Hospitals are reported. Of 16 patients, all of whom were under 4 years old, 14 originated from non-temperate areas and had a degree of skin pigmentation well above that of children from this country.

Although skin pigmentation was thought to be a major factor, nutritional deficiencies related to cultural variations were also important.

The need for attention to all aspects of nutrition in immigrant children is stressed.

We are grateful to Dr. Mary Wilmers, Mr. C. Catterall, Dr. Doyné Bell, Dr. P. R. Evans, Dr. R. C. Mac Keith, and Sir Wilfred Sheldon, under whose care most of these cases were treated. We would like to thank the department of medical illustration of King's College Hospital for Figs. 1 and 2.

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DIAGNOSIS AND TREATMENT OF SPRAINED ANKLE

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The sprained ankle is probably the commonest minor traumatic condition which the casualty officer and general practitioner is called upon to treat. Often, however, the immediate pain and disability are prolonged unnecessarily, and occasionally permanent harm may result. The fault may be in misdiagnosis, a severe injury being overlooked, and consequently inadequately treated, or it may be due to unsuitable treatment when the sprain is correctly diagnosed.

It is the purpose of this paper to discuss the diagnosis of ankle sprains with particular reference to the exclusion of more serious injury, and to suggest a method of treatment which is more satisfactory than that in common use.

Diagnosis

Injury to the ankle-joint may result in simple sprain with stretching, partial rupture, or partial avulsion of a ligament from its attachment with or without a flake of bone. The joint is stable, the talus remaining firmly in the mortise. More serious injury results in complete rupture or complete avulsion of the ligaments, a fracture, or a combination of these injuries, which may result in immediate instability or give rise to instability if treatment is inappropriate.

The diagnosis of a simple sprain is therefore a matter of excluding a more extensive injury to ligaments and bone. The latter can be excluded by x-ray examination,

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