INVESTIGATIONS ON THE PREVALENCE AND CHARACTERISTICS OF ADOLESCENT RICKETS IN SOUTH AFRICAN INTERRACIAL SCHOOLCHILD POPULATIONS IN THE TRANSVAAL

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Thesis submitted in fulfilment of the requirements

for the degree

DOCTOR SCIENTIAE

to the Potchefstroom University for Christian Higher Education

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January 1974

ABSTRACT

Certain skeletal abnormalities of legs and chest are normally regarded as sequelae of rickets, and due to vitamin D deficiency. In seeking to throw light on the prevalence and characteristics of these abnormalities in South African schoolchildren, prior to and during adolescence, it was deemed important, firstly, to assess prevalences of these abnormalities and secondly, to investigate their relationship to vitamin D status. Hence, prevalences of bowing and knock knee, and of chest abnormalities such as Harrison's sulcus, pigeon breast, etc., were determined, and dietary, radiological and biochemical studies were made.

Appropriate measurements in the four ethnic groups (Bantu, Coloured, Indian, White) revealed that bowing and knock knee were unexpectedly common, affecting a fifth, and a third of all children, respectively. Prevalences were not obviously affected by a higher dietary intake of vitamin D, rural-urban location, habitual exposure to radiation, or skin pigmentation. Prevalences of chest abnormalities, Harrison's sulcus, 0-5%; pigeon breast, 0-2%, also appeared unrelated to these factors.

An important finding was that with age, bowing frequency increased mainly during the adolescent growth spurt. Prevalence of knock knee was little affected by age, but increased markedly with body mass. There was no age trend with prevalences of chest abnormalities.

Since leg abnormalities were approximately equally common in groups accustomed to a high compared with a low dietary intake of vitamin D, it is inferred that a primary deficiency of vitamin D is not the causal factor. The most plausable explanation is that a secondary deficiency of vitamin D is involved, arising from an impairment in metabolic availability. However, the possibilities cannot be excluded that the leg abnormalities, in large measure, are physiological variants, or that an unknown factor or factors may bear, or share, in the responsibility.

What is urgently required is an attempt to correlate individual vitamin D status of schoolchildren during their pubertal growth spurt; also the carrying out of long-term studies from birth to post-adolescence, of the type already undertaken.

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SUMMARY

Introduction

In the course of dietary, anthropometric and other studies on Bantu schoolchildren the commonness of bowed legs (genu varum) was noted. Later studies revealed knock knee (genu valgum) to be even more prevalent. Both these abnormalities are usually regarded as sequelae of rickets. In view of the observations made the following questions arose. Are abnormalities seen in children, just before, during, and after adolescence, due primarily to an earlier vitamin D deficiency, or to other factor/s? Do they differ in prevalence among the various ethnic groups resident in South Africa? Are there changes with age? How are prevalences affected by the growth spurt of adolescence? Are there other manifestations of skeletal involvement, especially in the chest? Do the latter correlate with prevalences of leg abnormalities? To what extent are abnormalities related to infantile or to adolescent rickets? In this thesis, attempts have been made to supply answers to these and other questions.

In seeking to add to knowledge, anthropometric and clinical observations were made, with special reference to skeletal abnormalities. Estimations were made of intakes of vitamin D of schoolchildren in the Transvaal, as well as in groups of high and low fish-eating Coloured communities near Cape Town. Prophylactic practices were enquired into. Various radiological and biochemical studies were made, furthermore, investigations were carried out on other likely influencing factors, such as exposure to ultraviolet radiation, also skin pigmentation.

Diligent search of the literature, both past and present, unexpectedly

revealed that no previous studies on random populations had been made in respect of prevalences of leg abnormalities, bowing of legs or knock knee, or of chest abnormalities, such as Harrison's sulcus, and pigeon breast. Although almost all workers in this field have referred to these abnormalities as sequelae of rickets, none have sought to measure degrees of abnormalities.

In order to throw light on the aetiology of the skeletal abnormalities mentioned, studies were made on groups of Bantu, Coloured, Indian and White children of 7, 12, and 16 - 17 years. In all, 5573 subjects were studied.

Prevalences of bowing and knock knee. In sex-age comparisons, there were no outstanding differences in prevalences of bowing and knock knee between the four ethnic groups. Within composite series of particular ethnic-sex-age groups, ranges of prevalences were occasionally wide.

At 7 years, mean prevalences for bowing ranged from 4 to 11%; at 12 years, 6 to 22%, and at 16 - 17 years 10 to 26%. The exception was Indian girls, among whom respective prevalences remained similar, namely 8,2%, 8,7% and 7,5%. At 12 years, girls generally had higher prevalences of bowing than boys, which could correspond to the earlier growth spurt of girls.

Knock knee was present in 12 to 32% of pupils at 7 years, 13 to 43% at 12 years, and 12 to 30% at 16 - 17 years. The abnormality was usually commoner in girls than boys at all ages studied. In boys, the proportion with knock knee decreased between 12 to 16 - 17 years, but remaining fairly constant in all groups of girls. Knock knee was

commoner in obese compared with non-obese subjects, and, as would be expected, prevalences of knock knee were higher in girls than boys.

Prevalences of chest abnormalities. There were no outstanding differences in prevalences of chest abnormalities in the four ethnic groups. Harrison's sulcus was present in from 0 to 5%, and pigeon breast, 0 to 2%. Prevalences of other abnormalities, such as flaring of ribs and funnel-shaped sternum, were present in 5 to 15%, and 2 to 10%, respectively. There was no age trend in chest abnormalities.

Skeletal abnormalities and the pubertal growth spurt. The increasing prevalence of bowing appeared to be associated with, or was linked with, the active growth phase of puberty, which occurred earlier in Whites than in Non-Whites. A similar association, in respect of knock knee, however, was not clearly apparent. Prevalence of the latter, as already mentioned, was linked rather with prevalence of obesity. Prevalences of chest abnormalities did not correlate with prevalences of leg abnormalities.

Vitamin D status. In all groups, studies revealed a low intake of vitamin D, with the exception of high fish-eating Coloured communities living at the coast near Cape Town, who understandably had higher intakes. In spite of the adequate vitamin D intake of the latter group, pupils evinced similar prevalences of bowing, knock knee, and straight legs, when compared with (i) groups of pupils at Elsies River, some kilometres inland, where less fish was consumed, also (ii) those eating still less fish, namely, the groups of Coloured children studied in Johannesburg. Thus, the higher intake of vitamin D was not associated with lower prevalences either of leg or chest

abnormalities. It must be kept in mind that in all populations studied, and in all areas studied, the production of vitamin D was unlikely to be deficient since there is an annual mean of 6 - 9 hours of sunshine daily.

Vitamin D prophylactic measures take the form of fortification of milk formulae and cereal foods for babies. In the Transvaal it was found that medical and associated professional workers played a minor role in the promotion of active vitamin D prophylaxis for infants, and even less for older children.

Rural-urban location. There were no marked differences in prevalences of leg abnormalities between groups of pupils resident in the country compared with the towns.

Skin pigmentation. No marked differences could be found in skin colour between Bantu pupils of 16 - 17 years, who had marked bowing, or knock knee, compared with those with straight legs.

X-rays of wrists and hands. No evidence of active rickets was detected in bones of the wrists and hands of Bantu 16 - 17 year pupils with marked bowing, or knock knee, compared with those with straight legs. In these three groups, mean cortical thickness and cortical scores in the second, or index, metacarpal were found to be similar.

Biochemical studies. In groups of Coloured pupils of 12 years, and Bantu of 16 - 17 years, no significant differences in mean values for serum alkaline phosphatase, calcium or inorganic phosphorus were found, in respect of subjects with marked bowing, or knock knee,

compared with those with straight legs.

Aetiology of leg and chest abnormalities. Since a much higher vitamin D intake was not associated with lower prevalences of leg and chest abnormalities, it is judged that sufficient vitamin D is being derived from endogenous sources. In other words, a primary deficiency of vitamin D was not the prime causal factor for the abnormalities observed. The suggestion is made that a secondary deficiency, arising from a metabolic block, inhibition, or impairment of the involved synthesis of the vitamin occurs, particularly over the period of the increased demands of puberty. However, the possibilities cannot be excluded that leg abnormalities, in large measure, are physiological variants, nor that an unknown factor, or factors, may bear, or share, in the responsibility.

Suggestions for future investigations. Further methods of approach would be to attempt to correlate individual vitamin D status of schoolchildren during their pubertal growth spurt, also to carry out long-term studies from birth to adolescence, of the type already undertaken. The value of such studies would be tremendously increased once a reasonably simple biochemical test for vitamin D status becomes available. At present, the rather involved technique for the determination of 25-hydroxycholecalciferol in serum does not lend itself to detailed epidemiological studies of the type envisioned.

CHAPTER I

INTRODUCTION

1.1. THE GENERAL PROBLEM

What is rickets? Rickets is a disease of growing bone and is the result of a deficiency of vitamin D. It affects a number of systems of the body, but by far the most important is the skeletal system. Rickets was defined by Findlay, in 1918, '... as a disease, the chief manifestation of which is to be seen in growing bone, in which there is an excessive and irregular development of its earlier stages with arrest or delay in the formation of fully-formed, firm and normally calcified bone.'

Clinically, active rickets is characterised by skeletal abnormalities which develop due to pressures exerted on softened bone. Swelling of the epiphyseal ends of long bones is visible and palpable, as at the ends of ribs, wrists and ankles. Other skeletal stigmata include the bending of mass-bearing bones, and abnormalities of chest, head and spine; moreover other systems may be involved with results such as muscular atony and head sweats. The skeletal abnormalities, genu varum (bowing of legs) and genu valgum (knock knee), are measurable; other abnormalities such as Harrison's sulcus and pigeon breast, etc. are observable. These stigmata may remain as sequelae.

Histologically, the epiphyseal or growing portion of bone presents a characteristic picture which reveals failure of the normally formed cartilaginous elements to ossify, resulting in osteoid or soft bone formation. Satisfactory ossification occurs only in the presence of adequate vitamin D status, which, together

with calcitonin and parathyroid hormone, maintain calcium homeostasis.

From a review of radiological studies, Caffey (1961) described the histological changes observed, as shadow images of the structural changes, which are seen as a 'rarefaction and irregular fraying of the epiphyseal plate.' With advancing disease,

> 'The shadow of the epiphyseal plate is absent and the terminal segment of the shaft - the rachitic metaphysis - is partially or totally invisible; this is seen only in rickets ... In all cases, cupping and spreading become more conspicuous roentographically when the disease is partially healed ... The entire shaft shows a diffuse rarefaction caused by the loss of lime ... Sometimes sharply defined radioluscent transverse bands, or Umbazonen, are found in the shafts; these are more common in juvenile rickets.'

Public health importance. Rickets, as a public health problem in Western countries, tended to recede into the background following the understanding and introduction of effective prophylactic measures. This led to a false sense of complacency, which was dispelled by the re-appearance of nutritional rickets in Scotland (Arneil and Crosbie, 1963; Arneil, 1964). Since then, in many countries, reports on the appearance of nutritional rickets have been increasing. In addition, and closely relevant, great interest is now being shown in the new hormone, the polar metabolite of vitamin D_3 namely 1,25-dihydroxy-cholecalciferol (1,25 - DHCC).

1.2. THE SPECIFIC PROBLEM

In South Africa there are four ethnic groups, Bantu, Coloured (Eur-African-Malay), Asian and White.

Note: The Asian children studied were drawn from the local Indian community, and are therefore designated by the term Indian. The more embrassive term, Asian is used only when reference is made to published work in Britain and elsewhere, which included not only people of Indian origin but also from other areas of Asia as well.

The South African context. How common is rickets in infants and children in the different ethnic groups in South Africa? Formerly although the disease was believed to be rare Feldman (1950), followed by others (Dancaster and Jackson, 1960, 1961; Taitz and de Lacy, 1962; Robertson 1969; Joffe et al., 1972), showed that rickets was far from uncommon, especially in large centres of population. However, little is known of its prevalence in non-hospital populations both in urban and rural areas. Rickets in the older child has received little attention, although instances have been noted in most series reported. Mission hospitals are aware of the disease; at some centres osteotomies have been performed for the correction of limb deformities in older children, although the operation is undertaken infrequently.

Preliminary studies. In early studies it was quickly noted that bowing of the legs and knock knee, usually regarded as classical signs or symptoms associated with rickets, were common among schoolchildren of all ethnic groups. Furthermore, in earlier studies on Bantu pre-school children, stigmata associated with rickets, namely, leg and

chest abnormalities, appeared to be as common among rural as among urban dwellers (Richardson, 1971).

Definition of the problem. The following are relevant questions to which answers are urgently needed.

(i) What are the prevalences of the stigmata, in particular, bowing of the legs and knock knee, and various chest abnormalities (Harrison's sulcus, pigeon chest, etc.), in random samples of children in the different ethnic groups, especially those dwelling in different regions?

(ii) It is known that abnormalities of these types are seen in otherwise healthy subjects.

(a) Can they be attributable, partly or wholly, to inadequate vitamin D status?

(b) Do abnormalities lie within the normal range of human physiology? or

(c) Are they attributable, partly or wholly, to unknown factors?

(iii) To what extent are the prevalences of abnormalities influenced by the growth spurt, with its known increased hormonal and other demands?

(iv) To what extent are chest deformities (of the type known to be closely associated with rickets) seen at later years, manifestations of early involvement? To what extent are chest deformities related to

the prevalences of leg abnormalities noted in schoolchildren?

(v) Does intensity of skin pigmentation play a significant role in the aetiology of rickets, and/or, in the stigmata under review?

(vi) What are the relative importances of diet and of ultraviolet irradiation in their respective contribution to vitamin D status, as judged by possible sequelae of rickets in schoolchildren?

(vii) To what extent are biochemical parameters, measured in later years, associated with chest or leg abnormalities?

1.3. SPECIFIC AIMS

The specific aims of the research work to be undertaken on adolescent or late rickets are as follows:-

(i) To investigate the prevalences of late rickets, and/or sequelae attributable to rickets, at ages 7, 12 and 16 - 17 years, in four ethnic groups of South African schoolchildren, namely, Bantu, Coloured, Indian and White.

(ii) To seek to elucidate to what extent the bony abnormalities present in older schoolchildren (a) are attributable to inadequate vitamin D status, (b) are variants of normal physiology, (c) are affected by the growth spurt?

1.4. LITERATURE BACKGROUND

1.4.1. Introduction

Deficiencies of nutrients when severe give rise to various stigmata. The deleterious effects may be widespread, as in protein-calorie malnutrition; here protein metabolism is disturbed, and growth fails to proceed satisfactorily, the degree of failure being dependant on the severity of the deficiency. On the other hand, in the presence of other deficiencies, the ill effects may be specific, as occurs in some vitamin deficiencies; for example, lack of niacin or of ascorbic acid causes pellagra or scurvy, respectively. When vitamin D status is inadequate, whether from dietary deficiency or from insufficiency of solar radiation, the results are specific, and lead to the development of rickets in children, and osteomalacia in adults.

1.4.2. Derivation of the Term Rickets

The term 'The Rickets' may have originated from one of several sources. It has been asked, was it derived from the old English verb 'wrikken' meaning to wrest or twist awry, as in 'to wrick one's ankle' (Cheadle and Poynton, 1913; Findlay, 1918; Dick, 1922)? Or, since victims of the disease often had humped backs, was it evolved from 'ricq' meaning hump (Weick, 1967)? Early writers suggested that 'The Rickets' may even have been a 'vulgar' method of pronouncing 'rachitis' (Greek 'rhacia' - spine), perhaps given to it by some learned Grecian, who had accurately observed the striking weakness of the spine in this disease. Although 'The Rickets' was in common usage in England, in the Seventeenth Century, Glisson and his associates

(1650) considered that a more sophisticated Latin or Greek term would be more appropriate. Thus 'rachitis', derived from the Greek word for the spine was thought to be not only more suitable, but also more distinctive, and, at the same time descriptive. However, it continued to be known as the 'English disease', so rife was it in England during the late 19th and early 20th centuries. Whatever the derivation - and Hunter (1972) has speculated upon 'Rickets, ruckets, rickets or rackets' reaching the conclusion that the nomenclature remains 'doubtful - to say the least' - the term rickets was, and still is, widely used, and known thus to both the medical profession and the public.

1.4.3. Description of Rickets

In describing rickets Parker (1888) viewed the condition

> '... as an inherited constitutional condition ... manifesting itself in infants as a tendency to catarrh of the pulmonary and intestinal tracts, and of the ventricles of the brain; in young children, as a tendency to disease at the epiphyseal junctions; in older children, in the curvature of the long bones; in young adolescents, as a relaxed condition of ligaments, which favoured the onset of genu valgum, flat foot, angular curvatures of the spine, etc. In this sense rickets was a condition which might last from birth up to the 15th or 17th year of life.'

A clear picture of a child with rickets was given by Cheadle and

Poynton (1913).

> 'The square forehead with rounded, boss-like projections on the frontal bone; the enlarged ends of the long bones - the deformities produced by the giving way of soft and yielding bones, such as the contracted chest, the deformed ribs, the curved spine, the protuberant abdomen, the bent arms and clavicles, the bowed legs or knock knees, the yielding ankles, the defective, decaying teeth, the stunted growth - are features which cannot be mistaken. In many instances the child is plump or even fatter than normal, others again are thin, wasted and puny.'

1.4.4. Early History

Rickets has been known since early times, indeed even at the dawn of history in Neanderthal man it has been asked were his 'beetling brows and stooped posture' not indicative of rickets? The few bones and skulls of children of this period which remain show severe rachitic changes (Ivanhoe, 1970; Anon., 1970). The Court Jester at the time of Homer (900 B.C.) is thought to represent a typical picture of rickets, with his bandy legs, rounded shoulders, etc., but this description was regarded by Findlay (1918) as being rather more akin to the village idiot or 'natural', as mental aberration, so characteristic of the Court Jester, is not true of rickets.

Lortet of Lyons (cited by Findlay, 1918), described rickets in

the remains of an ape kept captive in a temple in Thebes. Although some passages from early writings have been thought to describe rickets, there is no evidence that the disease existed in Ancient Egypt (Findlay, 1918). The reputed absence of the disease in bones in cemetries in Ancient Egypt was questioned by Elliot Smith, who believed that many of the bones which were recovered exhibited distortions difficult to explain except as due to rickets. However, the fact that Hippocrates (460 B.C.) and Galen (2nd Century A.D.) dealt so fully with conditions of bones and joints, without reference to anything which resembled true rickets, was thought by Dick (1922) to constitute contrary evidence of its existence during that period. Hippocrates and Galen both wrote extensively of deformities of the spine, and it was to Hippocrates we owe the terms, kyphosis, lordosis and scoliosis. Bones from ancient graves in California, North West Peru and from other sites in South America, did not yield signs of rickets, nor did those from early Britons.

A very apt and careful clinical description of what is thought to have been rickets was given by Soranus Ephesius (98 to 138 A.D.), a physician of Rome, also the biographer of Hippocrates, who lived for some time at Alexandria, where, at that time, there was a well known medical school. Soranus gave very explicit instructions to those in care of young infants, in the art of sitting, standing and ultimately walking, in order to prevent the weight of the body from causing deformity 'since the bones are not yet stiff' and 'while as yet the body has no sinews to resist the strain.' He advocated the use of a 'chair on wheels', no doubt the precursor of the modern

walking ring for toddlers. It was not until the late 16th and the early 17th Century that rachitic changes were found to occur in bones in Britain, such as those found at Hythe, near London, and those examined from churchyards of Bluecoat School, Newgate Street, and at Clare Market, London (Dick, 1922).

The skulls of this period represented an interesting transitional type with a tendency to the modern cranium, with its deformed upper and diminished lower jaws, crowding and decay of teeth; such are so marked a contrast to the well-formed dental arches, little tooth decay, and no overlapping of jaws, which were characteristic of the primitive cranium.

In the past spontaneous rickets was known to occur in many animal species kept in unnatural conditions. The disease has been seen in young monkeys confined in Zoological Gardens. Rickets also occurs in horses, sheep, and in dogs, particularly the larger breeds.

It is to Francis Glisson, of anatomical fame, to whom, in 1650, we are indebted for the finest, and most accurate description of rickets, and for its establishment as a clinical entity. The name 'Glissonian rickets' is still used to describe 'the older child, dwarfed, distorted and bow-legged.' He differentiated between rickets and scurvy, but confusion arose later which was dispelled by Cheadle (1888). The superficial resemblance of rickets to scurvy continued to cause much confusion for some time.

1.4.5. Rickets in the 20th Century

By the turn of the century Cheadle and Poynton (1913) recognised that 'perverted nutrition' played a large role in the aetiology of rickets, maintaining that as surely as scurvy was produced by a scorbutic diet, so certainly was rickets the result of a rachitic diet. But the disease was 'aggravated in degree by evil hygienic conditions ...' and modified by conditions such as 'the concurrent existence of congenital syphilis or of scurvy.'

It was during the period of the 19th and the early part of the 20th Centuries that the prevalence of rickets reached its peak. According to Weick (1967), at the turn of the century, almost two-thirds of children in the smoke-laden, industrial cities of temperate zones were reported to have evidence of the disease, moreover, no race nor nationality was exempt. Even well cared for babies were affected, and Weick (1967) quoted from Morse (1900) that 'at one time 318 of the 400 babies at Boston Infants Hospital were rachitic.' Hess and Unger (1922) observed 250 babies for four years; they concluded that rickets was universal in bottle-fed babies, and averred furthermore that 50% of a group of well-nourished breast-fed babies had rickets (Hess and Lundagen, 1922). In New Orleans, of 197 breast-fed babies, all showed signs of rickets within a year. Statistics were alarming and the prevalence of the disease was so high that Eliot (1925), in a three-year project begun in 1923, stated that 'our investigations have shown that a slight degree of early rickets is well-nigh universal in our climate and in our state of society.' The situation was no better in England nor on the Continent, and this

stimulated investigation and led to the carrying out of a large amount of experimental work, not only on animals, but on children.

It was the classical experimental work carried out in Vienna during 1918 to 1922, by the British workers Harriet Chick and her colleagues (1923), that firmly established the aetiology of rickets. In exhaustive dietary and light therapy studies, they dispelled many uncertainties and fallacies previously linked with views on the causation of the disease. It had been believed by many, for instance, to be due to infection. Indeed this view was held largely by eminent authorities on the Continent, of whom Professor Pirquet, at the Kinderklinik, Vienna (where the studies were carried out) admitted that he was one (Chick et al., 1923).

At about the same time that Chick and her colleagues (1923) were carrying out research in Vienna, Dick (1922), a keen observer, was extensively investigating rickets and its sequelae in London. He made valuable studies on schoolchildren; he helped to elucidate the role of breast feeding, and of socio-economic status and environment. In his very exhaustive studies on poor East End children in London, he found that at Hackney, of 1000 schoolchildren examined, 798 had rickets, i.e. 80%. He stressed that children at school, when affected, were not necessarily suffering from rickets at that time, but had suffered from it, and the rachitic stigmata had persisted through the child's school life. In schools in Whitechapel and Stepney, a 1000 children were also examined, of whom 80% were Jewish. Rickets was found to be as common among Jewish as among non-Jewish children.

