in Asia, these manifestations of vitamin D deficiency have not been seen in Britain for many years, and unfamiliarity with their clinical presentation may delay diagnosis.

The paediatrician may encounter Asian rickets as hypocalcaemic tetany which does not respond to the administration of calcium supplements or in the infant of an Asian mother who does not use welfare foods. The general practitioner or school doctor may encounter it in an adolescent who complains of painful legs when walking, running, or doing gymnastics; physical examination may show the genu valgum characteristic of "knock-kneed" late rickets in severe cases. The obstetrician may meet osteomalacia in a patient with thoracic, back, and limb pains, who has bony tenderness over the rib cage and elsewhere; the general physician or general practitioner may encounter a similar clinical picture in Asian patients of any age.

The condition is often missed at an early stage—as reports of osteotomies for rachitic deformity in Asian children testify.[3] Once considered, the diagnosis is easily confirmed by finding low serum concentrations of calcium or inorganic phosphate combined with a raised alkaline phosphatase. In severe cases radiological evidence of rachitic epiphyses and the typical pseudofractures of osteomalacia may be found combined with bony deformity. Treatment is simple: patients respond readily to 2-3000 units of vitamin D daily or to a single intramuscular injection of 600 000 units.

The reasons for the prevalence of Asian rickets are not clear. The dietary vitamin D intakes of many children with Asian rickets are similar to those of White or West Indian children, who are unaffected beyond infancy.[9-11] Ultraviolet deprivation has been suggested,[12,13] but measurements of the outdoor exposure of Asian children have shown no differences from White controls.[14] The consumption of high-extraction chapati flour by most Asians has been blamed,[15,16] but the mechanism by which chapati flour might produce vitamin D deficiency remains obscure. Phytic acid,[15,16] phytate-derived inositol polyphosphates,[17] dietary phosphorus,[14] or malabsorption of vitamin D induced by a diet high in phytate and vegetable fibre[18] have all been suggested. Finally, genetic differences in intermediary vitamin D metabolism may be partly responsible.[7,19]

Patients with Asian rickets show low serum concentrations of 25-hydroxy-vitamin D,[20] and 400 units of vitamin D daily will restore these to normal,[21] providing effective prophylaxis. The problem for the preventive health services is to ensure that the population at risk gets this simple prophylactic measure. Clinical cases of rickets and osteomalacia are the tip of an iceberg of hypovitaminosis D—which is effectively the whole Asian population. The record of the last 14 years shows that exhortations to Asians to consume more vitamin D-containing foods such as oily fish or margarine are unlikely to be effective, and more vigorous action is needed. Welfare services for preschool children are in general well organised, but there is no evidence of any concerted action on the part of school health services to provide vitamin D supplements for Asian schoolchildren: 3000 units of vitamin D weekly would be sufficient. The adult population is more difficult to reach, but much more could be done by community health teams to distribute information on the need for vitamin D supplements to Asian families through Indian and Pakistani social and cultural associations.

The simplest way to eliminate vitamin D deficiency from the Asian community is to fortify a foodstuff consumed exclusively by most of the population at risk. Vitamin D-fortified chapati flour will raise levels of serum 25-hydroxy-vitamin D to normal and heal the rickets of those consuming it.[21] Fortification is technically straightforward, and the additional cost could be minimal. The introduction of this method of prophylaxis should be considered urgently by the Committee on Medical Aspects of Food Policy. A study in Bradford[21] has indicated that as many as 1 in 40 Asian children may expect admission to hospital with rickets before 16 years. Many Asian children have rachitic deformities. These are distressing reminders of a failure of the preventive medicine services. Further delay in introducing effective prophylactic measures is difficult to defend.

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Replacement of the knee joint

Replacement of the knee joint is still at an early stage of development, unlike the operation on the hip that has now become routine. Yet the demand equals that for the hip, and since arthrodesis of the knee leads to severe disability[1] the need may be as great if not greater.

There are four indications for replacement of any joint—pain, instability, stiffness, and deformity. In the case of the knee pain and instability are the most important. Stiffness alone would not be accepted as grounds for operation by most surgeons unless it was associated with stiffness of other joints and these contributed to severe incapacity, and deformity would warrant surgery only if it was associated with instability or pain.

A suitable prosthesis must relieve pain, simulate the normal joint movements, provide stability, and correct deformity. In addition arthrodesis should be possible if the prosthesis has to be removed later. The hinge joints used in the 1950s and early 1960s had disadvantages in that they allowed movement around a fixed axis only and therefore tended either to break or to become loose. Their relatively large size meant that so much bone had to be excised at insertion that in the event of a failure arthrodesis was difficult or impossible. The two-piece prostheses which came next were smaller and depended on the patient's own ligaments for stability, but they were not suitable when gross joint destruction or instability was present. A recent advance has been the concept of a two-piece prosthesis with a mechanical link to give stability. Such a joint allows the gliding movements of flexion and extension with some rotation and lateral mobility, as in the normal knee.[2] The mechanical link acts in place of the

cruciate ligaments and supplements the collateral ligaments. This prosthesis is therefore suitable for use in patients in whom gross joint destruction has occurred.

