Delay in skeletal maturity in Malawian children

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The atlas of Greulich and Pyle for skeletal maturity and epiphyseal closure is widely used in many countries to assess skeletal age and to plan orthopaedic surgery. The data used to compile the atlas were collected from institutionalised American children in the 1950s.

In order to determine whether the atlas was relevant to sub-Saharan Africa, we compared skeletal age, according to the atlas, with chronological age in 139 skeletally immature Malawian children and young adults with an age range from 1 year 11 months to 28 years 5 months. The height and weight of each patient were also measured in order to calculate the body mass index.

The skeletal age of 119 patients (85.6%) was lower than the chronological age. The mean difference was $20.0 \pm 24.1$ months ($t$-test, $p = 0.0049$), and the greatest difference 100 months. The atlas is thus inaccurate for this group of children.

The body mass index in 131 patients was below the normal range of 20 to 25 kg/m$^2$.

The reasons for the low skeletal age in this group of children are discussed. Poor nutrition and chronic diseases such as malaria and diarrhoea which are endemic in Malawi are likely to be contributing factors. We did not find any correlation between the reduction in body mass index in our patients and the degree of retardation of skeletal age.

Results

Difference between chronological and skeletal age. Of the 300 patients entered into the study, 161 were excluded as they were skeletally mature and could not have a specific skeletal age assigned. The remaining 139 (93 boys and 46 girls) were skeletally immature and their skeletal age was determined using the atlas of Greulich and Pyle.

Patients and Methods

The atlas of Greulich and Pyle$^1$ comprises a large series of standard anteroposterior radiographs of the hand. Each radiograph is assigned to a specific age in years and months and the patient's skeletal age is determined by comparing his or her radiographs with the standard in the atlas for the appropriate gender.

All patients who attended the radiography department of our hospital for a radiograph of the hand following injury between April and August 2000 and who consented or whose parents consented were registered in the study. The radiographs were divided into skeletally mature, with fully fused epiphyses, and immature, with unfused epiphyses. The chronological age to the nearest month and the height and weight of the individual were recorded.

Skeletal maturity was determined using the atlas by two independent orthopaedic surgeons (WJH, CPL) who were unaware of the chronological age. If the two surgeons differed in their opinion a third surgeon (CBDL) reviewed the radiograph. If all three surgeons differed a meeting was held to discuss the relevant radiographs and an age determined. The age differential was then calculated in months by subtracting the skeletal age from the chronological age. The body mass index (BMI) calculated by dividing the weight in kilogrammes by the height in metres squared, was used as a measure of nutrition. All data were recorded and analysed using Microsoft Excel.
The mean chronological age was 10 years 8 months (13 months to 28 years 5 months). The skeletal age was less than the chronological age in 119 patients (85.6%) (Fig. 1). The mean discrepancy between the chronological and skeletal age was 20 months (+137 to -36) which was statistically significant (p = 0.0049; Fig. 2). The mean age difference for girls was 18.6 months (p = 0.0458) and for boys 20.7 months (p = 0.0157).

Figure 2 shows the distribution of bone age discrepancy for both sexes. If the skeletal age and chronological age were similar the bell curve would be centred over zero; the curve is shifted markedly to the right as skeletal age is less than chronological age in 85.6% of patients.

BMI. In order to investigate whether the relative skeletal immaturity of our patients was related to nutrition we calculated the BMI. The normal value for the BMI is

![Graph of skeletal age against chronological age in months. Most of the points fall below the line indicating a retardation of skeletal age compared with chronological age.](image1)

![Distribution curve of the discrepancy between chronological and skeletal age. The bell curve is shifted markedly to the right as skeletal age is less than chronological age in 85.6% of patients.](image2)

![Graph of the relationship of the number of patients to the normal BMI. Most have a BMI below the normally accepted range.](image3)

![Graph of the BMI against the difference between the chronological and skeletal age (age difference).](image4)
between 20 and 25 kg/m². Most of our patients were well below this. Only eight had a normal BMI (Fig. 3).

**Relationship of BMI to bone age.** In order to determine if there was any association between the BMI and skeletal immaturity we plotted the BMI against the difference in age (Fig. 4). We found no significant correlation (correlation coefficient = 0.0225). Therefore, in this study, although most children had a low BMI, there was no tendency for those with a higher BMI to have a skeletal age which was closer to their chronological age.

**Discussion**

Our study has shown a significant mean reduction in skeletal age in Malawian children compared with the group which was used to compile the atlas of Greulich and Pyle¹ in the 1950s. Few studies have been published on the relevance of this atlas to different populations in the 50 years since it was published. Rikhasor et al² showed that Pakistani children of both genders were a few months in advance of their skeletal development compared with the atlas until puberty, but then fell behind by a few months. Loder et al³ investigated black and white children in the geographical area from which the atlas originated and found minor changes. For example, black girls were skeletally advanced by between four and eight months, compared with the atlas, for most of their growing period. The cause of the markedly reduced skeletal age in our group is not clear. It is tempting to blame poor nutrition and our results showed that the children in our study were undernourished. We were unable, however, to show a relationship between the degree of undernourishment and the degree of skeletal delay. Two other studies of delay in skeletal development in poor communities by Fleshman⁴ and Mackay⁵ also suggested poor nutrition as being the cause of skeletal delay, but they were unable to show statistically significant support for this hypothesis.

If the reduction in skeletal age was entirely due to nutrition it is likely that we would have found a smaller bone age differential in those children with higher BMIs. A similar study on the children of families of higher income would be of interest since they would have a better nutritional status. It is likely that factors other than nutrition are involved in reducing skeletal age, for example, the prevalence of chronic diseases such as malaria and diarrhoea. We did not specifically determine the prevalence of chronic diseases in our patients. It would have been of interest to reassess them from that point of view, especially those with the greatest skeletal delay, but recalling them for review would have been virtually impossible since most of the patients did not have postal addresses or telephones. Nevertheless, the large number of children admitted to our hospital with malaria and diarrhoea suggests that these diseases are endemic among Malawian children.

The finding of a reduced skeletal age in these children has three main implications for orthopaedic practice. Firstly, epiphyseal injuries can occur in an older group than we would normally expect. Secondly, clear mediocolegal statements about age cannot be made from radiographs alone. Thirdly, care should be exercised when using the atlas of Greulich and Pyle¹ to predict years of remaining growth when this affects orthopaedic surgery as in leg lengthening or epiphyseal closure surgery. It may become necessary to develop a new bone age atlas for subSaharan Africa.

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**References**