In Vienna, from intensive histological studies on post-mortem material from young infants, Dalyell and Mackay (1923) clarified the changes that occur in bone morphology. Moreover, also arising from the studies in Vienna, clinical diagnosis could now be confirmed by X-ray. This was a valuable asset, not only for the initial diagnosis, but by using serial radiographs, the progress of the healing process could be followed. In the Vienna study, X-ray procedures were used extensively to detect the first appearance of rachitic changes in the 'healthy infants under study, also for weekly follow-throughs' (Wimberger, 1923). Some doubts have recently been raised as to the validity of X-ray detection of <u>early</u> rickets (Anon., 1971a),owing to technical errors in the procedure. However, once active rickets is firmly established there is no question as to the value of radiography. For epidemiological studies, the procedure is of limited value (Richardson and Walker, 1971) because the active process, having long since undergone regression, is no longer detectable (Caffey, 1961).

In the past, in countries experiencing much sunshine, such as New Zealand, South Africa and Australia, rickets was reported to be rare, particularly in rural areas. In Australia, however, in 1892, the prevalence of the disease, usually of a mild type, was reported to be increasing in spite of the sunny climate. A report from the Medical Officer of Health of the Victoria Education Department (1911 - 12) (cited by Dick, 1922) described a careful enquiry into the presence of rickets in schoolchildren in Melbourne. It was shown that a quarter of the children examined were suffering from

rickets, or had suffered from rickets, in a mild form. The writers pointed out that

> 'The cases recorded are all mild cases, and as such are very likely to escape the observation of the parent. Only a systematic examination of the child, actually looking for evidence of the disease, would reveal the existence of these cases.'

Dr. Harvey Sutton (1920), Principal Medical Officer of Health of New South Wales, reported that studies indicated that 25% of schoolchildren in New South Wales had definite signs of rickets, or its sequelae. That these were mild cases was stressed in the report; but it was contended that '... even mild rickets has far-reaching results.'

The eventual isolation of vitamin D in 1922 by McCollum and his co-workers (1922), together with knowledge of its curative and preventive effects, also in 1927 (Bills, 1935) the discovery of the ability to irradiate ergosterol (derived from yeasts and moulds) to yield vitamin D_2, led to the introduction of public health preventive measures. So effective were they that the disease declined and became of little public health importance. Irradiated dried milk and other infant foodstuffs proved to be the best therapeutic agents, as mothers did not avail themselves of the cheap or free supply of cod liver oil (Arneil, 1964). This was no new situation, as in 1922 Dick wrote '... rickets is rampant in Oldham and in the colliery districts, in spite of the fact that malt and cod liver oil are freely administered to these ailing

children, ...'

The importance of rickets, as a public health problem in Western countries, rapidly receded into the background. Sporadic cases continued to appear, but the D-resistant types came more prominently to the fore, and were much studied. It was therefore a surprise and a shock when reports of nutritional rickets began to become more frequent. One early report, in 1944, concerned rickets in poor Jewish children in Jerusalem (Klasmer, 1944). In 1959 the disease had re-appeared in Scotland (Arneil and Crosbie, 1963, Arneil, 1964). Reports of rickets came from the Far North in Kaluga Oblast (Frenkel et al., 1962), and even from Manilla in the tropics (Stransky et al., 1959). By 1962, cases of rickets were reported as occurring in many countries, and by 1970 at least 33 papers had been published on its increased appearance. These reports were widely spread, emanating from as far afield as Murmansk in the Arctic (Gershkovich and Belogorskii, 1962), also from the Far North (Nemzer and Belogorskii, 1963, 1965); Sydney (Lipson, 1970) in the South; and China (Maxwell, 1923) in the East; and U.S.A. (Welck, 1967) in the West.

The discovery that rickets could be common in the lands of sunshine was unexpected and contrary to orthodox thinking; it seemed unbelievable that a vitamin D deficiency could be common where 'sunshine - the cheapest "vitamin" of them all' (Anon., 1966) was abundantly available. Rickets was reported from South Africa (Kark and le Riche, 1964; Feldman, 1950; Dancaster and Jackson, 1960, 1961, 1962; Taitz and de Lacy, 1962), and Greece (Lapatsanis et al., 1968). Most, but not all publications were from centres of

population. In Britain, reports from Glasgow (Arneil and Crosbie, 1963; Arneil, 1964) blamed the laxity of parents and public health departments in not maintaining effective vitamin D prophylaxis. Vitamin D enrichment was an effective prophylactic measure, the substance being incorporated into most baby foods, dried milk, butter, etc. Health departments had been lulled into a state of complacency, as were doctors, for not anticipating, or even entertaining, the possibility of the re-emergence of rickets. In Glasgow, family practitioners had failed to make the correct diagnosis of rickets in 36 of 40 cases reported among White children (Arneil, 1964). Who and what was to blame? Immigrants, who accounted for most of the cases, could perhaps not afford or were perhaps unaware of vitamin D enriched infant formulae, and instead were using fresh cow's milk or condensed milk. Also relevant was absence of immigrant children from baby clinics, so that they could not receive cod-liver oil or similar preparations. These possible reasons, however, could only partly explain the situation in which Scottish children appeared to be markedly affected? Richards et al. (1968) reported that a 'considerable number of Scottish infants are not receiving sufficient vitamin D ...' In East Germany, according to Harnapp (1969), preventive treatment with vitamin D was not insisted on nor consistently practiced.

Recently an increasing number of reports have appeared in Britain which confirmed the presence of rickets and osteomalacia in immigrant Asian communities (Dunnigan et al. 1962; Felton and Stone, 1966; Swan and Cooke, 1971; Ford et al., 1972b; Holmes et al., 1973). The majority of cases of rickets appeared to occur at puberty.

1.4.6. The Aetiology of Rickets

Environment

In the late 19th and early 20th centuries in England it was believed that rickets was caused mainly by the close, and 'vitiated atmosphere' in which children, mainly of the poor, were brought up. Lack of airspace in dark, often dank, crowded tenement buildings was the subject of much investigation, and, among other studies, rooms were carefully measured, seeking to relate such data to the alarmingly high prevalence of rickets. Dick (1922) was appointed by the Government to look into the situation in London. By detailed studies he showed that rickets was present not only in children of the poor, but also in well-nourished, otherwise healthy children, who suffered as much as those less privileged, and among whom (the healthy children), apart from confinement indoors, other causative factors were considered to be absent.

Diet

In 1892, Turner (Dick, 1922) considered that improper feeding was not the exclusive cause of rickets. Such feeding was more common in Australia, where rickets was less frequent, than it was in England, where rickets was common. Unlike Cheadle and Poynton (1913), Dick (1922) believed that dietary conditions alone could not cause rickets, mainly because of the geographical distribution of the disease. Opinion as to whether it was a deficiency disease or not was confused. Although many still rejected the idea of the presence of a particular nutritional deficiency, this view became accepted

by workers such as Mellanby (1921, 1925), McCollum (1922), Korenchevsky (1922), and their respective co-workers. Finally, Hume (1923), and Chick et al., (1923) showed that a deficiency of vitamin D was the specific cause.

The conclusive studies of Chick et al., (1923) demonstrated that Findlay's (1918) view, namely, that exercise was the prime factor in the causation of rickets, was not true. This conclusion had been arrived at by Findlay (1918) on the basis of his studies on puppies. Mellanby (1921, 1925), who also worked with puppies, stressed the importance of a dietary factor. The 'interfering action of cereals on bone calcification' appeared to be linked with an associated deficiency of an anti-rachitic vitamin. This substance he described as fat-soluble, and believed it to be probably vitamin A. He found that puppies developed rickets on an oatmeal diet, which was richer in calcium and phosphorus than white flour, but apparently contained little or no anti-rachitic vitamin. Oatmeal was thought to contain more of the 'substance or substances interfering with ossification', which would explain the lower deposition of calcium and phosphorus in growing bones on an oatmeal diet, than the better but still defective deposition on a white flour diet. In humans, the fat intake would presumably counteract this rachitogenic effect in those, for example, who were accustomed to a diet high in oatmeal. McCollum and co-workers (1922), in their extensive studies on rats, concluded that the anti-rachitic fat-soluble vitamin was not vitamin A, as they had originally thought, although so similar were their distributions in nature that it appeared possible that there were

two properties for the one vitamin. Margaret Hume (1923) showed that the anti-rachitic factor was not vitamin A by her carefully controlled experiments with rats, and also her studies on infants' diets. Korenchevsky (1922), also from experiments on rats, deduced and stressed the importance of the anti-rachitic factor in the mother's diet, particularly during pregnancy and lactation, and, of course, in the infant's diet. He went so far as to suggest that it was even of importance in the father's diet. He did much to elucidate the mechanism of the rachitic process, and concluded that the anti-rachitic factor, together with light, fresh air and muscular exercise, was curative. After much experimental work, the anti-rachitic vitamin, vitamin D, was finally isolated in 1922 by McCollum et al. (1922).

Although breast feeding may have given a measure of protection, Dick (1922), Hess and Unger (1922), also Chick et al., (1923) showed that breast fed babies were not exempt from rickets, nor were those who otherwise had obviously good nutritional status. In the classical investigations in Vienna, it was found that breast fed infants, in an otherwise healthy and hygienic environment, developed rickets. Of the 1000 Jewish schoolchildren studied by Dick (1922) in London, 81,4% had been breast fed for 6 months or longer; of the 1000 non-Jewish schoolchildren, at Hackney, 57,5% had been entirely breast fed, and 20,5% part breast and part bottle fed. Of the breast-fed children, 72,5%, and of the bottle fed children, 87% had rickets. From this evidence it was concluded that breast feeding could not guarantee protection against the disease. To learn more of the

role of previous nutrition, Dick (1922) reported that in his studies in which 80% of children were found to have rickets, 23% were in a very good nutritional state, 57% in a good, but only 20% were revealed to be in a poor nutritional state. Much later, in his excellent study, Lipson (1970) showed that the vitamin D content of mothers' milk was not sufficient for the infant's daily needs, although it had a higher content of the vitamin than cows' milk. He gave the value for vitamin D in human milk to be 0,4 - 10 I.U./100 ml, and in cows' milk, 0,3 - 40 I.U./100 ml. The duration of breast feeding had also been implicated, but Ferguson (1918) found this factor to have no relation to the occurrence of the disease.

In spite of the earlier definitive studies, the belief that breast feeding is protective still persists (Anon., 1969; Kendall, 1972). It was the adequate nourishment usually implied by breast feeding that presented a barrier to the acceptance of the nutritional origin of rickets in children who have been 'well cared for', with apparently every attention being paid to diet. The 'well-cared-for' syndrome clouded the issue for many years, although, even in 1888, Cheadle maintained that in children 'being fat and apparently well nourished, the co-existent rickets was often overlooked.'

In several studies made, during 1966 and 1967, on the intake of vitamin D in Scottish children, the amount consumed was found to be inadequate. Children were probably receiving less than 100 I.U. per day from all sources, apart from that derived from sunshine (Arnell, 1967; Richards et al., 1968). Estimated intakes of vitamin D in White children and adults in the United Kingdom, made by the

Panel of Child Nutrition (1970) were found to be low, and similar to those of immigrant rachitic children studied in Glasgow where the mean figure was 189 I.U. daily (Ford et al., 1972). In a report from Morocco, Guignard et al., (1971) found that dietary vitamin D intake was very low and the prevalence of rickets high, even in Nomad children. In the city of Rabat-Salé only 40 - 50% of the large population of 34,800 infants under 2 years received regular treatment with vitamin D preparations. None of the treated children, however, developed rickets. Similarly in Melbourne, Australia, of the children presenting at hospital with rickets, only very few had had supplementary vitamin D (Mayne and McCredie, 1972).

Phytic acid and vitamin D deficient states

Phytic acid has been thought to be incriminated in the causation of rickets since the early work of Mellanby (1921, 1925) on cereals and their anticalcifying activity. The belief was that phytic acid precipitated calcium in the intestinal tract and hence reduced the amount available for absorption and retention. Its importance in the aetiology of rickets in human nutrition was questioned by Cruikshank et al., (1945) and by Walker (1951, 1973).

In recent years, controversy over the role of phytic acid in the causation of rickets and osteomalacia has been renewed. In their studies on Pakistani women and children with osteomalacia and late rickets, Ford et al. (1972a) were able to raise the level of serum calcium to near normal levels, by use of a chupatti-free diet. Levels fell after resumption of the chupatti-containing diet. In Northern India and Pakistan, Vaishnava and Rizvi (1971) found rickets

and osteomalacia to be common where unleavened bread was consumed, but rare in rice-eating populations of Southern India. However, these workers have since found nutritional osteomalacia in rice and maize-eating Kashmiri women of low economic state, also in patients in New Delhi consuming a mixed chupatti and rice diet. 'Therefore, we believe that phytate hardly plays a role in the production of this type of osteomalacia.' (Vaishnava and Rizvi, 1973).

That phytates play a negligible role in the aetiology of rickets has also been postulated by several groups of workers (Dent et al., 1973; Anon., 1973a; Preece et al., 1973). The latter authors conclude '... there have so far been no reports of clinical healing of rickets or osteomalacia when the phytate intake has been reduced without altering vitamin-D intake.'

Sunlight and ultraviolet irradiation

The importance of sunlight as a curative factor in rickets has had a checkered history. On epidemiological grounds Palm in 1890 drew attention to its importance, and concluded, as had the Polish physician Sniedecki in 1822, that sunlight had both preventive and curative properties (cited by Lipson, 1970). In Vienna, the curative role of sunlight had been demonstrated by Huldschinsky in observations on children in 1919 - 1920 (Chick et al., 1923). In 1921 Hess and associated workers found that radiation from a mercury vapour lamp could cure rickets. Shortly after, this practice was followed by successful treatment of the disease with the carbon arc (Hess and Unger, 1922). The contribution of Chick's research group, however, was to establish, without any doubt, that a factor, present in

sunlight, the carbon arc, and in cod-liver oil, was able to promote the healing of active rickets. It was found that ultraviolet rays, present in both sunlight and the carbon arc, were anti-rachitic. Later, these rays were shown to have the capacity to activate the provitamin 7-dehydrocholesterol, present in skin, to vitamin D_3 (cholecalciferol). The metabolically similar vitamin D_2 was found to be present in cod-liver oil. Subsequently, other fish-liver oils were proved to be rich sources of the substance. In 1927, the irradiation of yeasts and moulds yielded vitamin D_2, ergosterol (Bills, 1935). In Britain, after World War II, National dried milk was fortified with calciferol (Richards et al., 1968); it contained not less than 10 I.U. per g (280 I.U. per ounce). The practice led to the discovery that an overdose of vitamin D from fortified dried milks, cereals, and vitamin D preparations, could on occasion cause the serious condition of hypervitaminosis D. A Joint Sub-Committee on Welfare Foods investigated the situation (Bransby et al., 1964), and in consequence the vitamin D content of enriched foodstuffs was reduced by half from 1959 onwards (Anon., 1966), and a daily intake of 400 I.U. was recommended. In the United States, fortification of foods with vitamin D remains statutory (Holmes et al., 1973), with the result that serum vitamin D values in adults, 27,3 ng/ml, are higher than those in Britain, 16,0 ng/ml (Preece et al., 1973).

Recent views on the exogenous and endogenous sources of vitamin D

On reviewing the recent controversial literature on the aetiology of late rickets and osteomalacia in Asian immigrants to Britain, Hodgkin et al., (1973)conclude that, in conjunction with the low

dietary intake of vitamin D, there is also inadequate exposure to solar radiation. Recently, reliance on exogenous sources of vitamin D has been stressed rather than reliance on that obtained from the diet, except in environments where solar radiation is markedly inadequate. In their excellent review on the 'Regulation of vitamin D metabolism and function' Omdahl and DeLuca (1973) reached the following conclusions:

> 'Under normal circumstances it is reasonable to expect that vitamin D is not required in the diet since adequate ultraviolet irradiation of skin will produce sufficient vitamin D. In that respect vitamin D should not be considered a normal dietary requirement but instead a prohormone, ...'

Support for this view has now been provided by Haddad and Hahn (1973) in the first direct quantification of the separate contributions of cutaneous and dietary ingestion of vitamin D in man. They showed, using sophisticated biochemical techniques, that of ingested and exogenous sources of vitamin D, the latter account for the majority of circulating 25-HCC. The ingested source only becomes of importance when the diet is unusually rich in fish products or when there is significant fortification of foods, as occurs in U.S.A. (Palmisano, 1973).

Pigmentation of skin

As indicated, much of the daily vitamin D requirement is synthesised in the skin by a process of photoactivation of 7-dehydro-

cholesterol (Reinertson and Wheatley, 1959), which is present almost exclusively beneath the stratum corneum. Skin has also been found to contain a mean of 3,2 µg cholecalciferol/g skin (Rauschkolb et al., 1969). The physiological range of vitamin D synthesis (0,01 to 2,5 mg per day) prevails throughout man's 'worldwide habitat' (Loomis, 1967). Synthesis is regulated by the twin processes of pigmentation and keratinisation. From available data, Loomis (1967) calculated that 1cm^2 of the skin of Whites synthesises up to 18 I.U. vitamin D in 3 hours; thus, approximately 20 cm^2 of a White infant's nearly transparent pink cheeks, could synthesise approximately 400 I.U., i.e. 10 mg (1 I.U.= 0, 025 mg), the required daily amount, within 3 hours.

The pigmented layer of skin (stratum Malphigian) lies immediately below the melanised stratum corneum, which, according to Thomson (1955), forms an effective barrier through which ultraviolet radiation of wavelengths below 436 nm cannot pass. Heavily pigmented skin of the Negro did not allow the passage of these waves, whereas the skin of Whites allowed transmission of waves of 365 to 405 nanometer bands. Although considerable variation occurred within each group, the skin of Whites allowed an average of 64% (53% - 72%) of waves of these wavelengths (300 - 400 nm) to pass through the stratum corneum, whereas only 18% (3% - 36%) passed through the skin of Negroes (U.S.A.). Further, the darker the Negro skin, the lower the percentage of ultraviolet radiation that was transmitted (Loomis, 1967; Kahlon, 1972). The corneum stratum of Africans, therefore, filters out at an equivalent of 50 - 95% of ultraviolet rays reaching

the vitamin-D-synthesising region of White skin (Thomson, 1955). This is in accordance with 'the fact, agreed to by all, that of all races the Negro is the most susceptible to rickets' (Hess and Unger, 1917).

Metabolic processes and vitamin D_3

Post mortem examination of rachitic bone enabled histologists to study the changes associated with the disease. In the absence of exact knowledge of the biochemical mechanism, the failure of the body to deposit bone components led to the putting forward of several possible mechanisms. Early experiments on animals indicated the possible importance of the increase or absence of some internal secretion in rickets. Wells (1905 - 1906) introduced costal and tracheal cartilage, which does not normally ossify, into the peritoneal cavity of rabbits, together with epiphyseal cartilage, which does normally ossify; he found that even after sterilisation by heat, both presented a great affinity for calcium. Findlay (1918) noted that rachitic cartilage absorbed as much calcium as non-rachitic cartilage under the same conditions. While the importance of 'some internal secretion' necessary for the satisfactory development and calcification of growing bone continued to be much discussed, experimental proof was lacking. This had to await the advent of radio-isotape studies; the relevant sophisticated techniques were only developed nearly half a century later.

Radio-isotopic studies using labelled calcium indicated areas of bone where calcium not only was actively laid down, but accumulated

rapidly, as at the knee metaphyses, the lower metaphyses being more active than the shaft. In the presence of rickets, the rate of accumulation was found to be low, but it returned to normal after treatment with vitamin D. A fracture site in a patient with healing rickets showed a high rate of accumulation of calcium (Harris et al., 1965).

In his study of rachitic and non-rachitic control children, Barak (1970) found a greater number of osteoblasts, which are responsible for the laying down of bone salts, to be present in all rachitic subjects. Osteoblasts were significantly correlated with serum alkaline phosphatase activity. He demonstrated positive cytochemical alkaline phosphatase activity in these cells, and suggested that the demonstration of the osteoblastic origin of serum alkaline phosphatase, in persons among whom there were unexplained high values, would be confirmatory of metabolic disorder.

"Biochemical" rickets

This term connotes the identification of rickets by means of biochemical parameters. One of the early suggestions of rickets causation concerned abnormalities in the deposition of bone salts. In the mineralization of normal bone, calcium and phosphorus are deposited as 'bone salts' and form the principal components of hydroxyapatite. In rickets, the level of serum phosphorus is depressed, whilst that of calcium is little affected. This affects the solubility product. This process was explained by Guyton (1961) as follows:

> 'The solubility product for calcium phosphate ($CaHPO_4$) at body temperature is 3,4 times 10^{-6} ... When the product of the two concentrations is greater than 3,4 times 10^{-6}, calcium phosphate will crystallize out of the solution, but when the product is less than this value, crystals of calcium phosphate that have already been formed will be absorbed.'

However, with normal concentrations of calcium and phosphate in the extracellular fluid,

> 'Multiplying these molar concentrations of calcium and phosphate times each other (0,0012 times 0,0011) one obtains a product of 1,32 times 10^{-6}, which is approximately three times less than the solubility product ... precipitation of calcium phosphate from these fluids cannot occur without a specific physiological mechanism to cause this. ... This process has been called <u>crystal seeding</u> or <u>nucleation</u>. ... crystals progressively change into hydroxyapatite ... extracellular fluids are always supersaturated in relation to hydroxyapatite.'

During prolonged deficiency of calcium and phosphorus increased parathyroid hormone protects the body against hypocalcaemia. However, overaction of the parathyroids leads to a lowering of the phosphorus level resulting in hypophosphataemia. This in turn lowers the solubility product of serum calcium and phosphorus. If this fails

below 30, calcium triphosphate cannot be incorporated into the hydroxyapatite bone crystals, and uncalcified osteoid results (Taitz and de Lacy, 1962). Bone becomes progressively weaker. An increasing physical stress on weakened bone stimulates osteoblastic activity, with the result that,

> 'The osteoblasts lay down large quantities of organic bone matrix, which does not become calcified because even with the "crystal seeding" effect of the matrix the product of calcium and phosphate ions is still insufficient to cause calcification. Consequently, the newly formed, uncalcified organic matrix gradually takes the place of other bone that is being reabsorbed.
>
> Obviously,hyperplasia of the parathyroid glands is very marked in rickets because of the decreased blood calcium level, and the alkaline phosphatase level in the blood is markedly increased as a result of the rapid osteoblastic activity.' (Guyton, 1961).