The early results from the use of this implant have been encouraging. In a series of 53 operations over half the patients had no pain after the operation and almost all had only a slight ache or no pain. Walking was greatly improved, and the range of movement increased in most of those joints where stiffness was present before operation. No prosthesis became loose, but one had to be removed because of infection, and the knee was then successfully arthrodesed. Sheehan,[3] using a prosthesis of similar type, has also reported very satisfactory results in a series of 45 arthroplasties.

As with all new procedures enthusiasm must be tempered with caution. In younger patients with osteoarthrosis of the knees tibial osteotomy is still the operation of choice. In elderly patients, however, whose independence is threatened by advanced bilateral osteoarthrosis, knee replacement may allow normal life to continue at home. Patients with rheumatoid arthritis in whom a number of major joints may be destroyed derive particular benefit from joint replacement. Not only is pain relieved but mobility is restored to an immobile joint, relieving strain on other regions of the limbs. The reported series so far are small, but there are indications that these encouraging results are being maintained. If the promise is fullfilled, the prospect of relief from pain and immobility now available to patients with hip disease may lead to a similar demand for treatment from those with arthritis of the knee.

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An ulcer in the family

It is always frustrating when no satisfactory explanation can be found for a biological phenomenon. Yet such is the case in the link between blood groups and duodenal ulcers. The observation[1] by Aird *et al* that such ulcers were commoner in patients of blood group O than would be expected from the population distribution of blood groups has been confirmed all over the world. Why should this be? Do patients with some blood groups secrete more or different digestive juices than others? Does one blood group (or the blood-group substances secreted in digestive juices) confer better mucosal resistance to ulceration than another? Or are there no direct links with blood groups themselves but simply pleiotropic effects of the ABO genes? It has even been suggested[2] that the gene for duodenal ulceration enhances ABO incompatibility between fetus and mother, the O group fetuses being spared because they are invariably compatible. There are weaknesses in all these theories, but new information is constantly appearing and may change our perspective.

Duodenal ulcers in children have always been perplexing because of their rarity and, like other non-infectious diseases in the young, because their occurrence raises the question of a genetic predisposition. This possibility seems to be supported by the strong positive family history in most such children. One careful study of children with duodenal ulcers in Newcastle upon Tyne[3] confirmed the high family incidence, but surprisingly there was no excess of blood group O in the patients, a finding at variance with other analyses of childhood duodenal ulcers, where group O seemed to predominate.[4–6] Some recent work in Hong Kong[7] has concentrated on the age of onset of duodenal ulcer symptoms. Patients whose symptoms began in the fourth decade or later had the expected preponderance of blood group O, but those with an earlier onset contained more with groups A, B, and AB. When analysed by age patients of blood group O tended to be older than those of group A, B, and AB. In this series at least the well-known blood group O predominance seemed to apply only to late-onset duodenal ulcers. About half the early-onset patients had a positive family history, compared with less than a fifth of the late-onset ones, and this applied regardless of blood groups. Now it is to be expected that the occurrence in his family of any particular disease will be better known by a patient who suffers from the condition than by an unaffected person.[8] This trend may be all the more definite when the disease occurs unusually early in life. But it seems unlikely that this is the explanation for the findings from Hong Kong. The somewhat revolutionary conclusion reached by Lam and Ong is that possession of group O delays the onset of duodenal ulceration, implying a protective effect, in contrast to the common assumption that the ownership of group O somehow weakens defences against the development of an ulcer. Nevertheless, this defensive role of group O would explain the eventual overall preponderance of group O ulcer patients only if the young ones failed to survive. In fact none of them died.

An alternative explanation would be that, while all duodenal ulcers may have a genetic element in their aetiology, only the late onset ones are related to blood group inheritance, whereas the early onset ulcers with their strong family history have other inherited risk factors—one of which might be hypersecretion of acid.[9 10]

Another feature noted in the Hong Kong patients was that over half of them who had gastrointestinal bleeding were of blood group O. This preponderance has been observed elsewhere,[9–13] but in this study it was explained entirely by the incidence in the late-onset patients, in whom 64% of those who bled were group O. In the early-onset patients who bled group O showed no excess, despite the fact that in 88% of them bleeding had been the presenting symptom.

Studies of inherited factors in common diseases are notoriously difficult, not least because of the inevitable difficulty in properly matching the requisite large number of patients and controls. Unconscious bias may all too easily creep in. Nevertheless, the convoluted genetic influences which may render us liable to duodenal ulceration are slowly being unravelled. Despite that, we are still a long way from applying such knowledge to prevention of this modern epidemic disease, which McConnell[14] rightly reminds us must have environmental factors as its prime causes. Paradoxically, it may be genetic studies that will lead us to identify them.

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