Thus the biochemical test most commonly used, and believed to be most revealing, is serum alkaline phosphatase estimation. Raised values occur during periods of active bone growth and occur in young and actively growing children and in pregnant women. Gastro-intestinal alkaline phosphatase, not of osteoblastic origin, has been found in similar concentrations in the duodenal juice of children with vitamin D deficient rickets compared with control children (Ozsoylu and Turhan, 1971).

In rickets, coincident with increased osteoblastic activity,

raised serum alkaline phosphatase levels would be expected as long as the disease remains active. There is a strong body of opinion that a raised value for serum alkaline phosphatase is diagnostic and represents 'the best criterion of rickets' (Lapatsanis et al.,1968), the most 'sensitive indicator of rickets' (Anon., 1969), and 'the sheet-anchor of diagnosis' (Anon., 1971a). Values higher than 25 or 30 King Armstrong (K.A.) units have been regarded as indicative of rickets (Richards et al., 1968; Barsky, 1969; Ford et al. 1972b; Cooke et al., 1973) with or without associated clinical, radiological or other biochemical criteria.

There is, however, an equally strong body of opinion holding opposing views, such as that of Klasmer (1944) '... determination of the phosphatase activity can be regarded only as an indirect test for vitamin D ...'; and of Mayne and McCredie (1972) 'No single biochemical test can be considered diagnostic.' In one recent contribution, a confusing picture has arisen, in which children, receiving vitamin D supplements, were reported to have as wide a range of alkaline phosphatase values above 25 K.A. units as those not receiving a supplement, among whom no precise upper limit was definable (Stephan and Stephenson, 1971). Reddy and Srikantia (1967) reported several children, who despite radiological evidence of rickets, had normal alkaline phosphatase values. Recently Cooke et al. (1973) found no significant differences in alkaline phosphatase values in Asians, Whites or West Indians; about 40% of the children studied had values greater than 30 K.A. units at 14 - 17 years, and yet radiologically confirmed rickets was more common in the Asian group. Workers in

the Far North considered radiological diagnosis to be 'more exact than biochemical studies' (Belogorskii, 1962 and Belogorskii and Nemzer, 1967). In a recent very important and critical study, Round (1973) showed that a high level of serum alkaline phosphatase was normal at 7 years; in girls, by 15 years values fell to levels found in adults, but boys at 18+ years were found to have slightly higher values than adults. Round (1973) demonstrated a pubertal "flare" in boys between the ages of 10 - 14 years, and in girls between 8 - 12 years (among whom this was less marked); these paralleled the adolescent growth spurt.

Recent biochemical advances - the new hormone

Recent advances in knowledge of the new hormone have been made by many workers in various countries, on the subject. The information acquired is of absorbing interest and will therefore be described in some detail.

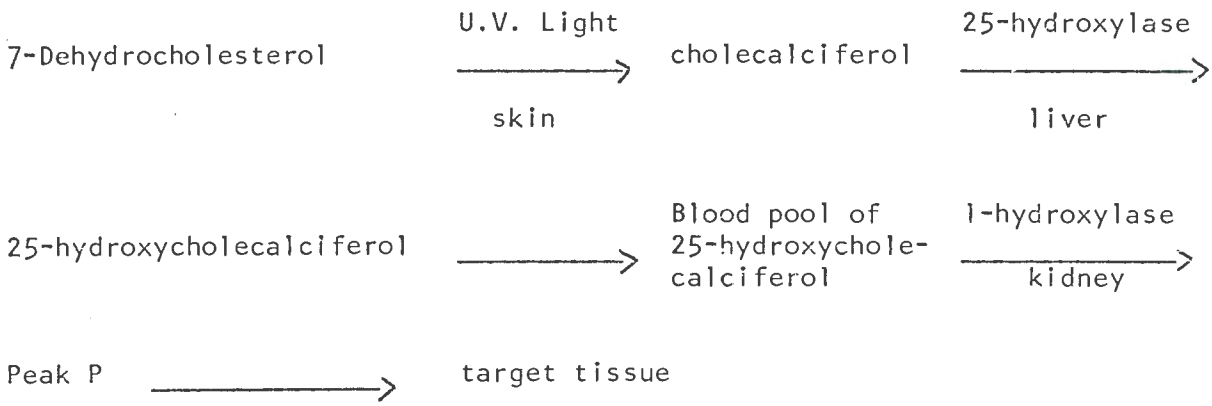
Early experimental studies on rats using vitamin D labelled with radioactive C^{14} showed that the lipid soluble radio-activity in the tissues was due almost entirely to vitamin D, cholecalciferol, (Kodicek and Ashby, 1960). Later, in very sophisticated studies on rats and chicks, Lawson (1971) found that after drastic reduction of the previously high dosage of labelled vitamin D administered, he isolated the major polar metabolite of cholecalciferol, namely, 25-hydroxycholecalciferol (25-HCC). This metabolite had 'about 40% more biological activity than cholecalciferol.' It was reported that on chromatograms of intestinal lipids the presence of a 'peak more polar even than 25-hydroxycholecalciferol ... accounts for the major

part of the radioactivity (60%).' This polar chromatographic fraction was called peak P, and was quite distinct from that occurring in blood lipids. Peak P, however, did not raise serum calcium levels any more effectively than equivalent doses of cholecalciferol and 25-hydroxycholecalciferol, which, the authors concluded, raised the 'intriguing possibility' that the mobilisation of calcium from bone by vitamin D is not mediated through peak P. Of many tissues studied, only kidney had been found to be involved in the formation of peak P.

Lawson (1971) indicated the various steps in the biosynthesis of peak P to be as follows:-

Biosynthesis of peak P

(polar chromatographic fraction from kidney lipids, after Lawson (1971))



As may be seen from the diagram, three tissues are largely responsible for the metabolism and 'synthesis' of vitamin D and its polar metabolites. The skin is essential for the conversion of the provitamin to cholecalciferol, whereas only the liver is said to be able to form the 25-hydroxy-derivative (Horsting and DeLuca, 1969);

this metabolite is rapidly secreted into the plasma. The third tissue is the kidney, which appears to be solely responsible for the conversion of the 25-hydroxycholecalciferol into the polar metabolite known as peak P, of which only 'finite amounts are found in the target tissue', thereby indicating a specific, but as yet unknown control over its production.

This final step in the biosynthetic sequence led Lawson (1971) to put forward that 'vitamin D, in the form of its metabolite, at present known as peak P, should be reclassified as a hormone since it fulfils all the necessary criteria; it is secreted in small amounts by only one tissue and is carried by plasma to its target tissues, with the whole process being under fine control.'

The proposed hormone became known as peak P in Kodicek's laboratory (Fraser and Kodicek, 1970); as peak V in DeLuca's laboratory (Ponchon et al., 1970); and as metabolite 4B in Norman's laboratory (Norman, 1971). The new hormone is highly active not only in facilitating the absorption of calcium through the intestinal mucosa, but also in stimulating the resorption of bone calcium. However, it is found only in minute quantities in bone, and as yet, is not thought to play a major metabolic role (Norman, 1971).

At the 4th International Parathyroid Conference held at North Carolina, U.S.A., in March, 1971, new developments in the understanding of vitamin D_3 were reported by Parfitt (1971). Three dihydroxy metabolites of 25-HCC have now been isolated from blood. They are (i) 21,25-DHCC acting only on bone, (ii) 25,26-DHCC acting only on gut, and (iii) 1,25-DHCC about which far more is known, and which acts

on both gut and bone, but is not capable of curing rickets. It is of interest that 5 new analogues of vitamin D synthesised with 1 or 2 hydroxy groups did not show anti-rachitic activity (Bontekoe et al., 1970).

Recent experimental work on parathyroid hormone and its relation to vitamin D metabolism has also opened up intriguing possibilities. On rats, Galante et al. (1972) demonstrated a direct effect of parathyroid extract on vitamin D metabolism. It was found that parathyroid hormone suppressed the conversion of the circulating form of vitamin D (25-HCC) by the kidney to 1,25-DHCC. It enhanced the conversion to 21,25-DHCC; this was regarded as a direct action of the hormone on kidney cells. 'Intracellular calcium concentration is the major controlling factor in the type of metabolite produced ... '; on a low calcium diet 1,25-DHCC was maximally produced, but on a high calcium diet rats converted 25-HCC to 21,25-DHCC, a metabolite much less active on bone and gut. In a leading article (Anon., 1972a), it was suggested that the possibility of such a mechanism being operative in man would provide a means of adaptation to different levels of calcium intake, and could explain the severity of bone disease found in chronic renal failure. Assays of 25-HCC by a specific protein-binding technique can now be carried out in human plasma and are proving specific (Belsey et al., 1971; Haddad and Chyu, 1971). According to Stamp and Round (1973) 'they provide the most accurate available index of vitamin D nutritional status in man.'

With the recent improvements in understanding of vitamin D and its metabolites, in respect of their function in the target organs,

knowledge of the role that they play in each step of the fascinating story of bone metabolism, will promote a greater understanding of what has for long been puzzling, namely, the causation and treatment of D-resistant rickets. A number of questions arise. Will there be a breakthrough in understanding to permit satisfactory treatment? What step in the pathway of synthesis of the 'newest' of hormones, peak P (1,25-DHCC) will prove to be defective? Parathyroid hormone controls the amount of 1,25-DHCC produced by kidney cells, so that a close interrelationship probably prevails between vitamin D, parathyroid hormone and calcitonin (Galante et al., 1972; Anon., 1972a).

In the treatment of rickets, the polar metabolites of vitamin D_3 have proved to be more effective than the parent vitamin D_3; they also act more rapidly (DeLuca, 1971). Already smaller doses of 25-HCC (the major plasma metabolite of vitamin D) of the order of 5,000 - 25,000 I.U., have been used effectively for conditions such as vitamin D-resistance, pseudovitamin D deficiency, hypoparathyroidism, Fanconi syndrome and renal osteodystrophy. Although the amounts required are larger than the physiological dose, they are, nevertheless, smaller than those of vitamin D_3 (cholecalciferol) currently employed, which range from 25,000 - 75,000 I.U. daily (West, 1971). For example, in vitamin D-resistant rickets, Menkign and Sotos (1969) used 25,000 - 600,000 'units' vitamin D_3 daily. This manner of treatment, which is often ineffective (De Prá and Canepa, 1961; Frederick et al., 1962), together with reports of vitamin D poisoning following the administration of large doses (Fateeva and Tatočenko, 1960), are disturbing, as is the fact that reactivation of the condition may occur when treatment is discontinued. In a case of twins, with

familial hypophosphataemic rickets, it was found that continued high doses of vitamin D in one twin exacerbated the condition, whereas in the untreated twin the condition improved (Stickler et al., 1971). In the unsuccessful treatment of 5 patients with familial hypophosphataemic and vitamin D-resistant rickets using 25-HCC, it appeared that a defect in the conversion of cholecalciferol may not be the only cause of the metabolic abnormalities prevailing (Earp et al., 1970). It has been recently suggested (Anon., 1972a) that the overt bone disease seen in chronic renal failure may be due in part to interference in the metabolism of vitamin D by high circulating levels of parathyroid hormone. The possibility that hyperparathyroidism may have a role in the bone condition seen in rickets has been suspected for a long time (Albright et al., 1948; Taitz and de Lacy, 1962), but convincing proof has only recently been forthcoming (Galante et al., 1972). In South African Bantu Joffe et al. (1972) found elevated parathyroid hormone concentrations in cases of nutritional rickets. Serum immunoreactive parathyroid hormone levels have been found to be raised in infants with vitamin D deficiency (Arnaud et al., 1972).

As already indicated, a good deal of space has been devoted to this section in view of the considerable progress made recently compared with the very slow increase in knowledge in the past.

1.4.7. Description of Types of Rickets

There are two broad categories of rickets, (i) nutritional rickets, and (ii) vitamin D-resistant rickets. The former is self-explanatory, and arises as the result of a vitamin D deficiency, whether of dietary or solar origin, or both. The disease is readily

responsive to vitamin D. The majority of cases of rickets are of this category. D-resistant rickets includes a group of diseases, attributable to vitamin D deficiency, but which are usually associated with a systemic defect which may block the effective utilization of even an abundant supply of the vitamin.

Nutritional Rickets

Rickets in infancy. A nutritional deficiency may occur at various periods of life. The most usual age of onset is during infancy (Chick et al., 1923); the disease seldom appears before 4 months and most often between 6 - 18 months. Recently, a Leading Article (British Medical Journal, 1971b) described rickets in premature babies while still in hospital. The disease has also been noted in low-birth-weight infants (Lewin et al., 1972).

Adolescent, juvenile or late rickets (rachitis tarda). The first recognition of this disease has been attributed to Sir William MacEwan (after whom an operative procedure for knock knee was named - MacEwan's operation). The disease, which begins at adolescence, was called Rachitis adolescentium (Ogston, 1888). Manifestations of active rickets have been described in older children before and at adolescence by many observers (Clutton, 1906; Chick et al., 1923 ; Antia, 1970; Vaishnava and Rizvi, 1971; Ford et al., 1972). In 1906, Clutton gave a description of adolescent rickets and regarded it as a new manifestation of the disease and not as a flare up of an old condition. On the other hand, Parker (1888) and Dick (1922) thought that rickets could flare up at puberty and they blamed the residual laxity of ligaments, a residium of experience of rickets of early life. The

extra mass-bearing imposed on young people in 'heavy' occupations, particularly boys, at a time of rapid growth, was considered to be responsible for bone deformities occurring at this period. In Vienna, Dalyell and Chick (1921) described a condition known as hunger osteomalacia, which occurred in adolescents but more particularly in old people, and which was directly linked with the severe dietary deprivation of vitamin D that occurred in the city at that time. From more recent literature, it would appear that this condition occurs in many areas. In Pakistani and Indian families living in Glasgow, late rickets has been reported by Dunnigan et al. (1962), also by Ford et al. (1972a, 1972b), and in Birmingham by Felton and Stone (1966) and Swan and Cooke (1971b). The disease has been reported from Nigeria by Antia (1970). The condition is recognised locally in Johannesburg (Joffe et al., 1972).

Osteomalacia or adult rickets. Severe osteomalacia occurs where women remain in dark confined surroundings. This has been reported from China (Maxwell, 1923); India (Wilson, 1929, 1931; Wilson and Widdowson, 1942; Vaishnava, 1967); Israel, in Bedouin in the Negev Desert, (Groen et al., 1965); also in Asians in Britain (Vaishnava and Rizvi, 1971; Teotia and Teotia, 1972; Ford et al., 1972b; Holmes et al., 1973).

Nutritional deprivation of vitamin D was stated to be the causative factor in the 'hunger osteomalacia' reported by Dalyell and Chick (1921) in Vienna during 1918 - 1923. Non-nutritional osteomalacia has been reported by Swan and Cooke (1971b) in Asian immigrants 'who appeared to be taking an adequate amount of vitamin-D-containing foods,

but nevertheless had florid rickets in an otherwise well-nourished body.' As with rickets, osteomalacia may be of two types, nutritional and vitamin-D resistant. Chamberlain and Hosking (1971) noted a response in 6 of 11 patients to small doses of vitamin D_2 (50 µg/day); however, 5 responded only to much larger doses of 1,25/mg/day (without signs of toxicity developing over 3 years) before healing, refleted clinically and biochemically, occurred. Hypophosphataemic osteomalacia typical of the sex-linked form of 'vitamin D-resistant rickets, with a high renal clearance of phosphate' has been reported by Dent and Stamp (1971).

The active phase of the disease affects the long bones, spine, ribs and pelvis. Pregnant women are particularly prone (Conybeare, 1945). A lack of vitamin D and calcium available for metabolism occasionally occurs as a result of steatorrhoea, in which condition the vitamin and calcium tend to be less well absorbed. The abosrption of these nutrients is so inadequate that adult rickets can occur (Guyton, 1961).

Vitamin D-resistant rickets

This section will be accorded a relatively large amount of space specifically on account of considerable current interest in the problem.

With the advent of adequate public health control in the late 1920's, as already described, the prevalence of nutritional rickets showed a sharp decline, with the consequence that vitamin D-resistant rickets began to be studied in much greater detail. Many publications have described this form of rickets which includes several different conditions all characterised by the typical rachitic replacement of bony tissue by osteoid; by the easy fracture of long bones, particularly

mass-bearing bones, also by other symptoms usually present in the nutritional syndrome. No differences are found between the two types of rachitic manifestations, whether clinically, histologically or radiologically. The distinction lies solely in the response to vitamin D therapy. Resistance to vitamin D therapy can arise not only as a primary disorder, but also as a disturbance secondary to conditions such as chronic renal failure and steatorrhoea (Anon., 1971c). However, the prevalence of vitamin D-resistant rickets in the general community, remains unknown, as no relevant studies have been undertaken.

In nutritional rickets, the vitamin, whether derived from sunlight or diet, or both, evokes an almost immediate response and healing occurs within a few weeks. On the other hand, the D-resistant forms of rickets do not respond readily. Indeed, large doses are often required to procure minimal response, and relapse may occur on cessation of treatment (De Prá and Canepa, 1961); Frederick et al., 1962). In some instances the largeness of the dose has proved toxic and has had to be discontinued (Fateeva and Tatočenko, 1960). In certain cases retrogression of symptoms has resulted with exacerbation of the condition (Stickler et al., 1971).

The D-resistant group of rickets includes:-

1) (i) Hypophosphataemic rickets (familial refractory)
   (ii) Congenital rickets
   (iii) Pseudovitamin D-resistant rickets

2) Chronic renal rickets (tubular rickets with related osteodystrophy) arising from:-

(i) Renal failure

(ii) Fanconi syndrome

(iii) Cystinosis

3) Hypo- and hyper-parathyroidism

4) Hypocalcaemic rickets

5) Coeliac disease

Although they are not of great importance, in terms of public health importance, it is proposed to give a brief description of the conditions cited.

Hypophosphataemic rickets. This syndrome appears to be an inherited or familial condition which is characterised by a low serum phosphorus level. It responds to treatment with phosphate compounds and is unrelated to an abnormal metabolism of vitamin D (Condon et al., 1971). The primary abnormality is a partial block in the intestinal absorption and renal tubular reabsorption of phosphate.

Congenital rickets. Sir William Jenner, lecturing on rickets in 1895, was emphatic that he had never seen a case of congenital rickets and moreover he knew of no factors that indicated rickets to be hereditary. Congenital rickets has since been recognised (Anon., 1968). Congenital hypophosphataemia is now known to be an inherited condition with a family history compatible with sex-linked dominant transmission (Anon., 1971a). Lapatsanis et al. (1968) have raised the interesting question - is there a locus on the X chromosome that has something to do with sensitivity to vitamin D? Earlier, in an excellent review,

Childs (1965) stated 'But it is undeniable that there is a locus whose mutants cause vitamin D-resistant rickets', and, discussing the possible role of this locus in vitamin D deficiency rickets he said '... but if they have some effect, however remote, on sensitivity to vitamin D, the possession of only one as opposed to two such genes might predispose more males to vitamin D deficiency rickets. The possession of two genes by the female may be part of the explanation of less rickets in females.' A further point of interest is - is the gene for skin colour in some way involved? That there is a gene for skin colour is known, and also that it is sex-linked; however, the linkage is very complicated and involves possibly some six genes (Jenkins, 1973 - personal communication).

Pseudovitamin D deficiency rickets. This is an unusual hereditary osteomalacic disease, with low serum calcium, but normal serum phosphate levels, and normal phosphate excretion index. In certain cases it has been found that vitamin D restored serum calcium to normal levels and reduced pain in bone (a feature of the condition) (Birtwell et al., 1970).

Chronic renal (tubular) rickets. There are three main syndromes in which this type of rickets occurs, namely, (i) renal failure, (ii) Fanconi syndrome and (iii) cystinosis. These conditions are associated with a related osteodystrophy which displays rachitic symptoms. The onset of renal rickets is insidious, and may not be observed until the age of 6 or 7 years. Genu valgum is usually the most marked bony lesion. Long-standing renal impairment induces increased excretion and diminished absorption of calcium which is

thus withdrawn from soft growing bones, with the production of characteristic bony deformities. Ultraviolet radiation aggravates the bony lesions (Conybeare, 1945). A possible explanation of the mechanism of renal rickets has been put forward by Guyton (1961). The kidney disease prevents normal excretion of acids and bases and leads to an acidosis. Due to this condition most of the monohydrogen-phosphate (HPO_4^-) of the blood is converted into the dihydrogen-phosphate ($H_2PO_4^-$), which is not precipitated in bone. The essential picture is thus a form of progressive osteomalacia.

It is now thought that a metabolic block possibly occurs in the formation of the newly proposed kidney hormone 1,25-dihydroxy vitamin D_3 (1,25-DHCC) (Brickman et al., 1972). Damaged renal cells are unable to respond to circulating parathyroid hormone (Galante et al., 1972), which is the controlling factor in the formation of 1,25-DHCC, and therefore possibly of calcium homeostasis (Anon., 1973b).

Renal failure. In the disease in children, rachitic symptoms are sometimes seen, due to the rapid growth rate of bone. But in adults, bone involvement seldom occurs as the renal failure proves fatal before such symptoms can become marked.

Fanconi syndrome. Fourman and Royer (1968) have described the defects of the renal tubular function as follows - 'When multiple defects of renal tubular function exist involving the reabsorption of phosphate, potassium, glucose, amino acids and water, the syndromes are usually named after De Toni, Debré and Fanconi.' The Fanconi syndrome is associated with an aminoaciduria (Dent, 1970).

Hypoparathyroidism. In this condition the parathyroid glands do not secrete sufficient parathyroid hormone and the osteoblasts of bone become inactive. Bone reabsorption is depressed and the level of calcium in the body fluids decreases. The formation of new teeth in the growing child almost ceases, possibly because of the decreased absorption of calcium from the renal tubules and gut caused by deficient quantities of parathyroid hormone. Such patients are also slow to calcify the organic matrix of new bone, possibly due to the same mechanism. In the treatment of this condition, however, parathyroid hormone is seldom used as it is very expensive, and difficult to obtain. It is only effective for 24 - 36 hours, and the body has a tendency to develop immune bodies against it, making it thus progressively less and less active. The condition can be treated satisfactorily with large quantities of vitamin D, as this increases absorption of calcium from the gut, and has a weak effect similar to that of parathyroid hormone in promoting calcium and phosphate resorption from bone (Guyton, 1961). One of the vitamin D compounds derived from ergosterol, dihydroxytachysterol, is used in treatment. This compound has properties similar to those of the blood metabolite of vitamin D_3 - 21,25-DHCC (Norman, 1971; Parfitt, 1971), formed in the liver, which stimulates calcium absorption from bone.

Pseudohypoparathyroidism. This is a recently described condition similar to true hypoparathyroidism, but characterized by greater than normal secretions of parathyroid hormone. In spite of this, patients have hypoparathyroid stigmata, e.g. a low serum calcium level, a high blood phosphate level, and a tendency for a reduced rate of bone

growth probably because of depressed osteoblastic activity. The cause is not known, and patients do not respond to parathyroid hormone, but to dihydroxytachysterol and calcium therapy (Guyton, 1961). This condition can now be explained in the light of work by Galante et al. (1972), namely, that an increased 21,25-DHCC production is stimulated by an increased parathyroid hormone activity.

Primary hyperparathyroidism. Some patients with hyperparathyroidism have been reported to respond to small doses of vitamin D over a long period, 11 - 16 months (Woodhouse et al., 1971). Remineralisation of bone occurred; moreover, bone lesions healed and serum alkaline phosphatase levels returned to normal. A vitamin D deficiency is possibly responsible for the development of bone disease in some of these patients; there may be an increased requirement for the vitamin. The explanation probably lies in the control of 1,25-DHCC exerted by parathyroid hormone (Galante et al., 1972).

Secondary hyperparathyroidism. A low concentration of calcium ions in the blood directly increases the secretion of parathyroid hormone. Therefore, any factor causing a low level of serum calcium initiates the condition known as secondary hyperparathyroidism. This may result from (i) a very low calcium diet, (ii) pregnancy, (iii) prolonged lactation, (iv) rickets and (v) osteomalacia. The hyperplasia of the parathyroid glands is a corrective measure for maintaining calcium homeostasis, which is mediated by the action of parathyroid hormone on kidney cells (Galante et al., 1972).

Hypocalcaemic rickets. This is an extremely rare condition. Only one report of a case in a young child has been published (Maltz,

et al., 1970), in which serum calcium fell to 3mg%, tetany may develop, and, unless intravenous calcium is administered the condition may prove fatal due to a tetanic respiratory spasm. Immediate relief, however, is obtained by the administration of calcium salts. Hypocalcaemia is not uncommon in dairy cattle after long lactation.

Coeliac disease. Although this disease is not a common cause of rickets, it can lead to secondary rachitic or osteomalacic symptoms. Rickets in children with coeliac disease does not respond to sunlight (Fanconi, 1928). An abnormally inadequate level of mucosal activity leads to the greatly increased amount of fat excreted in the faeces. Vitamin D is poorly absorbed and calcium excreted in large amounts. It is widely believed (but not proved) that calcium forms insoluble salts with fatty acids (Parfitt et al., 1962). Malabsorption is an important cause of osteomalacia, but early diagnosis results in the condition being recognised before bone disease develops. Treatment with vitamin D does not prove very effective as both the vitamin and calcium are poorly absorbed. Even in the presence of vitamin D therapy, calcium absorption remains poor. Nassim et al.(1959) have shown that this defect is not necessarily related to the proportion of fat in the faeces; there may also be an abnormally large endogenous secretion of calcium into the gut (Fink and Laszlo, 1957).

1.4.8. Rachitic Manifestations

In the growing child failure of normal ossification can result in bending of the soft bone by normal pressure variation 'as the rachitic metaphysis is soft and easily bent, compressed and deformed' (Caffey, 1961). The amount of rarefaction and hence the severity of

the abnormality are dependant firstly on the degree of deficiency of vitamin D, or,rather, of its active metabolite, secondly, on the stage of growth of the child, and thirdly, on the individual vitamin D requirement, which is known to vary.

The following abnormalities have been considered as manifestations of rickets.

Abnormalities of the lower limbs

Genu varum (bow legs) and genu valgum (knock knee). In a very detailed discussion of the mechanism responsible for the production of bow legs, knock knee and spinal curvatures, Arbuthnot Lane (1889) differentiated between mild or physiological variants and those caused by rickets. The first group he considered were due to 'the fixation and subsequently the exaggeration of a normal physiological attitude of rest.' The second is due to rachitic involvement of bone.

Prior to walking, the axes of the femoral shafts normally converge slightly towards the knee. On walking this natural bias may tend to become exaggerated, but in infants it is more common to find a general outward bowing from hip to ankle. In an attempt to explain the causation of these leg abnormalities, Dick (1922) considered that because ligaments are lax and muscle tone diminished in rickets, an easy separation of joint surfaces occurs. The older rickety infant frequently sits, tailor fashion, with feet crossed at the ankle, and knees drawn up. In this position the external condyles of the femur separate from the articular surfaces of the tibial tuberosity thus diminishing the pressure on the outer side of the knee with resultant osteoid overgrowth

of the external condyle. If the child now struggles to his feet while the bones are soft and such a bias is present, the mass of the body will cause a bowing outward of the legs. If, on the other hand, the rachitic child crawls on hands and knees (frequently for a prolonged period) the inner condyles bear on the floor and the reverse action takes place with a resultant knock knee.

There are two periods of very active bone growth; the first is from birth to 2 - 3 years and the second during adolescence, 10 - 15 years. The latter growth spurt is well described by Tanner (1962). It occurs in girls from about 10 to 13 years, and in boys from about 12 - 15 years. Peak height velocity is reached in girls of 12,1 and boys 14,1 years, respectively (Tanner et al., 1966). Sexual functions develop at this time. The growth spurt is part of the complex accompanying puberty (Tobias, 1972). If a vitamin D deficiency should occur during the pre-pubertal growth spurt it is conceivable that the above situation may recur in the lower limbs; these may tend to cause either a bowing or knock knee (Ogston, 1888; Clutton, 1906; Ford et al., 1972b). Such cases are known and this condition is referred to as late rickets or rachitis tarda.

Coxa varum. In the adult the normal angle of the neck of the femur is 125°; this reduces with age to approximate to a right angle. In rickets the angle tends to become less obtuse as in age, due to the natural pressure of body mass on rachitic bone. The condition is not very common and occurs at about 13 - 17 years. It may be congenital.

Genu recurvatum. This is a condition in which the knee is

habitually over-extended. Although it is commonly found in rickets, it may also be found associated with club foot, infantile paralysis, and other limb deformities. Double jointedness is also considered to be due to the laxity of ligaments in rickets. Many unusual positions can be assumed and held with ease.

Pes valgus (flat foot). This condition not infrequently follows as a result of rickets (Ogston, 1888; Lane, 1889). The foot turns over easily and there is frequent undue mobility of the ankle. The attitude of abduction persists and after a time becomes fixed. Symptoms of flat foot do not often supervene until adolescence. Ogston (1888) believed that flat foot was seldom due to a cause other than rickets.

Abnormalities of the chest wall

Asymmetry of chest. This is common in rickets. There is a tendency to flattening of the left apex with a projection of the chest wall to the right side of the lower part of the sternum where it meets the true ribs. This may be a result of scoliosis, but which apex is flattened, and/or which shoulder raised, varies with the form of lateral curvature present.

Elevation of manubrium sterni. This slight deformity gives the chest the appearance of being tilted forward and raised. It occurs mainly in long-chested adolescents and may persist throughout life.

Rachitic rosary or beading of the ribs. This is a very early and constant sign of rickets, and is characterised by a nodular swelling of the costochondral junction. The swelling, usually more

marked on the pleural surface, can therefore readily be missed by the examining finger. The very early and considerable thickening which takes place at the costochondral junction, is possibly an attempt on the part of 'nature' to buttress the ribs at their weakest point.

Pigeon breast. This condition was first described by Glisson (1650) who likened it to the prow of a ship. A longitudinal groove develops on the antero-lateral surface of the chest, just outside the nodules of the ribs, and extends from the 2nd or 3rd rib above to the 9th or 10th below. The groove passes downwards and outwards and may be shorter on the right, owing to the liver, and shallow on the left, due to the subjacent heart. This deep groove tends to thrust the sternum forward, producing different forms of pigeon breast.

Harrison's sulcus. This is a transverse constriction beginning at the xiphisternum and passing outward and slightly downwards just below the 6th rib, and, as a rule not reaching as far as the mid-axillary line. In normal respiratory action, during inspiration, the chest expands and the diaphragm contracts tending to protrude the abdominal wall. The elastic recoil of the healthy abdominal wall is an important factor in expiration. In rickets there is little or no movement of the abdominal wall, and this aid to respiration is lost. The action of the diaphragm tends to become reversed, acting from its origin, the central tendon, instead of from its insertion mainly into the ribs. This reversal of action draws the softened ribs inwards. The transverse sulcus can be seen

forming, particularly during respiratory distress. External air pressure on the weakened thoracic cage aids in its formation.

Gutter-shaped and funnel-shaped chest. The gutter-shaped chest occurs when the sternum, plus a small portion of the ribs, sinks en bloc into the chest - it is an acute condition occurring in scurvy and syphilis. More commonly, the lower part of the sternum is markedly retracted forming a funnel-shaped chest or, thorax en entonnoir. If whooping cough supervenes on rickets, a true funnel-shaped deformity not infrequently remains, in which the xiphisternum and lower part of the mesosternum are deeply retracted. This condition, however, is thought by Jelliffe (1966) to be non-nutritional in origin, but he may be referring to the congenital form known as cobblers' hollow, which has no specific connection with rickets.

Scapulae. These bones may become swollen and softened in rickets. Winging of the scapulae is thought to occur due to loss of muscle tonicity, and has been regarded as one of the stigmata of rickets (Cheadle and Poynton, 1913, Leary, 1969). Winging may be one of the early noticeable indications of a lateral scoliosis or a kyphosis and is often associated with a raised shoulder.

Clavicles. The inner two thirds of the clavicles normally curve forward and are bent somewhat upwards, whilst the outer third curves backwards. These normal curves become exaggerated and a characteristic projection of the acromial end is often very marked in the shoulder girdle of a rickety child, especially where the mass of the trunk has been thrown onto the upper extremities.

Spinal curvatures

Kyphosis. Convexity of the upper spine may be due to a faulty attitude rather than to an actual deformity. It may, however, develop at adolescence with rounded shoulders and arching of the back in the upper dorsal or cervico-dorsal region. The chest is flattened and concave, and the arms tend to hang forward and away from the body. The scapulae may be somewhat winged and prominent, but as noted above, this may also be associated with a scoliosis.

Lordosis. A lordosis of the lumbar region may occur once the child starts walking. Lane (1889) has named this condition dorsal excurvation, which has a strong tendency towards healing by the 4th or 5th year.

Scoliosis. This is more often seen in adolescence than earlier. The convexity is often to the right with a compensatory curve to the left. A 'high' shoulder or hip, or winged scapulae, may draw attention to its presence. Although torsion of the trunk on standing upright is visible it becomes more noticeable and may be prominent when the body is bent forward.

Pelvis. Deformity of the birth canal can occur due to rachitic involvement of the pelvis, causing a flattening or dimunition of the conjugate diameter. This may not only endanger the life of the unborn baby, but labour may be more difficult resulting in injury and infection in the mother. Caesarean section may have to be resorted to. This deformity can conceivably occur whilst the rickety child is still in the sitting position. Pressure from body mass

broadens the sacrum sideways with concomitant narrowing antero-posteriorly resulting in a narrowing of the pelvic inlet and giving rise to a wide gaping pelvic outlet.

Bones of the face and skull

Superior maxilla and teeth. In rickets the chief changes occur in the superior maxilla. In the rachitic skull, overcrowding defects in the development of the permanent teeth in the superior maxilla are already present by the 2nd year, although it is not until the 6th year that the first permanent molar erupts. Caries may be common even prior to eruption (Dick, 1916). The possibility of mastication being involved in the production of these defects is thus entirely ruled out. The teeth themselves, still in the jaws in the 2nd year, also show signs of stunting and develop enamel defects, but they are only exposed to detrimental oral milieu once they erupt.

Mouth breathing and adenoids have long been almost synonymous. Both result in the loss of an air current through the nasal passages.

Bossing. The frontal and parietal bones, either separately or together, are the usual ones affected in rickets. Due to a hyperostoses, marked bossing of these bones may occur which does not regress in later life.

Craniotabes. Softened areas appear in the cranial bones, which, on pressure, have, to the examining finger, the characteristic feeling of depressing a ping-pong ball. If severe, a 'parchmentlike crackling' can be felt. Rapid recovery is usual, and after about 6 - 8 months little craniotabes is found in infants.

Craniotabes has been considered by some to be rachitic in origin, but this remains controversial. Hess and Unger (1922b) regarded it as an unreliable sign, but Dalyell and McKay (1923) considered it indicative of rickets. Later workers, such as Bille (1955) disagreed, but Kaplan (1964), and Davidson and Passmore (1969) are in favour of it being rachitic in origin.

1.4.9. Prognosis of Rickets

Florid rickets used to be regarded as a serious disease of infancy, and was associated with high mortality (Jenner, 1895). But today, with the greater understanding of its aetiology and treatment, both specific and preventive, the disease is no longer feared. A description of rickets in the older child and its importance to health, are cited below.

The sequelae of rickets were described by Glisson (1650) and were seen in 'the older child, dwarfed, distorted and bow-legged.' This is still referred to as 'Glissonian' rickets.

Referring to bone deformities in rickets Cheadle and Poynton (1913) wrote, 'The curvature of the spine and the long bones slowly straighten, and,if moderate, disappear altogether; although in more pronounced cases they remain throughout life.'

Writing on rickets in school-going Australian children, Sutton, in 1920, wrote, 'It cannot, however, be argued that the disease even in a mild form is of no importance. It leads to much secondary disease.'

Dick (1922), who worked extensively among schoolchildren in London

during 1918 - 1922, wrote that although rickets tended to end in recovery, '... the stigmata produced by disease are still exceedingly common in later years among schoolchildren.'

Recently in a Leading Article in the Lancet , (Anon., 1971a), the author stated, 'Rickets matters; it retards and distorts skeletal development and increases the severity of penumonia in babies.'

However, precise information of the morbidity burden, short term and long term, due to rickets, is lacking.

CHAPTER 2

PLANNING AND METHOD OF INVESTIGATION

2.1. CONSIDERATIONS IN PLANNING

The South African context. The Republic of South Africa is situated in the southern part of the continent of Africa. The four main population groups living for the most part in large cities and towns can be divided as follows (data from Division of Agricultural Marketing Research, 1973):-

South African Bantu	(resident in White areas)	8,033,200
	(resident in Homelands)	7,000,000
Whites		3,750,700
	(farmers)	90,000
Coloureds (Eur-African-Malay)		3,850,000
Asians		640,000

The climate is largely temperate, but there are subtropical areas situated mainly on the Eastern seaboard. Large sections of the interior are semi-desert with scanty rainfall, experiencing high day and low night temperatures. Johannesburg situated in the Southern Transvaal is on the highveld, experiencing a mean of 8,4 hours sunshine per day and has an average mean minimum temperature of 13,2°C (55,7°F). The Eastern Transvaal experiences much the same amount of sunshine, namely, 7,3 hours per day, but has an average mean minimum temperature of 18,1°C (64,6°F), about 5°C higher. Cape Town, situated on the southern tip of the continent, experiences 6,7 hours of sunshine per day and has an average mean minimum temperature of 18,9°C (66,0°F) (Weather Bureau : Department of Transport, 1968).

All the main cities have excellent medical services, including

large well equipped hospitals and clinics, catering for all sectors of the population. Most smaller towns have hospital facilities and adequate medical services. The situation in regard of rural areas is less satisfactory, and people from such areas often have to travel many kilometers to the nearest doctor, hospital, or clinic, particularly in Homeland areas, where one doctor may have to attend some 100,000 patients.

Previous investigations. A study of the nutritional status of Bantu pre-school children revealed the unexpected observation that some stigmata of rickets appeared as common amongst Bantu rural as urban children (Richardson, 1971). As mentioned previously, rickets is known to occur in large centres of population in South Africa (Feldman, 1950; Dancaster and Jackson, 1960, 1961, 1962; Taitz, 1962; Taitz and de Lacy, 1962; Robertson, 1969; Joffe et al., 1972). In rural areas Mission hospitals report the occurrence of rickets, but it is seen infrequently. Its sequelae are seen particularly in women who experience difficult labours, due to contracted pelvises (Homaker, 1973 - personal communication).

Since rickets is being diagnosed in ill children in hospital, it could be inferred that much larger numbers of presumably healthy children might be suffering from an unrecognised subclinical deficiency of vitamin D.

From observations on school pupils it was found that the limb abnormalities of bow leg and knock knee were common. As these conditions are part of the classical picture of rickets, it was regarded as essential to investigate the situation in interracial

groups of schoolchildren. No data have been published on the prevalence of rickets in random groups of the general population.

Preliminary survey. To assess the situation roughly, large numbers of rural Bantu schoolchildren were visually examined in several parts of the Transvaal for bow legs and knock knee.

It was soon apparent that a purely visual assessment of these deformities was of little value. It was therefore decided to actually measure the amount of bowing or knock knee present with subjects assuming a standard stance. Unfortunately, chest and other skeletal abnormalities arising from the rachitic syndrome do not lend themselves to direct measurement and reliance has had to be placed on clinical observation. Dietary, biochemical and radiological aspects are also being investigated as well as the effect of exposure to the sun's actinic rays, in specific groups.

Availability of subjects. The commonness of bowed legs and knock knee in rural Bantu schoolchildren emphasised the desirability of investigating the situation in greater depth, also among the other three ethnic groups available in South Africa, namely Coloureds, Indians and Whites. (The use of the term Indian has been explained on page 3, section 1.2.). Accordingly the necessary permission was obtained from the various State and Educational authorities. Attention has been confined mainly to three age groups, although in some cases all age groups have been investigated. The groups most intensively studied were 7, 12 and 16 - 17 year old pupils, representing pre-pubertal pubertal and postpubertal groups.

2.2. SUBJECTS AND METHODS

Investigations were concentrated on pupils aged 7, 12 and 16 - 17 years, however, children ranging in age from 7 to 20 years provided additional information for prevalences of bowing and knock knee, height and mass data. The several aspects of the investigations undertaken are discussed as follows:-

2.2.1. Preliminary survey

2.2.2. Dietary surveys

2.2.3. Prevalence of skeletal abnormalities; anthropometric and clinical observations

2.2.5. Skin pigmentation determined by reflectance photometry

2.2.6. Biochemical studies

2.2.7. Questionnaire survey on vitamin D prophylaxis, developmental patterns and infection rates

2.2.1. Preliminary Survey

Subjects

During a rapid visual assessment of the prevalence of bow legs and knock knee in Bantu pupils, ranging in age from 6 to 20 years, 1732 girls and 1387 boys were examined, totalling 3119 pupils. Of these pupils 839 lived in Phokeng, Rustenburg area; 361 at Belfast on the Highveld; 1919 in the Eastern Transvaal Lowveld. Details of pupils age and sex are set out in Table 1.

Table 1: Age and sex structure of rural Bantu pupils

Preliminary survey

Sex	7 years	12 years	16-17 years	6-20 years
Male	159	128	133	1387
Female	167	146	255	1732
Total	326	274	388	3119

Method

In order to quantify and help to establish the magnitude of the problem of bowed legs and knock knee groups of rural Bantu schoolchildren (total school populations) were asked to stand with feet together in the school playground. This was on as level a surface as could be found, usually reasonably flat ground, devoid of grass. Class groups were lined up and rapidly surveyed for obvious bowing or knock knee. Employing this method was reasonably satisfactory for the more marked degrees of bowing, but far less so for knock knee, as such individuals on trying to put feet together, overlapped the knees. This was not readily spotted in such a cursory and rapid examination, particularly in less severely affected cases.

2.2.2. Dietary Survey

Subjects

Transvaal. Representative groups of 16 - 17 year old girls in each ethnic group completed dietary questionnaire forms. Of these 30 were Bantu, 44 Coloured, 56 Indian and 38 White girls. As dietary pattern is unlikely to be different at various age levels, only senior pupils took part in the survey.

Cape Province. Dietary questionnaire forms, which emphasised varieties of fish eaten, were distributed to 58 12 year-old Coloured schoolchildren in high fish-eating communities, namely at Hout Bay and Kalk Bay, situated about 20 and 37 km, respectively, south of Cape Town; and to 93 pupils from low fish-eating areas at Elsies River, situated about 35 km inland, north of Cape Town.

Method

Transvaal. In the dietary questionnaire used, a list of foods known to contain even small amounts of vitamin D were presented; amounts, in grams, of foodstuffs, eaten per week, were recorded. Girls assisted by their domestic science teachers, as well as giving the above information, were asked to state approximate quantities eaten by younger siblings. This information must be viewed with caution and can give the general pattern only. If there was a baby, was he/she breast or bottle fed, and, where appropriate, the name of the infant formula or milk preparation used.

Cape Province. The dietary questionnaire used in this study, included a more detailed list of the different varieties of fish caught, and eaten, in Cape waters; it also listed other vitamin D-containing foodstuffs. Different varieties of fish are abundant during

the various seasons and are then freely partaken of by fishermen and their families. These include many varieties of 'fatty' fish, namely yellowtail, harders (mullet), Cape salmon, pilchards, mackerel, etc., which contain greater amounts of vitamin D than non-fatty varieties such as hake (stock fish), which contains negligible amounts of the vitamin. Pupils completing the questionnaire were, in this instance, assisted by their parents.

2.2.3. Prevalence of Skeletal Abnormalities : Anthropometric and Clinical Observations

Transvaal. A large series of children ranging in age from 7 - 20 years were used for observations on height and mass, also those of 7, 12 and 16 - 17 years were measured for the prevalence of abnormalities of the legs, namely bowing and knock knee. These pupils were drawn from the schools listed below. Within this larger series, on those children aged 7, 12 and 16 - 17 years, detailed anthropometric also clinical observations for skeletal abnormalities were made. Pupils from schools involved in these latter studies are marked with an asterisk (*).

Subjects

Bantu : rural groups (7 - 20 years)

(1) Rustenburg Area ($\pm$ 280 km northwest of Johannesburg)

*Tlaseng Lower Primary	240 pupils,	- 105 boys,	135 girls
Tshare Lower Primary	82 pupils,	- 40 boys,	42 girls
Saulspoort Lower and Higher Primary	295 pupils,	- 130 boys,	165 girls

*Ramatshaba Lower and Higher Primary	67 pupils,	- 27 boys,	40 girls
*Phokeng and Tumagole Higher Primary	110 pupils,	- 60 boys,	50 girls
*Bafokeng High School	135 pupils,	- 63 boys,	72 girls
Matale Secondary School	156 pupils,	- 80 boys,	76 girls

(2) Eastern Transvaal

Hectorspruit (± 512 km east of Johannesburg)

Hectorspruit Community School	137 pupils,	- 71 boys,	66 girls

Nsikazi Reserve (± 480 km east of Johannesburg)

*Mthimba Lower and Higher Primary	34 pupils,	- 14 boys,	20 girls
*Malekutu Lower and Higher Primary	63 pupils,	- 25 boys,	38 girls

Inkomazi Reserve (± 560 km east of Johannesburg)

Siboshwa Lower and Higher Primary	164 pupils,	- 86 boys,	78 girls
Legudlane Lower and Higher Primary	195 pupils,	- 102 boys,	93 girls
Inkomazi High School	289 pupils,	- 156 boys,	133 girls

Johannesburg Area (± 32 km east of Johannesburg)

Witkoppen Farm School	202 pupils,	- 95 boys,	107 girls

Bantu : urban groups (7 - 20 years)

These comprised groups of children from the following middle class urban schools situated in Soweto, Johannesburg.

*Sizanani Lower Primary	99 pupils,	- 50 boys,	49 girls

*Shalom Manne Lower and Higher Primary	161 pupils,	- 77 boys,	84 girls
*Tshedisomo Higher Primary	93 pupils,	- 46 boys,	47 girls
*Orlando West High School	85 pupils,	- 39 boys,	46 girls
*Orlando East High School	303 pupils,	- 124 boys,	179 girls
*Morris Isaacson High School	318 pupils,	- 122 boys,	196 girls

Coloured : urban groups (7 - 20 years)

These comprised groups of children from the following middle-class urban schools situated in Coronationville and Bosmont, suburbs of Johannesburg.

*Coronationville Primary and Junior Schools	200 pupils,	- 106 boys,	94 girls
*Bosmont 1st Primary School	154 pupils,	- 88 boys,	66 girls
*Coronationville High School	326 pupils,	- 198 boys,	128 girls

Indian : urban groups (7 - 20 years)

Children were from middle to upper class schools situated in Lenasia, 48 km south west of Johannesburg; also from Fordsburg, a suburb of Johannesburg.

*Progress Primary	204 pupils,	- 101 boys,	103 girls
Model Primary	240 pupils,	- 120 boys,	120 girls
*Nirvana High School	234 pupils,	- 156 boys,	78 girls
Trinity State High School	217 pupils,	- 148 boys,	69 girls

Fordsburg

*Bree Street Indian Government School	148 pupils,	- 74 boys,	74 girls

White : rural groups (7 - 13 years)

Children were drawn from schools situated at Komatipoort and Malelane, Eastern Transvaal Lowveld, some 480 - 560 km east of Johannesburg.

*Komatipoort Laerskool	180 pupils,	- 89 boys,	91 girls
*Malelane Laerskool	48 pupils,	- 21 boys,	27 girls

White : urban groups (7 - 20 years)

Schools, all situated in Johannesburg, were chosen from the upper, also middle income groups.

Upper

*St. John's Preparatory and Senior Colleges		209 boys,	
*Rodean			234 girls

Middle

*Arthur Matthews Primary	738 pupils,	- 375 boys,	363 girls
Franklin D. Roosevelt Primary	173 pupils,	- 93 boys,	80 girls
Roosevelt High School	194 pupils,	- 98 boys,	96 girls
*Sir John Adamson High School	244 pupils,	- 129 boys,	115 girls

Prevalences of bowing and knock knee

Children of 7, 12 and 16 - 17 years were drawn from schools

detailed above. Numbers of subjects are shown in Table II in their respective ethnic, age and sex groups. There were a total of 5573 subjects, namely 2873 boys and 2700 girls.

Table II : Age and sex structure of schoolchild population groups used for prevalence studies on bowing and knock knee

Group	7 Years			12 Years			16-17 Years			Total		
	M	F	T	M	F	T	M	F	T	M	F	T
Bantu												
Rural	253	260	513	251	256	507	291	315	606	795	831	1626
Urban	74	78	152	78	70	148	284	413	697	436	561	997
Coloured												
Urban	86	83	169	108	77	185	198	128	326	392	288	680
Indian												
Urban	111	134	245	131	115	246	304	147	451	546	396	942
White												
Rural*	36	38	74	21	28	49	-	-	-	57	66	123
Urban	194	159	353	230	230	460	223	169	392	647	558	1205
Total	754	752	1506	819	776	1595	1300	1172	2472	2873	2700	5573

* Only small groups of rural White children of 7 and 12 years were available for study. The majority of older rural pupils attend boarding schools in urban areas.

Standardization of method for leg measurements

In order to standardize measurements on legs, two although preferably three observers, including the author, measured several large series of children in urban and rural areas in the case of Bantu, and urban areas only in the other ethnic groups. The subjects included pupils aged mainly 7, 12 and 16 - 17 years, and comprised Bantu, rural, 893 pupils (416 boys, 477 girls); urban, 871 pupils (371 boys, 500 girls); Coloureds, 480 pupils (286 boys, 194 girls); Indians, 692 pupils (425 boys, 267 girls); Whites, 890 pupils (470 boys, 420 girls).

Anthropometric and clinical observations

Anthropometric and clinical observations (limited to the presence of bone deformities of the chest, spine and maxillae) were made on children of 7, 12 and 16 - 17 years, drawn from schools marked with an asterisk (*), as detailed in section 2.2.3. above and set out in Table III.

Table III appears on page 70.

Cape Province. Coloured children (301), from the high fish-eating communities at Hout Bay, Kalk Bay and Ocean View were measured for prevalences of leg and chest abnormalities. They comprised 100 (44 boys, 56 girls) of 7 years; 88 (52 boys, 36 girls) of 12 years; and 115 (55 boys, 60 girls) of 16 - 17 years, from the high fish-eating group. From the low fish-eating groups resident at Elsies River, there were 100 (52 boys, 48 girls) of 7 years; 98 (43 boys, 55 girls) of 12 years; and 218 (113 boys, 105 girls) of 16 - 17 years. Only the 7 and 12 year old children had chest examinations for skeletal abnormalities, etc., other than bowing and knock knee.

Table III: Age and sex structure of population groups used for anthropometric and skeletal abnormality studies

Group Area	7 years M	7 years F	7 years T	12 years M	12 years F	12 years T	16-17 years M	16-17 years F	16-17 years T	Total M	Total F	Total T
Negro												
Rural	92	105	197	91	90	181	85	63	148	268	258	526
Urban	72	73	145	60	55	115	55	65	120	187	193	380
Coloured												
Urban	86	83	169	108	77	185	49	49	98	243	209	452
Indian												
Urban	72	80	152	49	49	98	45	44	89	166	173	339
White												
Rural	36	38	74	21	28	49	-	-	-	57	66	123
Urban	70	61	131	66	87	153	96	52	148	232	200	432
Total	428	440	868	395	386	781	330	273	603	1153	1099	2252

Methods

The following methods were used in both the Transvaal and the Cape Province.

Anthropometric measurements

The height, in centimeters, and mass, in kilograms, were recorded. For measuring height, an height apparatus, consisting of a wooden base, upright, and sliding arm, placed at right angles to the upright, was used. For mass, a bathroom scale (checked regularly against an accurate laboratory scale) was used. Using a fibre-glass tape measure, measurements were made of circumferences of head, arm, wrist and mid-thigh (in cm). A spreading caliper* was used for measuring the width, in cm, of wrist and head, and a meter rule for cristal height. A Harpenden skinfold caliper was used to record the triceps skinfold thickness.

Leg measurements for bowing and knock knee

When making leg measurements, subjects stood either on a laboratory stool, form or flat chair. A standard stance was assumed, namely feet parallel, ankles touching when possible, and knees pressed back comfortably with no tension in the femoral muscle groups. When knees overlocked, as in knock knee, and there was obvious difficulty in apposition of ankles, the feet were separated and placed parallel, at a distance where knees touched comfortably (just touching), when pressed back, but were not under strain. The buttocks were not protruded so that the subject stood in an easily maintained, naturally upright position.

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* GPM Anthropological Instruments. Siber Hegner & Co. Ltd., 14, Talstrasse, 8022 Zurich, Switzerland.

Caliper measurements. Using a spreading caliper* (footnote: see page71) measurements in centimeters were made on the left leg at (i) the level of the femoral condyles; and (ii) across the lateral fibial and tibial malleoli; and (iii) at the widest part of the calf, approximately one third of the way down the lower leg.

Internal or 'gap' measurements were made with a sliding caliper* (footnote: see page71). The distances measured in centimeters were between (i) the medial femoral condyles, which for practical purposes is approximately level with mid-patella, provided the latter is allowed to assume its relaxed position and is not drawn up by femoral muscles, and (ii) at the medial tibial malleolli. When bowing was present, internal measurements were made at knee, but not at ankles, as they were in apposition. When knock knee was present the knees were in apposition and only ankle measurements could be made. When legs were 'straight' both knees and ankles were in apposition and thus no measurements were possible. From preliminary studies a grading system was formulated according to the distance of 'gaps' (internal measurement), both for bowing and knock knee. Gradings were, slight 0,5 - 1,4 cm; 1,5 - 2,4 cm; 2,5 - 3,4 cm; and 3,5 cm or more. Plates I to IV show the above grades as far as possible, for both bowing and knock knee in White 12 year old girls and boys - actual measurements of gaps are given in plate legends.

Plates I and II show various grades of bowing, and Plates III and IV various grades of knock knee.

Plates I to IV appear between pages 80 and 81.

Regarding repeatability, in measurements in 100 consecutive pupils, by two observers, the percentage difference in readings for 'gaps' of 2,5 cm or more at knee was 10% and at ankles 7,5%. The error lies almost exclusively in the positioning of the legs prior to measurements. However, a variable degree of pressure on the soft tissue can occur when measuring at the knee, especially in obese subjects, but far less so at the bony ankle.

Cardboard rectangles. When a number of observers were making 'gap' measurements, for prevalence studies, on large series of the same children, it was found to be more satisfactory to use cardboard rectangles, cut to specific widths. Rectangles were introduced into gap at knee or ankle and appropriate measurements noted. The widths used were 1,5 cm, 2,5 cm, 3,0 cm and 3,5 cm. Any 'gap' greater than 3,5 cm was measured with a small plastic ruler, being careful to avoid errors of parallax. As these latter measurements were always well above the abnormal cut off level of 2,5 cm, any such errors failed to influence prevalence figures.

The use of calipers or cardboard rectangles proved to give similar prevalence figures, and differences using either method were no greater than was found to be inherent within age and sex groups. The latter method was adopted for use in measurements made to standardize 'gap' data for prevalence investigations, as well as in measurements of large series of pupils by more than one observer.

It must be noted that unless extreme care was taken in positioning correctly, prevalence figures could vary widely.

Clinical observation for skeletal abnormalities

Children were examined for the presence of skeletal abnormalities of the chest, spine, maxillae and feet, by observation and palpation where applicable. No other clinical examination was undertaken.

Chest abnormalities. Pupils stood upright with body mass evenly distributed on both feet. Note was made of any skeletal abnormality affecting chest wall, clavicles, or scapulae, such as asymmetry of chest, Harrison's sulcus, pigeon breast, funnel-shaped sternum, exaggerated curves of the clavicles, and winging of the scapulae, etc.

Spinal abnormalities. Spinal abnormalities such as scoliosis, lordosis, or kyphosis, were noted in pupils of 16 - 17 years. If scoliosis was suspected the pupil was asked to bend forward from the hips; this procedure usually aided in the visual diagnosis. A hanging-bar and plumb line were not used to differentiate between functional and non-functional abnormalities. Whether the right or left shoulder was raised, was recorded in tabular form as raised shoulder only.

Maxillae. Examination of the jaws was made, noting the presence or absence of crowding of teeth and the type of bite, with upper jaw overlapping the lower (overbite); teeth in apposition (together), or lower jaw extending beyond upper (prognathous).

Flat foot. Note was made of flat foot (pes valgus). This was taken as being present if the inner border of the foot rested on

the form, bench or other flat surface. Observations were only made in groups of children of 16 - 17 years, as younger subjects proved difficult to position satisfactorily.

Leg pains (growing pains). Older pupils readily responded to questioning as to whether they ever experienced non-specific pains in the legs - not including injury from falls, accidents, sprains, etc. They were able to indicate satisfactorily where such pains occurred and when. Younger White children (7 and 12 years) responded readily, but Indian and Coloured children were very shy and appeared not to understand, thus further attempts to obtain information were abandoned. However, in one group of young Indian children the class teacher was able to obtain satisfactory replies. No attempt was made to distinguish between the sexes, and combined data are presented in the appropriate table. In the case of younger Bantu children even an interpreter proved unable to elicit the required information satisfactorily.

2.2.4. Radiological Studies

Subjects

X-ray plates of the left hand and wrist were taken, at the Mafolo T.B. Clinic in Soweto, of 96 Bantu pupils aged 16 - 17 years, from Morris Isaacson High School, Soweto, Johannesburg. Of these pupils, 32 (14 boys, 18 girls) had marked bowing (knee gap width 2,5 cm or more); 32 (15 boys, 17 girls) had marked knock knee (ankle gap width 2,5 cm or more); and 32 (12 boys, 20 girls) had straight legs (no gap at knee or ankle). Gap measurements were made at the standard stance

as detailed in 2.2.3. (cardboard rectangles).

Methods

X-rays of left hand and wrist were taken in the anterior-posterior position at a standard tube distance of 100 cm. Plates were examined for the presence of active rickets, and possible healed rachitic lesions, and osteoporosis by a specialist radiologist, Dr. J.I. Levy. The prevalence of osteoporosis was based on subjective impressions only, as no standardized radiographic factors, such as measurement by objective densiometry, were included. Each plate was examined twice. Osteoporotic changes were not expected in this group of young adult subjects, however, the criteria used by Dr. Levy, for its assessment, were as follows:-

Grade 1. This grade included those pupils who evidenced diminished bone density and coarse trabecular striation in the juxta-articular bone ends at inter - and metacarpal-phalangeal joints.

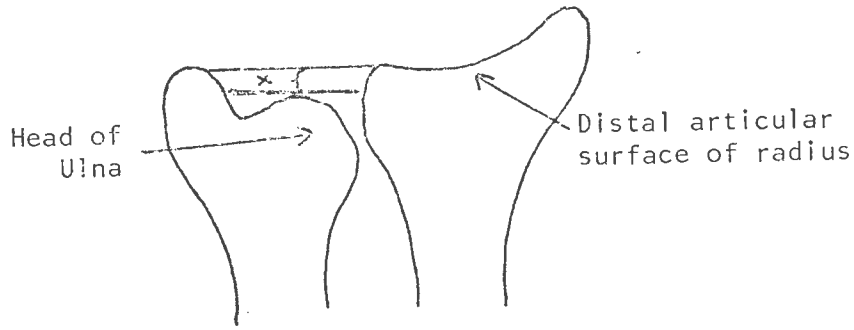
Grade 2. This grade included the above, and in addition, a similar appearance in the distal radius and ulna, also in the carpus.

Grade 3. This grade included both grades 1 and 2, and, in addition, the transverse width of the cortex of the second (index) metacarpal, at the widest part of the shaft, was found to be less than the transverse width of the spongiosa and medulla.

Where there was normal bone density, a diminished cortical width was considered to be an anatomical variant, and not a manifestation of osteoporotic change.

The distance in millimeters, where the head of the ulna did not reach the distal articular surface of the radius (a possible

manifestation of healed rickets), was measured, and graded, as indicated in the following diagram. X represents the above distance.



Grade 1. (X =<2 mm). The head of the ulna reached to within 2 mm of the distal articular surface of the radius.

Grade 2. (X = 2 - 4 mm). The head of the ulna was short of the articular surface of the radius by 2 - 4 mm.

Grade 3. (X =>4 mm). The head of the ulna was short of the articular surface of the radius by more than 4 mm.

Cortical thickness of the second (index) metacarpal was measured, using a standard viewing box to visualize the bone. Cortical scores were calculated by dividing the cortical thickness, namely, the difference of the outer width of the metacarpal (mid-shaft), D, and the inner (medullary) width, d, by the outer width, namely $\frac{D - d}{D}$.

2.2.5. Skin Pigmentation determined by Reflectance Photometry

Subjects

Skin reflectance of inner upper arm and forehead were measured on 94 urban Bantu 16 - 17 year old pupils, at filters 601 and 609, i.e. wavelengths of 425 and 685 nm, respectively. Of these pupils 32 (14 boys, 18 girls) had marked bowing, 32 (14 boys, 18 girls) had marked knock knee, and 30 (12 boys, 18 girls) had straight legs.

Method

A portable E.E.L. Reflectance Spectrophotometer, used for measuring human skin colour, was employed to measure the skin reflectance. Measurements at wave lengths of 425 and 685 nm, i.e. filters 601 and 609, were taken (i) on the medial aspect of the left upper arm, where there was little exposure to solar radiation, and (ii) on the forehead, a position of continued exposure to ultraviolet radiation (Weiner et al., 1964). About 10 minutes prior to measurement the skin was gently wiped with a soft, soapy cloth.

2.2.6. Biochemical Studies

Subjects

Transvaal. Groups of 16 - 17 year old urban Bantu pupils as detailed in 2.2.4. above, had blood samples collected. These comprised 35 with marked bowing of legs, 20 boys, 15 girls; 37 with marked knock knee, 20 boys, 17 girls; and 34 with straight legs, 17 boys, 17 girls. Serum alkaline phosphatase, calcium and inorganic phosphorus were determined on all blood samples.

Alkaline phosphatase was determined in groups of children of 7 and 10 - 12 years. Children of 7 years comprised 33 Coloureds (17 boys, 16 girls); 41 Indians (21 boys, 20 girls); and 12 Whites (9 boys and 3 girls). Children of 10 - 12 years comprised, 99 Bantu (52 boys, 47 girls); 45 Coloureds (23 boys, 22 girls); 177 Indians (87 boys, 90 girls); 17 Whites (9 boys, 8 girls). All groups were from urban areas.

Cape Province. Groups of Coloured children of 12 years, from

high and low fish-eating communities had blood samples collected. There were 51 pupils from high fish-eating areas, at Hout and Kalk Bays, of whom 13 (9 boys, 4 girls) had bowing; 19 (10 boys, 9 girls) had knock knee; and 18 (11 boys, 7 girls) had straight legs. There were 54 pupils from the low fish-eating community at Elsies River, of whom 19 (9 boys, 10 girls) had bowing; 16 (8 boys, 8 girls) had knock knee; and 19 (10 boys, 9 girls) had straight legs. Serum alkaline phosphatase, calcium and phosphorus were determined on all blood samples.

Method

Blood samples were collected from the antecubital vein, spun down and the serum separated. Alkaline phosphatase was determined as soon as possible after collection, i.e. within 18 hours, using the Ames Blood Analyser and the method modified of Bessey-Lowry-Brock. Serum calcium and inorganic phosphorus were processed on the auto-analyser. Serum was kept deep frozen ($-10^{o}C$) until required for the two latter determinations.

2.2.7. Questionnaire Survey on Vitamin D Prophylaxis, Development Patterns, and Infection Rates

Subjects

Questionnaire forms requesting information on vitamin D prophylaxis, development patterns, upper respiratory disease, infection rates, etc., were taken home by a total of 7384 schoolchildren for parents to complete. Pupils were sub-divided as follows, 933 Bantu (combined rural and urban groups); 1860 Coloureds; 1856 Asians; and 2735 Whites. Details of ethnic groups and sex appear in Table IV.

Table IV : Sex structure of schoolchild population groups: Questionnaire study

Group	Male	Female	Total
Bantu	430	503	933
Coloured	931	929	1860
Indian	895	961	1856
White	1325	1410	2735
Total	3581	3803	7384

Method

In this survey information was sought on the following aspects of the child's development and health. Questions related to (i) age at which the milestones of development, sitting, standing, walking and teething were reached; (ii) prophylactic measures practiced, such as the giving of vitamin D-containing syrups, etc., and upon whose advice this was instituted, medical practitioner, clinic sister, chemist, or on the mother's own initiative; (iii) upper respiratory conditions suffered, also if the child had experienced episodes of tonsilitis, adenoids, snoring and mouth breathing; (iv) information regarding filling and extraction of deciduous and permanent teeth; and (v) if arm and/or leg had been fractured.

PLATE I

Grades of bowing in girls of 12years.

Left to right - Straight legs, slight, 1,5 cm,

2,5 cm, 3,0 cm, and 3,5 cm.

PLATE II

Grades of bowing in boys of 12 years.

Left to right - Straight legs, slight, 1,5 cm,

2,5 cm, 3,0 cm, and 3,5 cm.

PLATE I

PLATE II

PLATE III

Grades of knock knee in girls of 12 years.

Left to right - Straight legs, slight, 1,5 cm,
2,5 cm, 3,0 cm, 4,5 cm, and 5,0 cm.

PLATE IV

Grades of knock knee in boys of 12 years.

Left to right - Straight legs, slight, 1,5 cm,
2,5 cm, 3,0 cm, and 6,5 cm.

PLATE III

PLATE IV

CHAPTER 3

RESULTS

3.1. PRELIMINARY SURVEY

The prevalences of bowing of legs and knock knee found in the preliminary investigations are set out in Table V.

Table V : Preliminary survey - visual assessment of bowing and knock knee in rural Bantu schoolchildren; prevalence according to sex, age and total school groups

Age	Sex	No.of Subj.	Bowing No.	Bowing %	Knock knee No.	Knock knee %
7 years	Male	159	19	12,0	12	7,6
	Female	167	21	12,6	6	3,6
12 years	Male	128	29	24,6	2	1,7
	Female	146	23	15,8	4	2,7
16 - 17 years	Male	133	28	21,2	1	0,7
	Female	255	41	16,1	10	3,9
Total group	Male	1387	270	19,5	40	2,8
6 - 20 years	Female	1732	262	15,2	57	3,3

3.1.1. Summary of Results

1) The visual assessment of bowing in rural Bantu schoolchildren proved to be a fairly reliable screening method and gave results

similar to those found in later more detailed prevalence studies.

2) However, due to rapidity of screening and consequent lack of attention to correct positioning, the prevalence of knock knee was found to be very low. This served to emphasise the need for extreme care in positioning, and was invaluable in subsequent studies.

3) The greatest value of the preliminary survey was that it drew attention to the problem of leg abnormalities in rural children.

3.2. DIETARY STUDIES

In Table VI estimated mean daily vitamin D intakes are given, also ranges, for 16 - 17 year children. Values at this age can be regarded as equivalent to adult values.

Table VI : Estimated mean daily intakes of vitamin D at 16 - 17 years

Group	No.of subj.	Vitamin D I.U./day	Range I.U./day
Bantu	50	202	102-378
Coloured	44	210	108-369
Indian	56	159	46-344
White	38	174	79-351

Table VII sets out the advice, on vitamin D prophylaxis for infants received by mothers, and by whom it was given, such as the taking of vitamin D-containing syrups, etc.

Table VII : Source of advice on vitamin D prophylaxis for infants

Group	No.of subj.	Advice received from Doctor %	Clinic sister %	Chemist %
Bantu	933	27,9	27,2	11,1
Coloured	1860	17,1	46,6	4,1
Indian	1856	20,7	31,2	3,8
White	2735	20,9	28,2	2,9

The prevalences of vitamin D prophylactic measures, as practised by the various ethnic groups of mothers, are given in Table VIII.

Table VIII : Percentage of children receiving vitamin-D containing syrups

Group	No.of subj. %	Vitamin D-containing syrups %
Bantu	933	55,8
Coloured	1860	62,0
Indian	1856	52,9
White	2735	47,2

3.2.1. Summary of Results

1) Dietary intake of vitamin D per diem was low. As these values are equivalent to adult values younger children would be proportionately lower as their food intake was less.

Note: The following information, from Tables VII and VIII, was obtained from mothers, using a questionnaire form. It must thus be regarded with a certain amount of reservation, which applies to, and is inherent in this form of data collection. However, the large representative groups, taking part in the survey, were drawn from various schools in different

areas, including upper, middle and lower socio-economic groups, which partly does away with these reservations.

2) Approximately one half of mothers in all groups received no advice on vitamin D prophylaxis.

3) Advice given by medical practitioners was only to some 20% of mothers, and from clinic sisters to a further 30%.

4) Coloured mothers receiving advice (some 50%) appeared to favour clinic attendance rather than the medical profession. In the other groups about equal numbers (25 - 30%) were advised by either doctor or clinic sister.

5) Advice from chemists was sought by only a negligible proportion of mothers, with Bantu mothers using this facility rather more than mothers from the other groups.

3.3. PREVALENCE OF SKELETAL ABNORMALITIES : ANTHROPOMETRIC AND CLINICAL OBSERVATIONS

3.3.1. Abnormalities of the Lower Limbs

Photographs of bowing and knock knee

In order to visualize the leg abnormalities described, photographs of 12 year old pupils, showing these abnormalities, in the four ethnic groups, are included.

Plates V to XII show pupils with bowing of approximately 1,5 cm, 2,5 cm, and 3,5+ cm and the same for knock knee. Children with bowing and knock knee are arranged to the left and right of the centre child, who has straight legs, in each ethnic and sex groups. Plates V and VI show Bantu; VII and VIII Coloured; IX and X Indian; and XI and XII White girls and boys, respectively. (Actual gap measurements are given in the legends to each photograph).

Plates V to XII appear between pages 87 and 88.

Photographs of obesity and knock knee

Plates XIII and XIV show obese pupils with knock knee. It is of interest to note that in the White group of boys and girls, knock knee was not always associated with obesity. In Plate XIV this was very noticeable in the boy on the extreme left, but less so in the girl on the extreme right, although both had a marked degree of knock knee.

Plates XIII and XIV appear between pages 101 and 102.

PLATE V

PLATE VI

PLATE VII

Bowing and knock knee in Coloured girls of 12 years.

Left to right - Bowing : 3,8 cm, 2,5 cm, 1,5 cm, straight legs,

Knock knee : 1,5 cm, 2,5 cm, 11,2 cm.

PLATE VIII

Bowing and knock knee in Coloured boys of 12 years.

Left to right - Bowing : 3,8 cm, 2,5 cm, 1,5 cm, straight legs,

Knock knee : 1,5 cm, 2,5 cm, 5,6 cm.

PLATE VII

PLATE VIII

PLATE IX

Bowing and knock knee in Indian girls of 12 years.

Left to right - Bowing : 3,6 cm, 2,5 cm, 1,5 cm, straight legs,

Knock knee : 1,5 cm, 2,5 cm, 5,7 cm.

PLATE X

Bowing and knock knee in Indian boys of 12 years.

Left to right - Bowing : 3,0 cm, 2,5 cm, 1,5 cm, straight legs,

Knock knee : 1,5 cm, 2,5 cm, 6,0 cm.

PLATE IX

PLATE X

PLATE XI

Bowing and knock knee in White girls of 12 years.

Left to right - Bowing : 3,5 cm, 2,5 cm, 1,5 cm, straight legs,

Knock knee : 1,5 cm, 2,5 cm, 4,5 cm.

PLATE XII

Bowing and knock knee in White boys of 12 years.

Left to right - Bowing : 3,5 cm, 2,5 cm, 1,5 cm, straight legs,

Knock knee : 1,5 cm, 3,5 cm, 6,5 cm.

PLATE XI

PLATE XII

Prevalences of bowing and knock knee

It must be borne in mind that variations of bowing and knock knee within sub-groups could be as large as between ethnic groups, due principally to difficulty in positioning of subjects.

The following tables (Tables IX - XI) show the prevalences of various grades of bowing and knock knee, also straight legs, that occurred in inter ethnic groups of schoolchildren at 7, 12 and 16 - 17 years, respectively. Table XII shows the above prevalences at each age for schoolchildren per se (ethnic groups combined).

Note: Where tables occupy a whole page they appear in sequence after their respective sections. Table and page numbers are indicated.

Table IX appears on page 92.
Table X appears on page 93.
Table XI appears on page 94.
Table XII appears on page 95.

Table XIII gives the prevalences, in schoolchildren, of bowing and knock knee of 2,5 cm, or more, by age, sex and ethnic group.

Table XIII appears on page 96.

The bearing of higher mean minimum temperatures experienced in the lowveld, compared with those of the highveld, on the prevalences of bowing and knock knee in Bantu and White children of 7 and 12 years, are set out in Table XIV.

Table XIV appears on page 97.

The means and standard deviations of 'gap' measurements, in different ethnic groups of schoolchildren, are given in Table XV.

Table XV appears on page 98.

3.3.2. Influencing Factors

Mean anthropometric measurements

Tables (XVI - XIX) show mean values for the various anthropometric measurements in schoolchildren according to ethnic group, age and sex, as follows:- Table XVI - height (cm) and body mass (kg); Table XVII - wrist and head widths, also circumferences in cm; Table XVIII - cristal height, mid-thigh circumference, and knee widths in cm; and Table XIX - triceps skinfold (mm) and armgirth (cm).

Table XVI appears on page 99.

Table XVII appears on page 100.

Table XVIII appears on page 101.

Table XIX appears on page 102.

Note: Plates XII and XIV appear between pages

The role of obesity on knock knee

Table XX shows the prevalence of obesity, 20% above expected mass for age *, and overweight, 10% above *, in interracial groups of schoolchildren.

(* The Iowa 50th percentile was used).

Table XX appears on page 103.

In Tables XXI and XXII the percentages of obese and overweight children with associated bowing or knock knee of 2,5 cm or more, respectively, or straight legs are given according to total number in each age, sex and ethnic group.

Table XXI appears on page 104.

Table XXII appears on page 105.

The proportion of obese children, with associated leg deformities of 2,5 cm or more, or straight legs, is expressed as a percentage of the total obese group in each age, sex and ethnic group in Table XXIII.

Table XXIII appears on page 106.

Table XXIV shows the proportion of obese children, with associated leg abnormalities of 2,5 cm or more, or straight legs, expressed as a percentage of those children with bowing or knock knee of 2,5 cm or more, or straight legs in each age, sex and ethnic group.

Table XXIV appears on page 107.

Comparative mean height, mass, body mass index, percentage obesity, mid-thigh circumference, and cristal height of urban Bantu pupils of 16 - 17 years with severe bowing, knock knee or straight legs, together with their standard deviations, are set out in Table XXV.

Table XXV appears on page 108.

Growth curves : the adolescent growth spurt

Growth curves for the various interracial groups of schoolchildren,

males and females, are depicted in Figures 1 - 4, showing height (Figures 1 and 2) and mass (Figures 3 and 4).

Figure 1 appears on page 109.

Figure 2 appears on page 110.

Figure 3 appears on page 111.

Figure 4 appears on page 112.

Table IX: Prevalences of bowing and knock knee at 7 years

Group		No.of Subj.		Straight legs	Bowing 1,5-2,4cm	Bowing 2,5+cm	Knock knee 1,5-2,4cm	Knock knee 2,5+cm
Bantu Rural	M	253	No.	137	43	11	25	36
			%	54,2	17,0	4,5	10,1	14,2
	F	260	No.	134	48	28	24	33
			%	51,5	18,5	10,5	9,4	12,7
Urban	M	74	No.	28	4	5	13	22
			%	37,9	5,4	6,8	17,6	29,7
	F	78	No.	22	13	7	9	25
			%	28,2	16,7	8,9	11,8	32,0
Coloured Urban	M	86	No.	36	6	7	14	22
			%	41,8	7,0	8,2	16,3	26,2
	F	83	No.	39	10	5	7	21
			%	47,0	12,1	6,0	8,4	25,9
Indian Urban	M	111	No.	50	10	11	22	19
			%	45,0	9,0	9,9	19,9	17,1
	F	134	No.	47	12	11	25	38
			%	35,1	8,9	8,2	18,7	28,3
White Rural	M	36	No.	11	8	4	4	9
			%	30,5	22,2	11,1	11,1	25,0
	F	38	No.	9	4	3	8	14
			%	23,7	10,5	7,9	21,1	36,7
Urban	M	194	No.	91	15	11	30	40
			%	47,0	8,1	5,7	16,3	20,9
	F	159	No.	102	20	11	26	39
			%	64,0	12,7	6,6	16,7	24,7

Table X: Prevalences of bowing and knock knee at 12 years

Group		No.of Subj.		Straight legs	Bowing 1,5-2,4cm	Bowing 2,5+cm	Knock knee 1,5-2,4cm	Knock knee 2,5+cm
Bantu Rural	M	251	No.	112	39	52	12	36
			%	44,6	15,7	21,0	4,9	14,5
	F	256	No.	109	45	57	12	33
			%	42,4	17,5	22,4	4,8	13,1
Urban	M	78	No.	25	13	7	8	25
			%	32,1	16,7	9,0	10,1	32,0
	F	70	No.	15	9	7	9	30
			%	21,4	12,8	10,0	12,8	42,9
Coloured Urban	M	108	No.	46	19	9	12	21
			%	42,5	17,6	8,3	11,1	19,9
	F	77	No.	21	10	13	10	22
			%	27,3	13,6	17,5	13,0	28,5
Indian Urban	M	131	No.	67	21	8	12	23
			%	51,0	16,1	6,1	9,1	17,4
	F	115	No.	42	18	10	19	24
			%	36,5	15,7	8,7	16,5	20,8
White Rural	M	21	No.	4	2	5	4	6
			%	19,0	9,6	23,8	19,0	28,5
	F	28	No.	8	3	10	2	4
			%	28,5	10,7	35,7	7,1	35,7
Urban	M	230	No.	80	27	34	21	49
			%	34,6	11,7	15,0	9,3	21,3
	F	230	No.	77	27	40	19	53
			%	33,4	11,9	17,4	8,2	23,0

Table XI: Prevalences of bowing and knock knee at 16-17 years

Group		No.of Subj.		Straight legs	Bowing 1,5-2,4cm	Bowing 2,5+cm	Knock knee 1,5-2,4cm	Knock knee 2,5+cm
Bantu Rural	M	291	No.	108	51	72	12	48
			%	37,1	17,5	24,7	4,1	16,6
	F	315	No.	100	50	71	18	82
			%	31,7	15,0	22,5	5,8	26,2
Urban	M	284	No.	88	54	75	17	48
			%	31,2	19,1	26,4	6,0	16,9
	F	413	No.	116	58	55	36	123
			%	28,2	14,1	13,3	8,7	29,9
Coloured Urban	M	198	No.	70	40	40	12	13
			%	35,3	20,2	20,2	6,1	17,1
	F	128	No.	43	25	13	9	37
			%	33,6	19,5	10,0	7,0	28,8
Indian Urban	M	304	No.	114	59	66	18	42
			%	37,5	19,5	19,4	5,9	12,3
	F	147	No.	56	19	11	17	41
			%	38,1	12,9	7,5	11,5	27,8
White* Urban	M	223	No.	81	48	51	15	31
			%	36,2	21,5	22,9	6,7	5,3
	F	169	No.	70	27	23	9	38
			%	41,4	16,3	13,6	14,6	22,7

* No rural white pupils of 16-17 years were available for study - see * table 11 on page 68.

Table XII: Prevalences of bowing and knock knee in combined groups of schoolchildren according to sex, age and degree

Age	Sex	No.of Subj.		Straight legs	Bowing 1,2-2,4cm	Bowing 2,5+cm	Knock knee 1,2-2,4cm	Knock knee 2,5+cm
7 years	M	754	No.	353	86	50	109	149
			%	46,8	11,5	6,7	14,5	19,8
	F	752	No.	353	107	65	100	171
			%	46,9	14,3	8,6	13,3	22,7
	T	1506	No.	706	194	115	209	320
			%	46,9	12,9	7,7	13,9	21,2
12 years	M	819	No.	334	121	116	70	161
			%	40,8	14,8	14,2	8,5	19,7
	F	776	No.	272	111	138	71	166
			%	35,1	14,3	17,8	9,2	21,5
	T	1595	No.	606	232	254	141	327
			%	38,0	14,6	15,9	8,9	20,5
16-17 years	M	1300	No.	461	252	304	74	183
			%	35,5	19,4	23,4	5,7	14,1
	F	1172	No.	385	179	173	89	322
			%	32,8	15,3	14,8	7,6	27,5
	T	2472	No.	846	431	477	163	505
			%	34,2	17,5	19,3	6,6	20,4

Table XIII: Prevalences of bowing and knock knee of 2,5cm or more, according to sex and age

Leg abnormality	Group	7 years Percentage Male	7 years Percentage Female	12 years Percentage Male	12 years Percentage Female	16-17 years Percentage Male	16-17 years Percentage Female
Bowing	Bantu						
	Rural	4,5	10,5	21,0	22,4	24,7	22,5
	Urban	6,8	8,9	9,0	10,0	26,4	13,3
	Coloured						
	Urban	8,2	6,0	8,3	17,5	20,2	10,0
	Indian						
	Urban	9,9	8,2	6,1	8,7	19,4	7,5
	White						
	Rural*	11,1	7,9	23,8	35,7	0,0	0,0
	Urban	5,7	6,6	15,0	17,4	22,9	13,6
Knock knee	Bantu						
	Rural	14,2	12,7	14,5	13,1	16,6	26,2
	Urban	29,7	32,0	32,0	42,9	16,9	29,9
	Coloured						
	Urban	26,2	25,9	19,9	28,5	17,1	28,8
	Indian						
	Urban	17,1	28,3	17,4	20,8	12,3	27,8
	White						
	Rural*	25,0	36,7	28,5	14,3	0,0	0,0
	Urban	20,9	24,7	21,3	23,0	14,6	22,7

* Percentage prevalences in rural white groups are influenced by small numbers and for this reason must be viewed with caution.

Table XIV: Prevalences of bowing and knock knee of 2,5cm or more, in Bantu and White Highveld and Lowveld dwellers of 7 and 12 years

Group	Sex	Bowing 2,5+cm 7 years No.	%	Bowing 2,5+cm 12 years No.	%	Knock knee 2,5+cm 7 years No.	%	Knock knee 2,5+cm 12 years No.	%
Bantu Highveld*	M	134	4,4	159	7,6	134	25,4	159	29,6
	F	151	7,9	170	12,9	151	20,6	170	26,5
Bantu Lowveld*	M	134	4,1	125	31,2	134	7,5	125	13,6
	F	125	9,6	112	24,2	125	16,2	112	17,4
White Highveld*	M	194	5,7	230	15,0	194	20,9	230	21,3
	F	159	6,6	230	17,4	159	24,7	230	23,0
White Lowveld*	M	36	11,1	21	23,8	36	25,0	21	28,5
	F	38	7,9	28	35,7	38	36,7	38	14,3

* Highveld - mean minimum temperature 13,2°C

* Lowveld - mean minimum temperature 18,1°C

Table XV: Means and standard deviations of 'gap' measurements of 2,5cm or more

Group	No.of subj.	Bowing Mean	Bowing S.D.	Knock knee Mean	Knock knee S.D.
Bantu					
Rural	572	3,43	0,93	3,89	1,19
Urban	298	3,49	0,86	4,89	2,08
Coloured					
Urban	200	3,32	0,66	4,47	1,82
Indian					
Urban	250	3,32	0,64	4,62	2,16
White					
Rural	123	3,78	1,02	4,43	1,70
Urban	284	3,61	0,95	4,13	1,75

Table XVI: Mean height and body mass* at 7, 12 and 16-17 years

Age	Group	Male No.of Subj.	Male Height cm	Male Mass kg	Female No.of Subj.	Female Height cm	Female Mass kg
7 years	Bantu						
	Rural	201	116,7	19,9	211	116,2	19,6
	Urban	72	117,1	21,6	73	116,5	21,3
	Coloured						
	Urban	86	118,2	21,0	83	119,0	21,1
	Indian						
	Urban	110	120,7	20,9	133	118,8	20,6
	White						
	Rural	36	122,3	23,5	37	122,5	23,1
	Urban	170	126,1	24,3	151	124,5	23,1
12 years	Bantu						
	Rural	200	138,6	30,3	209	139,6	32,4
	Urban	60	139,9	33,8	52	142,2	35,5
	Coloured						
	Urban	108	143,0	33,8	77	147,7	38,8
	Indian	85	143,5	34,0	83	147,4	36,5
	White						
	Rural	21	149,6	42,6	28	153,6	43,4
	Urban	171	149,1	40,0	176	152,7	43,8
16-17 years	Bantu						
	Rural	180	160,2	50,3	200	155,0	50,2
	Urban	152	162,7	51,3	298	156,1	54,7
	Coloured						
	Urban	109	168,0	57,3	74	156,9	52,3
	Indian						
	Urban	256	166,7	50,8	119	154,6	46,7
	White						
	Urban	205	173,1	60,8	153	162,8	57,0

* Mass - In the International Unit Metrication System weight has been designated as mass.

Table XVII: Mean wrist and head widths and circumferences in cm at 7, 12 and 16-17 years

Age	Group	Male					Female				
		No.of subj.	Wrist width	Wrist circum-ference	Head width	Head circum-ference	No.of subj.	Wrist width	Wrist circum-ference	Head width	Head circum-ference
7 years	Bantu Rural	125	4,0	11,9	13,3	52,6	139	3,9	11,7	13,0	50,7
	Bantu Urban	72	4,1	12,4	13,2	52,4	73	4,0	12,1	12,9	52,1
	Coloured Urban	83	4,1	12,8	13,5	53,6	82	3,9	12,3	12,6	51,4
	Indian Urban	72	4,1	12,3	13,6	51,2	80	4,0	12,2	13,2	50,4
	White Rural	25	4,2	12,9	13,6	52,1	18	4,0	12,4	13,4	51,0
	White Urban	70	4,2	13,2	13,8	53,1	61	4,1	12,8	13,3	51,8
12 years	Bantu Rural	127	4,6	13,5	13,4	53,7	123	4,5	13,6	13,3	52,9
	Bantu Urban	60	4,7	14,0	13,3	54,5	52	4,6	14,1	13,1	54,2
	Coloured Urban	108	4,7	14,2	13,4	53,5	77	4,7	14,4	13,3	54,3
	Indian Urban	49	4,7	14,0	13,3	53,0	49	4,6	14,1	13,3	52,9
	White Rural	21	4,8	15,0	14,0	53,9	28	4,8	14,8	13,8	53,5
	White Urban	66	4,8	14,8	14,0	54,8	87	4,8	15,0	13,8	54,5
16-17 years	Bantu Rural	71	5,0	15,4	13,8	55,0	45	4,7	15,3	14,1	55,5
	Bantu Urban	51	5,4	16,4	14,3	61,2	62	4,9	15,7	13,8	56,2
	Coloured Urban	49	5,5	16,4	14,0	57,0	49	4,8	14,9	13,6	55,9
	Indian Urban	45	5,3	16,1	14,1	55,9	44	4,7	14,8	13,3	54,1
	White Urban	96	5,6	17,0	14,6	57,8	52	5,0	15,1	13,5	55,0

Table XVIII: Mean cristal height, mid-thigh circumference, and widths of left ankle, calf and knee at 7, 12 and 16 - 17 years

(all measurements given in cm)

Age	Group	Male						Female					
		No.of subj.	Cristal height	Mid-thigh circum.	Left Leg ankle	Left Leg calf	Left Leg knee	No.of subj.	Cristal height	Mid-thigh circum.	Left Leg ankle	Left Leg calf	Left Leg knee
7 years	Bantu Rural	145	69,1 (24)	30,0 (27)	5,6	6,2	7,6	159	67,4 (31)	30,8 (44)	5,3	6,1	7,3
	Urban	72	70,5 (50)	33,4	5,5	6,5	7,6	73	69,2 (49)	34,3	5,3	6,6	7,2
	Coloured Urban	83	75,1 (42)	34,3	5,6	6,9	8,0	83	71,3 (43)	34,2	5,3	6,6	7,5
	Indian Urban	72	71,4 (51)	32,0	5,4	6,2	7,5	80	71,8 (48)	33,8	5,2	6,3	7,3
	White Rural	36		33,1 (25)	5,8	6,9	8,0	37		34,7 (23)	5,5	7,0	7,8
	Urban	70		35,1	5,7	7,0	8,1	61		36,2	5,5	7,1	7,8
12 years	Bantu Rural	147	87,1 (28)	36,1 (44)	6,4	7,3	8,7	145	87,7 (30)	37,6 (46)	6,0	7,4	8,6
	Urban	60	89,9 (31)	39,5	6,3	7,8	9,0	52	90,3 (31)	42,3	6,1	8,1	9,1
	Coloured Urban	108	88,7 (43)	40,4	6,4	8,0	9,0	77	92,1 (23)	44,2	6,1	8,4	9,1
	Indian Urban	49	87,9 (25)	39,3	6,2	7,6	8,9	49	90,0 (29)	41,3	5,9	8,0	8,8
	White Rural	21		42,7	6,7	8,7	9,7	28		44,6	6,3	8,7	9,4
	Urban	66		41,8	6,6	8,3	9,6	87		46,6	6,3	8,9	9,5
16-17 years	Bantu Rural	88		43,9	6,7	8,6	9,4	59		48,8	6,1	8,9	9,8
	Urban	51	105,0 (39)	46,2	7,1	9,5	10,0	62	99,5 (48)	54,5	6,3	9,5	10,6
	Coloured Urban	49	105,8	48,4	6,9	9,6	10,0	49	96,5	49,1	6,2	9,3	10,0
	Indian Urban	45	105,4	44,5	6,8	9,1	9,5	44	97,8	46,8	6,1	8,7	9,4
	White Urban	96	110,0 (55)	50,0	7,1	10,3	10,4	52	101,1	50,6	6,4	10,1	10,4

Note: Where number of subjects differ they appear in brackets

Table XIX : Mean triceps skinfold and upper arm-girth at 7, 12 and 16 - 17 years

Age	Group	Male			Female		
		No.of subj.	Triceps skinfold mm	Arm-girth cm	No.of subj.	Triceps skinfold mm	Arm-girth cm
7 years	Bantu						
	Rural	42	5,7	15,7	54	6,0	15,3
	Urban	72	6,9	16,8	73	8,3	17,1
	Coloured						
	Urban	83	7,8	17,6	83	9,5	17,0
	Indian						
	Urban	72	7,4	16,4	80	9,8	17,1
	White						
	Rural	35	7,7	18,3	37	10,7	19,2
	Urban	70	9,0	18,3	61	11,1	18,4
12 years	Bantu						
	Rural	52	5,1	18,1	60	6,9	18,6
	Urban	60	7,7	19,4	52	10,3	20,2
	Coloured						
	Urban	108	7,8	19,9	77	9,8	20,9
	Indian						
	Urban	49	9,4	19,9	49	11,1	19,9
	White						
	Rural	21	10,4	23,7	28	11,2	21,9
	Urban	66	10,5	21,5	87	12,1	22,2
16-17 years	Bantu						
	Rural	88	6,1	22,3	59	13,2	23,5
	Urban	51	7,5	23,8	62	16,0	25,5
	Coloured						
	Urban	49	6,1	24,6	49	12,2	23,9
	Indian						
	Urban	45	6,5	24,1	44	12,2	23,0
	White						
	Urban	96	7,7	26,2	52	13,9	24,6

PLATE XIII

Obesity and knock knee in Coloured pupils of 12 years.

PLATE XIV

Obesity and knock knee in White pupils of 12 years.

Note - The boy on the extreme left, and the girl on the extreme right, are not obese but have knock knee.

PLATE XIII

PLATE XIV

Table XX: Prevalences of obesity and overweight according to age and sex

Age	Group	Area	Sex	No.of subj.	Percentage Obesity*	Overweight+
7 years	Bantu	Rural	M	200	0,0	0,0
			F	260	0,0	0,0
		Urban	M	74	2,7	2,7
			F	78	1,3	1,3
	Coloured	Urban	M	86	3,5	0,0
			F	83	2,4	2,4
	Indian	Urban	M	111	1,8	0,0
			F	134	5,2	4,5
	White	Rural	M	36	11,1	16,6
			F	38	7,9	7,9
		Urban	M	194	7,2	15,5
			F	159	8,7	3,1
12 years	Bantu	Rural	M	193	0,5	0,5
			F	202	0,0	1,9
		Urban	M	78	0,0	2,5
			F	70	10,0	2,8
	Coloured	Urban	M	108	4,6	3,7
			F	77	7,8	13,0
	Indian	Urban	M	131	6,1	1,5
			F	115	5,2	7,8
	White	Rural	M	21	28,5	9,5
			F	28	25,0	14,3
		Urban	M	230	15,2	11,3
			F	230	17,8	13,9
16-17 years	Bantu	Rural	M	246	0,8	0,4
			F	281	10,3	8,2
		Urban	M	263	0,3	1,1
			F	392	12,5	12,0
	Coloured	Urban	M	198	2,5	5,5
			F	128	9,4	10,1
	Indian	Urban	M	304	2,0	2,3
			F	147	2,0	2,7
	White	Urban	M	223	14,4	20,2
			F	169	21,9	21,4

* 20% above Iowa 50th percentile

\+ 10% above Iowa 50th percentile

Table XXI: Obese children with bowing or knock knee of 2,5 cm or more, or straight legs, expressed as percentages of total number in each group

Age	Group	Male				Female			
		Total no.in group	Bowing %	Straight legs %	Knock knee %	Total no.in group	Bowing %	Straight legs %	knock knee %
7 years	Bantu	274	0,0	0,0	0,3	338	0,0	0,0	0,3
	Coloured	86	0,0	0,0	3,5	83	0,0	0,0	2,4
	Indian	111	0,0	0,0	0,0	134	0,0	0,0	3,7
	White	230	0,8	3,0	2,6	197	0,0	2,5	4,6
12 years	Bantu	271	0,4	0,0	0,4	272	0,4	0,0	1,1
	Coloured	108	0,9	0,0	3,7	77	2,6	0,0	2,6
	Indian	131	0,0	0,7	5,3	115	0,0	0,8	4,3
	White	251	1,2	3,2	9,9	258	2,7	3,8	9,3
16-17 years	Bantu	592	0,2	0,0	0,2	707	0,3	3,8	7,3
	Coloured	198	0,5	0,0	3,0	128	0,8	2,3	5,5
	Indian	304	0,3	0,0	1,9	147	0,0	0,0	2,7
	White	223	2,7	5,4	3,6	169	2,9	3,5	12,5

Table XXII: Overweight children with bowing or knock knee of 2,5 cm or more, or straight legs, expressed as percentages of total number in each age group

Age	Group	Male % Total no. in group	Bowing	Straight legs	Knock knee	Female % Total no. in group	Bowing	Straight legs	Knock knee
7 years	Bantu	274	0,0	0,0	0,7	338	0,0	0,3	0,3
	Coloured	86	0,0	0,0	0,0	83	0,0	1,2	1,2
	Indian	111	0,0	0,0	0,0	134	0,7	0,0	3,0
	White	230	0,8	4,8	4,8	197	0,0	1,5	1,5
12 years	Bantu	271	0,0	1,1	0,0	272	0,0	0,7	0,4
	Coloured	108	0,0	0,9	2,8	77	2,6	3,9	7,8
	Indian	131	0,0	0,7	0,7	115	0,0	2,6	4,3
	White	251	1,2	3,5	2,8	258	1,9	5,8	3,1
16-17 years	Bantu	592	0,3	0,3	0,3	707	1,1	3,8	5,1
	Coloured	198	0,5	1,0	3,5	128	0,8	5,5	3,9
	Indian	304	0,3	1,3	0,6	147	0,0	1,3	1,3
	White	223	4,0	4,5	3,1	169	1,6	2,3	5,9

Table XXIII: Obese children with bowing or knock knee of 2,5 cm or more, straight legs, expressed as percentages of total number in obese groups

Age	Group	Male % No.of obese subj.	Bowing	Straight legs	Knock knee	Female % No.of Obese subj.	Bowing	Straight legs	Knock knee
7 years	Bantu	2	0,0	0,0	50,0	1	0,0	0,0	100,
	Coloured	3	0,0	0,0	100,0	2	0,0	50,0	100,
	Indian	2	0,0	0,0	100,0	7	0,0	0,0	71,
	White	18	11,1	38,9	33,3	17	0,0	29,4	52,
12 years	Bantu	1	100,0	0,0	100,0	7	14,3	0,0	42,
	Coloured	5	20,0	0,0	80,0	6	33,3	0,0	33,
	Indian	8	0,0	12,5	87,5	6	0,0	16,7	83,
	White	41	7,3	19,5	61,0	48	14,5	20,8	50,
16-17 years	Bantu	3	33,3	0,0	33,3	78	2,5	35,9	66,
	Coloured	5	20,0	0,0	100,0	12	8,3	25,0	58,
	Indian	6	16,7	0,0	100,0	3	0,0	0,0	100,
	White	32	18,7	37,5	25,0	37	13,5	16,2	56,

Table XXIV: Obese children with bowing or knock knee of 2,5 cm or more, or straight legs, expressed as percentages of total with these abnormalities, also straight legs

Age	Group	Male % Bowing	Male % Straight legs	Male % Knock knee	Female % Bowing	Female % Straight legs	Female % Knock knee
7 years	Bantu	0,0	0,0	1,7	0,0	0,0	1,7
	Coloured	0,0	0,0	13,4	0,0	0,0	9,3
	Indian	0,0	0,0	10,6	0,0	0,0	13,2
	White	6,7	6,7	12,1	0,0	4,5	16,9
12 years	Bantu	1,7	0,0	1,6	1,5	0,0	4,7
	Coloured	11,1	0,0	18,6	14,9	0,0	9,1
	Indian	0,0	1,5	30,4	0,0	2,4	20,7
	White	7,6	9,5	45,5	14,0	11,8	42,1
16-17 years	Bantu	0,7	0,0	1,3	1,6	1,2	25,3
	Coloured	2,5	0,0	46,1	7,7	7,0	18,9
	Indian	1,5	0,0	14,3	0,0	0,0	9,7
	White	11,8	14,8	25,4	21,7	8,6	54,5

Table XXV: Means and standard deviations of height, mass, body mass index (wt/ht^2), mid-thigh circumference and cristal height, also percentage obesity, of urban Bantu pupils of 16-17 years with bowing or knock knee of 2,5 cm or more, or straight legs

Leg abnormality	Sex	No. of subj.		Height cm	Mass kg	Mass / ht^2	Mid-thigh circum. cm	Cristal height cm	Obesity %
Bowing	M	22	Mean	159,8	48,4	19,1	43,9	102,9	0,0
			S.D.	11,1	5,9	3,9	2,2	4,3	
	F	16	Mean	157,1	49,9	20,2	48,6	98,5	0,0
			S.D.	4,9	5,0	1,8	2,7	3,6	
Knock knee	M	21	Mean	163,8	50,8	18,9	44,8	104,8	0,0
			S.D.	4,2	5,2	1,8	2,7	3,2	
	F	19	Mean	152,3	60,4	26,0	54,9	99,0	47,4
			S.D.	10,5	8,7	4,4	6,7	4,6	
Straight legs	M	21	Mean	161,8	49,9	19,0	44,3	102,3	0,0
			S.D.	7,1	5,4	1,3	2,9	5,3	
	F	17	Mean	156,4	48,4	19,7	48,0	97,9	0,0
			S.D.	4,6	5,9	1,7	3,4	4,0	

Figure 1

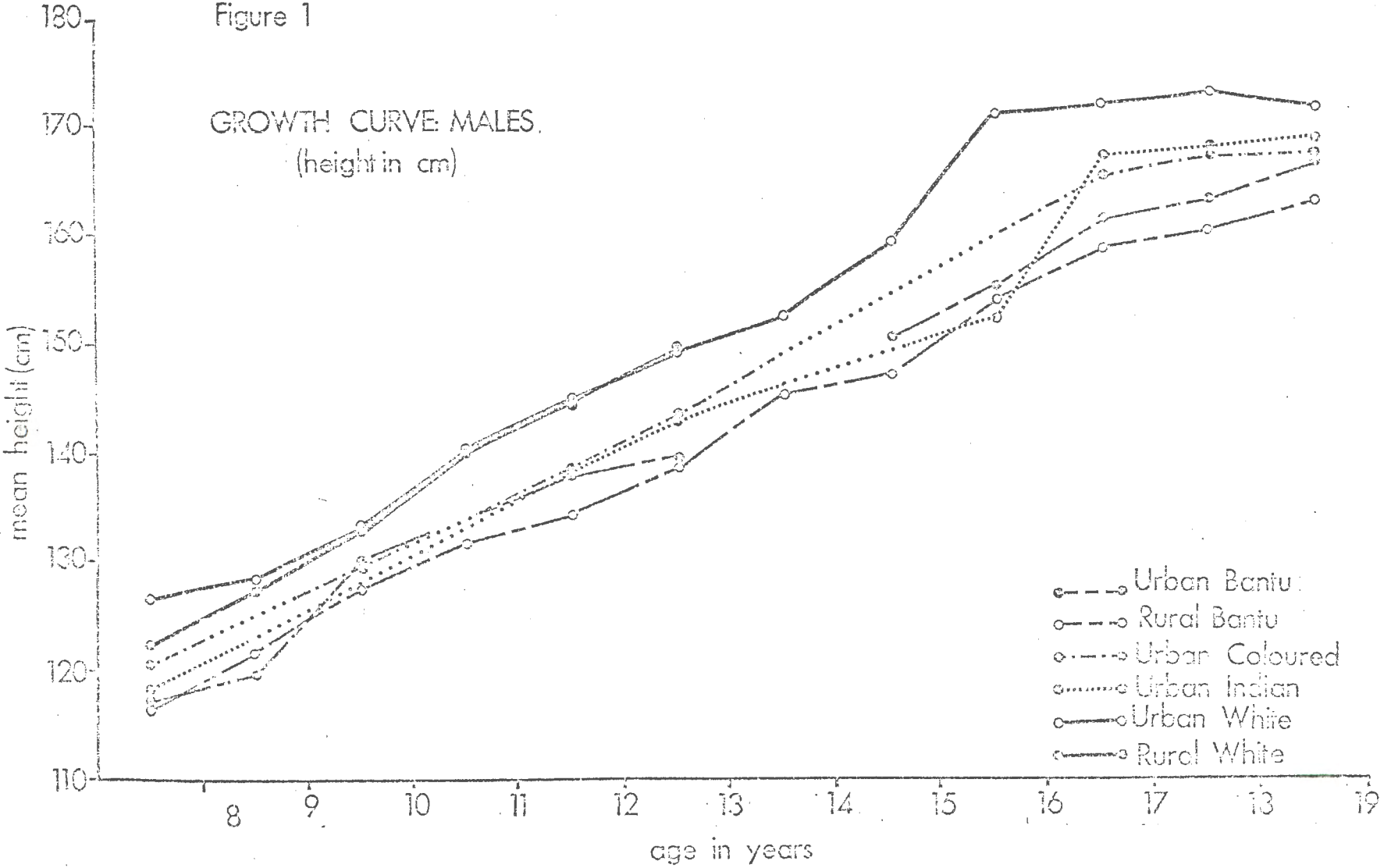

GROWTH CURVE: MALES
(height in cm)
mean height (cm)
180
170
160
150
140
130
120
110
8
9
10
11
12
13
14
15
16
17
18
19
age in years
Urban Bantu
Rural Bantu
Urban Coloured
Urban Indian
Urban White
Rural White

Figure 2

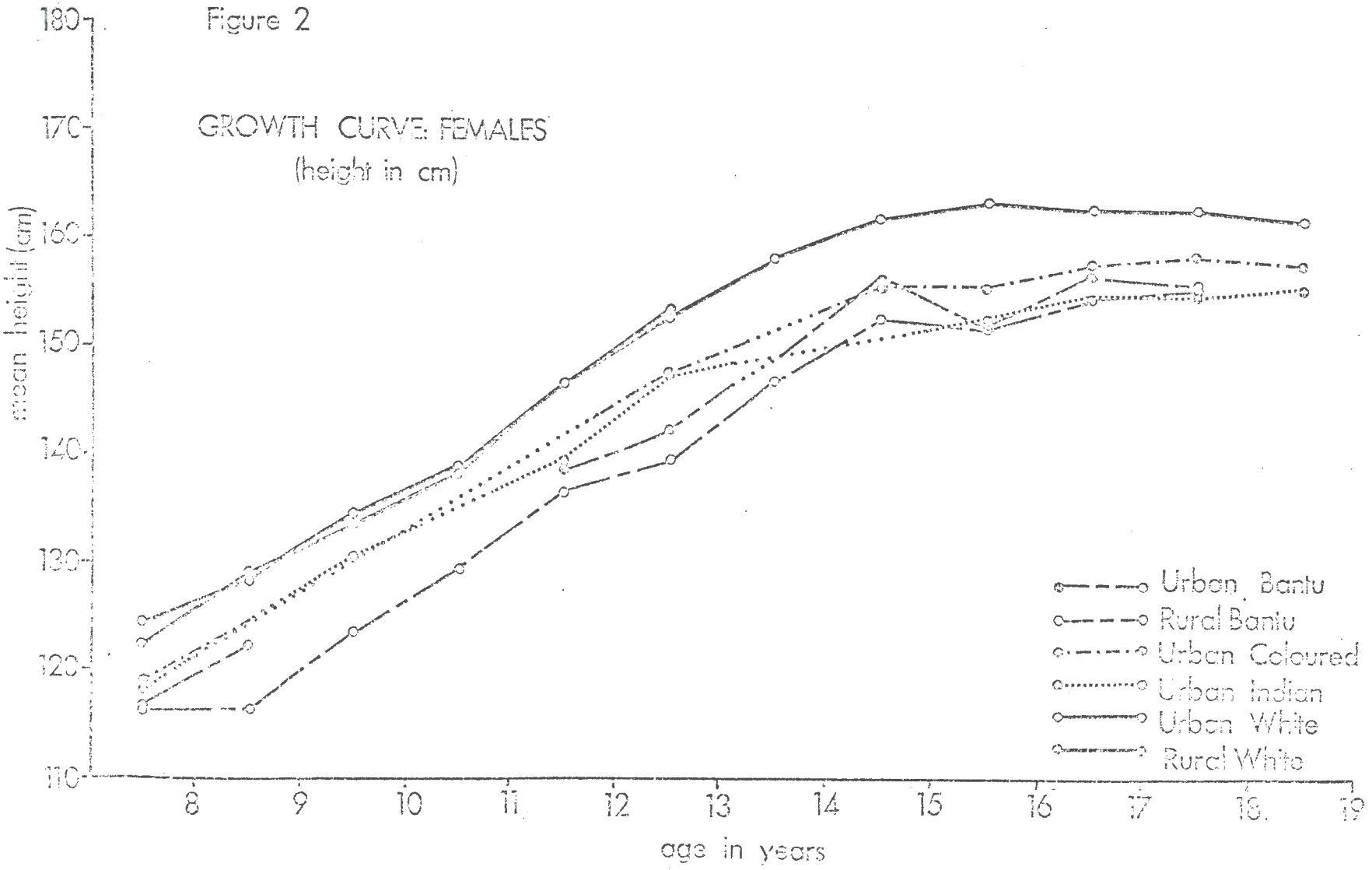

GROWTH CURVE: FEMALES
(height in cm)
mean height (cm)
180
170
160
150
140
130
120
110
8
9
10
11
12
13
14
15
16
17
18
19
age in years
Urban Bantu
Rural Bantu
Urban Coloured
Urban Indian
Urban White
Rural White

Figure 3

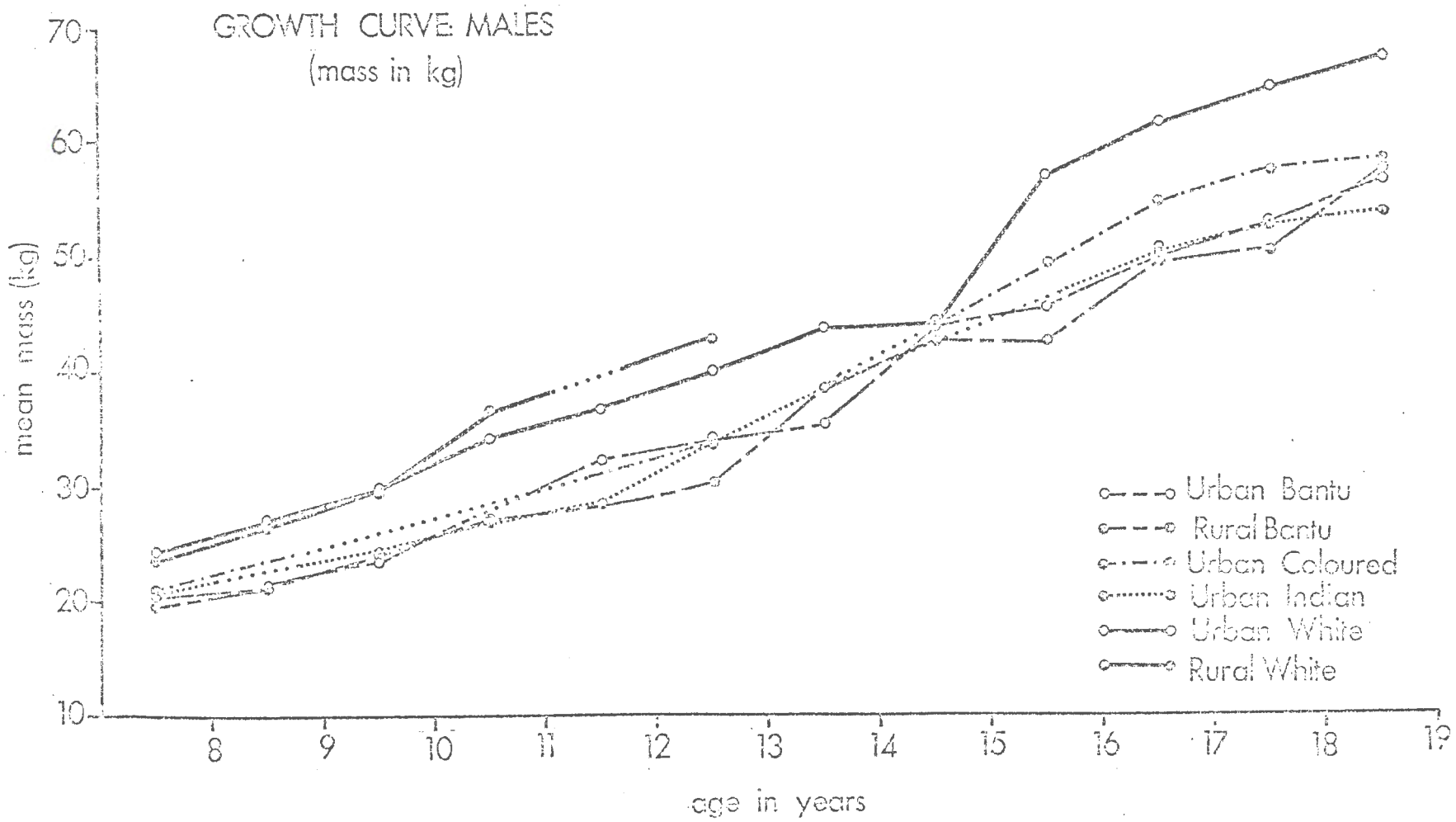
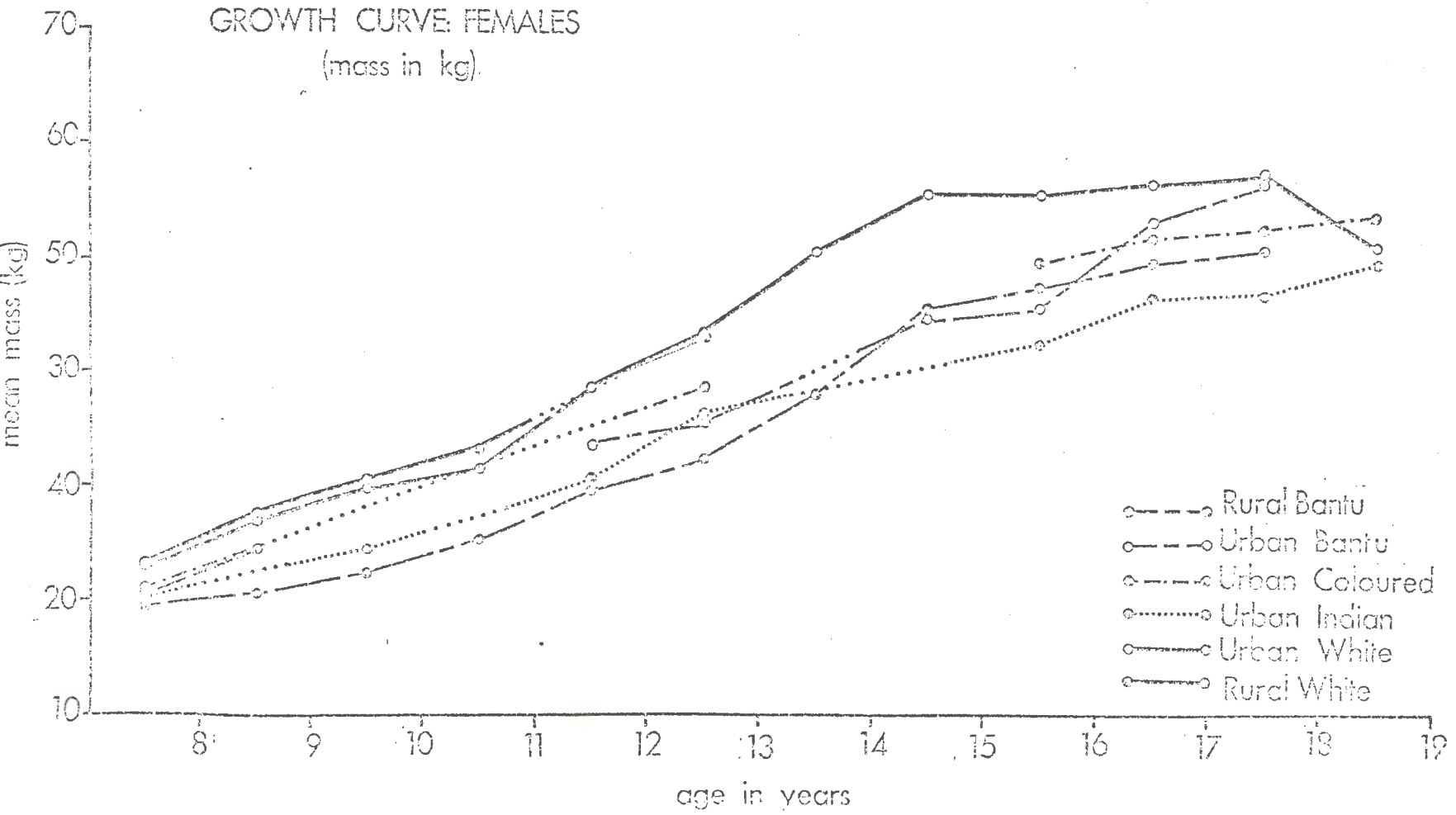

GROWTH CURVE: MALES
(mass in kg)
mean mass (kg)
70
60
50
40
30
20
10
8
9
10
11
12
13
14
15
16
17
18
19
age in years
Urban Bantu
Rural Bantu
Urban Coloured
Urban Indian
Urban White
Rural White

Figure 4


GROWTH CURVE: FEMALES
(mass in kg)
mean mass (kg)
70
60
50
40
30
20
10
8
9
10
11
12
13
14
15
16
17
18
19
age in years
Rural Bantu
Urban Bantu
Urban Coloured
Urban Indian
Urban White
Rural White

3.3.3. Summary of Results

Prevalences of bowing and knock knee

Regarding repeatability, as has previously been stressed, in measurements of 100 consecutive pupils, by two observers, the percentage difference in readings for gaps of 2,5 cm or more at knee was 10,0%, and at ankle, 7,5%. Differences were due almost exclusively in the positioning of the legs prior to measurements.

Bearing this limitation in mind:

1) Leg abnormalities were more common than expected. Less than one half of all schoolchild populations studied had straight legs. Bowing was less common than knock knee.

2) In the case of bowing more children of 7 years had minor degrees (1,5 - 2,4 cm) rather than more marked (2,5 cm or more) degrees of abnormality; proportions, however, became similar at 12; but less had minor degrees at 16 - 17 years (Table XII). Thus minor degrees of bowing appear to progress to more marked degrees during this age range, which included the adolescent growth spurt period.

3) In the case of minor degrees of knock knee, a decreasing prevalence occurred, but the more marked forms remained much the same, namely, about 20% (Table XII).

4) The decrease in prevalence with age was also apparent in the case of straight legs; more children had such at 7, than at 12 or 16 - 17 years (Table XII).

5) There were no clear cut interracial differences in the

prevalences of bowing of 2,5 cm or more. Thus, privilege did not play a role in the case of White children, as no obvious differences could be found between White or other groups. In all groups there was a rise with age, with the exception of Indian girls, who remained much the same (Table XIII).

6) Girls of 12 had, on the whole, greater prevalences of bowing than boys of the same age (Table XIII). Boys begin the growth spurt later than girls, namely, 12 - 15 compared with 10 - 13 years, respectively. This difference may account in part for the sex differences in prevalence.

7) Interracial differences were not obvious in knock knee, except in rural Bantu, who had values slightly lower. A tendency for knock knee to decrease with age was apparent in all groups of boys, except rural Bantu, whose data remained much the same. As a rule girls had a higher prevalence of knock knee than boys, particularly urban Bantu girls (Table XIII). Privilege did not play a role in the case of White children, although there was more obesity in this group, however, knock knee increased markedly in all obese groups.

8) There were almost no differences in prevalences of bowing or knock knee between rural and urban groups, bearing in mind the percentage difference that can occur in constituent subgroups.

9) The higher mean minimum temperature ($5^{o}C$ greater) and thus less clothing worn and more exposure to ultraviolet irradiation, experienced by lowveld dwellers, whether Bantu or White children, did not appear to exert a difference on prevalences of knock knee or bowing at 7 or 16 - 17 years, but at 12 years bowed legs were somewhat

more common. However, numbers of White rural children studied were too small to draw firm conclusions (Table XIV). It was of interest that highveld Bantu had less bowing and more knock knee than lowveld dwellers. The latter may be due to greater body mass of the urban children (included in the highveld group).

10) There were no significant differences ($P<0,05$) in subgroups of children whose gap measurements were 2,5 cm or more, indicating that there were no differences in the severity of either bowing or knock knee between the various ethnic groups (Table XV).

Influencing Factors

Mean Anthropometric Measurements

1) Mean average mass varied among the ethnic groups. At 7 years no sex differences were apparent. Lowest body mass was found in rural Bantu, and highest body mass in Whites. At 12 years, boys were lighter than girls in all groups; rural Bantu were lightest and Whites, heaviest. An exception was Indian girls whose body mass was only as much as that of rural Bantu girls. At 16 - 17 years boys now had a greater body mass than girls, with the exception of Bantu girls, who were about 5 kg heavier than Bantu boys, and almost as heavy as White girls. Rural Bantu were the lightest and Whites the heaviest, with once again the unexplained exception of Indian girls who had the lowest body mass (Table XVI).

2) The Bantu girls were the most interesting group. At 7 years they had much the same mass as Bantu boys; by 12 years their mass was similar to that of Coloured girls, but not as heavy as White girls;

by 16 - 17 years, however, they had almost reached the body mass attained by White girls.

3) Regarding height at 7 and 12 years, Bantu were shortest and Whites tallest. At 16 - 17 years, Indian girls were the shortest; Bantu urban girls as short as their rural counterparts, being some 10 cm shorter than White girls (Table XVI).

4) The salient point of interest is that Bantu girls of 16 - 17 years were much shorter than White girls, although weighing almost as much.

5) Mean wrist widths were remarkably constant in all groups at all ages; wrists of White pupils were only slightly wider than those of other groups. This was also true in regard of wrist girth or circumference. Width of wrist is therefore an unsuitable epidemiological tool in general population groups for use in the detection of wrist thickening associated with rickets (Table XVII).

6) In the different ethnic groups the mean widths of the head (measured across the parietal bones just above the ear) were found to vary little with only a small bias in favour of Whites. Parietal bossing was thus not a feature in any one group (Table XVII) of these older children.

Head circumference was measured at greatest circumference around the occipital protuberance, posteriorly, and frontal bones, anteriorly, immediately above the level of the eyebrows. Bantu and Whites had similar measurements, but Coloured and Indian pupils had smaller heads. This probably is an ethnic difference and not associated with hyperostoses

of cranial bones found in rachitic bossing (XVII).

7) Girls have longer, thicker, wider and therefore heavier legs than boys at 7 and 12, but at 16 - 17 years boys exceeded girls in leg length but not in the other parameters, except in the case of ankles, which were slightly narrower in girls than in boys. White children had the highest values for all measurements (Table XVIII).

8) Girls have mean armgirths greater than those of boys. Their triceps skinfold exceeds that of boys, except at 7 years, where values were higher only in White girls. Bantu girls of 16 - 17 years had similar armgirths to Whites, but greater values for skinfolds. In all other measurements Whites had the highest mean values (Table XIX).

The role of obesity on knock knee

1) Increase in obesity and overweight occurred with rise in privilege (Table XX).

2) There was also a rise with age in the prevalences of obesity and overweight in all groups except Indian females, who showed a decrease at 16 - 17 years, compared with that at 7 and 12 years (Table XX).

3) Prevalences of obesity and overweight for Bantu girls were about half those for White girls. A greater number of White boys were overweight than obese, but proportions were similar for girls (Table XX). Generally, more girls than boys were obese, but this was variable, particularly in the case of Indian girls, who have the lowest prevalence of obesity after puberty.

4) In the cases of obesity, associated with leg abnormalities,

by far the greater proportion of obese subjects had knock knee rather than bowed or straight legs (Table XXI). With overweight subjects, the above was largely true but a greater proportion fell into the straight legs category (Table XXII).

5) When considering only obese pupils about three quarters of girls and one half of boys had knock knee (Table XXIII).

6) If one had a leg abnormality of 2,5 cm or more, and was obese, there was less likelihood of that abnormality being bowing than knock knee. However, by no means all obese children had knock knee. The converse also holds true that not all with knock knee were obese (Table XXIV).

7) A salient point of interest was a remarkable increase in the prevalence of obese-with-knock knee in girls from 7 to 16 - 17 years, except Indian girls, who had a lower prevalence at this age than at 7 years, as was also the case with Indian boys. Coloured and White boys exhibited the same rise with age, but Bantu boys had a low prevalence throughout the age period (Table XXIV).

8) Although there was a close association between the pubertal growth spurt and leg abnormalities, particularly bowing, also knock knee and obesity, it cannot be assumed that these abnormalities are invariably due to rickets, until more definitive studies are undertaken.

Growth curves : adolescent growth spurt

1) Growth curve data as seen in Figures 1 - 4 indicate that Bantu children reached the growth spurt approximately one year later than White children, namely, between 11 - 14 years for girls and 13 - 16

years for boys; compared with 10 - 13 years for White girls and 12 - 15 years for White boys. Coloured and Indian children were similar to Bantu, i.e. growth spurts were delayed by approximately one year.

3.3.4. Abnormalities of Chest

Prevalences of chest abnormalities

In Table XXVI the prevalence of abnormalities of the chest occurring in each age, sex and ethnic group are given.

Table XXVI appears on page 120.

Abnormalities of the chest occurring with bowing, or knock knee of 2,5 cm or more, or with straight legs, are given in Table XXVII.

Table XXVII appears on page 121.

Table XXVIII gives the prevalences of exaggeration of the curvature (handle-bar-effect) of the clavicles and winging of the scapulae, as observed in each age, sex and ethnic group.

Table XXVIII appears on page 122.

Table XXVI: Prevalences of chest abnormalities according to age, sex, and ethnic group

Age	Group	Sex	No.of subj.	Harrison's sulcus %	Flaring of ribs %	Pigeon breast %	Funnel-shaped sternum %	Narrow, flat chest %
	Bantu	M	92	1,1	17,4	1,1	2,2	19,5
	Rural	F	105	1,0	15,2	0,0	1,9	20,0
	Urban	M	72	2,8	16,7	1,4	5,6	23,6
		F	73	1,3	16,5	0,0	2,7	12,4
	Coloured	M	86	1,2	14,0	0,0	2,3	20,9
	Urban	F	83	1,2	6,0	0,0	0,0	13,5
7	Indian	M	72	0,0	8,3	1,3	0,0	11,1
years	Urban	F	80	0,0	7,5	0,0	0,0	3,6
	White	M	36	2,7	5,6	0,0	5,6	8,3
	Rural	F	38	0,0	5,3	0,0	2,6	7,9
	Urban	M	70	4,3	11,4	0,0	0,0	10,0
		F	61	1,6	4,9	0,0	1,6	8,2
	Bantu	M	91	2,2	8,8	0,0	3,3	17,5
	Rural	F	90	2,2	5,5	0,0	0,0	25,5
	Urban	M	60	1,7	13,3	0,0	5,0	20,0
		F	55	1,8	10,9	0,0	1,8	16,4
	Coloured	M	108	1,8	11,1	0,0	3,7	23,1
	Urban	F	86	0,0	8,1	0,0	2,3	8,1
12	Indian	M	49	0,0	8,2	0,0	0,0	8,2
years	Urban	F	49	0,0	4,1	0,0	0,0	8,2
	White	M	21	0,0	0,0	0,0	4,8	4,8
	Rural	F	28	3,6	0,0	0,0	0,0	0,0
	Urban	M	66	4,5	9,1	0,0	1,5	6,1
		F	87	0,0	4,6	0,0	1,1	2,3
	Bantu	M	85	0,0	12,9	0,0	1,2	20,0
	Rural	F	63	0,0	4,8	0,0	0,0	14,9
	Urban	M	55	5,5	5,5	0,0	3,6	18,2
		F	65	0,0	6,2	0,0	1,5	16,9
	Coloured	M	49	2,0	6,1	0,0	12,2	57,1
	Urban	F	49	2,0	10,2	0,0	6,1	59,2
16-17	Indian	M	45	2,2	0,0	0,0	2,2	53,3
years	Urban	F	44	4,5	15,9	0,0	2,3	18,2
	White	M	96	1,0	7,3	1,0	8,3	31,3
	Urban	F	52	0,0	5,8	0,0	3,8	44,2

Table XXVII: Prevalences of chest deformities in schoolchildren with bowing or knock knee of 2,5 cm or more, or straight legs

Leg abnormality	Age yrs.	No.of subj.	Harrison's sulcus %	Flaring of ribs %	Pigeon breast %	Funnel-shaped sternum %	Narrow, flat chest %
Bowing	7	117	1,7	7,7	0,0	0,0	17,1
	12	166	1,2	6,6	1,2	1,8	17,5
	16-17	164	3,0	9,8	0,0	10,3	46,8
Knock knee	7	183	1,1	12,1	0,0	4,3	6,0
	12	156	1,3	6,4	0,0	1,9	8,3
	16-17	108	0,9	5,5	0,9	3,7	12,1
Straight legs	7	340	2,3	13,5	0,8	2,3	16,7
	12	256	2,7	7,8	0,0	3,9	16,7
	16-17	177	2,2	7,9	0,0	2,2	23,6

Table XXVIII: Prevalences of abnormalities of clavicles, scapulae and shoulders

Age	Group	Sex	No.of subj.	Exag. curve of clavicles %	Winging of scapulae %	Shoulders Round %	Shoulders Raised %
7 years	Bantu	M	164	45,6	61,0	12,2	15,9
		F	178	49,3	56,2	15,7	15,2
	Coloured	M	86	50,0	57,0	20,9	15,1
		F	83	30,1	59,0	20,5	10,8
	Indian	M	72	47,2	61,1	20,8	11,1
		F	80	33,8	51,3	33,8	5,0
	White	M	106	25,5	61,3	2,8	12,3
		F	99	24,2	48,4	10,1	15,2
12 years	Bantu	M	151	77,2	67,0	14,5	10,6
		F	145	67,5	62,5	11,0	8,9
	Coloured	M	108	55,6	68,5	9,3	5,6
		F	86	40,7	43,0	14,0	4,7
	Indian	M	49	71,4	53,1	15,4	2,0
		F	49	59,2	51,0	10,2	4,1
	White	M	87	47,0	42,5	6,9	10,3
		F	115	33,9	38,2	9,6	6,9
16-17 years	Bantu	M	140	60,0	52,2	15,7	17,1
		F	128	62,3	26,6	25,7	17,1
	Coloured	M	49	81,6	46,9	57,1	65,3
		F	49	55,1	36,7	65,3	69,4
	Indian	M	45	57,8	60,0	60,0	60,0
		F	44	38,6	45,5	50,0	56,8
	White	M	96	12,5	62,5	40,6	34,4
		F	52	57,7	25,0	42,3	59,6

3.3.5. Summary of Results

Prevalence of chest abnormalities

1) Abnormalities affecting the chest wall, such as Harrison's sulcus, pigeon breast, funnel-shaped sternum, flaring or flailing of ribs, and flattened, narrow chests, occurred more often in boys than in girls (Table XXVI).

2) The above chest abnormalities occurred as often in children with straight legs as in those with bowing or knock knee. A greater prevalence of flattened, narrow chests occurred in pupils of 16 - 17 years than in younger children, particularly in those with bowed legs (Table XXVII).

3) The prevalence of exaggerated clavicular curves (handle-bar-effect) was higher in boys than girls, and at 16 - 17 and 12 years, than at 7 years, except in the case of White 16 - 17 year old boys, in whom this abnormality was less frequent. Bantu, Coloureds, and Indians had higher prevalences than Whites (Table XXVIII).

4) More boys than girls had winged scapulae, which occurred in one half to two thirds of children, affecting all groups similarly, except White girls of 16 - 17 years among whom only one quarter were affected (Table XXVIII).

5) Similar prevalences of round shoulders occurred in both boys and girls, with fewer White children of 7 and 12 years affected. The condition was more common at 16 - 17 years. Raised or high shoulders were usually more common in boys than girls, except in urban White girls, who had a higher prevalence than boys. Prevalences were less

at 12 than at 7 years. Noteworthy were the very low prevalences in 12 year-old Indians (Table XXVIII).

3.3.6. Abnormalities Resulting from Other Skeletal Involvement

Prevalences of dental abnormalities

The prevalences of crowded or widely spaced teeth in both upper and lower jaws according to age, sex and ethnic group are given in Table XXIX).

Table XXIX appears on page 126.

Table XXX gives the percentages of children who have had deciduous or permanent teeth filled or extracted (data collected by questionnaire survey - section 2.3.7.) together with the presence of crowded or widely spaced teeth (data from anthropometric survey - section 2.3.3.) in the various interracial groups.

Table XXX appears on page 127.

The percentage of children with various types of 'bite' are shown in Table XXXI; overbite (upper jaw extends over lower); prognathous (lower beyond upper), or together (teeth meeting on closure) in interracial groups of schoolchildren.

Table XXXI appears on page 128.

Prevalences of flat foot (pes valgus)

The prevalences of flat foot (medial border of foot in contact with floor or flat surface) in interracial groups of schoolchildren according to age and sex are given in Table XXXII.

Table XXXII appears on page 129.

Prevalences of leg pains (growing pains)

In Table XXXIII prevalences of leg 'pains' in groups of school-children are shown. These 'pains' were non-specific and appeared to occur mainly in knees and thighs.

Table XXXIII appears on page 130.

Table XXIX : Prevalences of crowded and widely spaced teeth in upper and lower maxillae

Age	Group	Sex	No.of subj.	Crowding Upper %	Crowding Lower %	Widely spaced Upper %	Widely spaced Lower %
7 years	Bantu	M	164	18,3	47,0	18,9	9,7
		F	178	21,3	38,3	18,5	8,4
	Coloured	M	86	10,5	65,1	3,5	1,2
		F	83	18,1	59,0	7,2	1,2
	Indian	M	72	27,8	68,1	8,3	6,9
		F	80	27,5	73,8	10,0	5,0
	White	M	106	16,1	61,5	14,2	1,9
		F	99	25,2	63,5	18,2	1,1
12 years	Bantu	M	151	12,6	27,2	7,3	5,3
		F	145	14,5	31,0	13,8	8,9
	Coloured	M	108	29,6	63,9	1,9	0,9
		F	86	17,4	34,9	5,8	1,2
	Indian	M	49	22,4	53,1	6,1	0,0
		F	49	34,7	49,0	4,1	0,0
	White	M	89	18,3	44,6	4,6	1,1
		F	115	25,2	47,6	7,8	0,0
16-17 years	Bantu	M	119	5,0	7,5	9,2	8,4
		F	109	4,5	11,0	4,5	6,4
	Coloured	M	49	18,4	30,6	8,2	0,0
		F	49	8,2	36,7	10,2	2,0
	Indian	M	45	35,6	44,4	13,3	0,0
		F	44	36,7	34,1	2,3	0,0
	White	M	96	29,2	52,1	6,3	2,1
		F	52	32,7	42,3	3,8	0,0

Table XXX: Percentage of schooldchildren with milk or permanent teeth filled or extracted, also spacing of teeth

Group	Milk teeth		Permanent teeth		Crowding of teeth		Widely spaced	
	Filled*	Extracted+	Filled*	Extracted+	Upper jaw	Lower jaw	Upper jaw	Lower jaw
	%	%	%	%	%	%	%	%
Bantu	12,4	10,2	16,6	19,9	12,3	26,1	11,5	7,1
Coloured	6,1	43,0	14,5	34,6	18,2	51,4	5,4	1,8
Indian	5,9	36,1	12,2	31,0	27,1	56,9	7,7	2,6
White	49,6	54,3	43,5	21,9	23,7	52,6	11,5	1,8

* Filled, and + extracted: data collected by questionnaire.

Table XXXI: Prevalence of types of 'bite' according to age and ethnic group

Group	Age years	No.of subj.	Oberbite (upper jaw overlaps lower) No.	%	Prognathous (lower beyond upper) No.	%	Together (teeth meeting) No.	%
Bantu	7	342	218	64,0	18	5,2	104	30,5
	12	296	248	83,5	4	1,3	45	15,2
	16-17	228	145	63,3	2	0,8	78	34,1
Coloured	7	169	123	72,9	12	7,1	34	20,1
	12	194	164	84,5	3	1,5	19	9,7
	16-17	98	76	77,5	2	2,0	19	19,4
Indian	7	152	124	81,5	4	2,6	24	15,8
	12	98	92	94,0	0	0,0	3	3,1
	16-17	89	77	86,5	0	0,0	12	13,5
White	7	225	146	65,0	7	3,1	15	6,6
	12	202	168	83,0	2	0,9	19	9,4
	16-17	148	130	88,0	1	0,6	15	10,1

Table XXXII: Prevalences of flat foot (pes valgus)* at 16 - 17 years on visual examination

Group	Sex	No.of subj.	16 - 17 years Flat foot No.	%
Bantu	Male	385	142	36,8
	Female	570	207	36,4
Coloured	Male	198	38	19,2
	Female	128	30	23,4
Indian	Male	304	60	19,7
	Female	147	25	17,1
White	Male	98	6	6,1
	Female	96	7	7,3

* Flat foot - inner border of foot touching the ground or flat surface.

Table XXXIII: Prevalences of leg 'pains'

Group	Sex	No.of subj.	7 years No.	%	No.of subj.	12 years No.	%	No.of subj.	16-17 years No.	%
Bantu	M							247	32	12,9
	F							293	52	17,7
Coloured	M							198	39	19,7
	F							128	37	28,9
Indian	M							148	43	28,9
	+	126	24	19,1	81	20	24,6			
	F							69	20	28,9
White	M	139	43	30,8	56	18	31,5	71	10	14,1
	F	114	34	29,8	46	17	36,1	100	50	50,0

3.3.7. Summary of Results

Prevalences of dental abnormalities

1) There was a lower prevalence of crowding in all groups in the upper than in the lower jaw, and more widely spaced teeth occurred more frequently in upper than in lower jaws. Crowding was common, particularly in White boys, less in Bantu, who, on the other hand, had a higher prevalence of widely spaced teeth (Table XXIX).

2) The higher the prevalence of crowded teeth the higher the extraction rate, almost invariably indicating a high prevalence of dental caries. In Whites crowding was also associated with the greater number of teeth filled, both in deciduous and permanent dentitions. This situation may, however, only reflect better dental care, or awareness of its necessity, in Whites.

The greater prevalence of widely spaced teeth in Bantu was associated with much lower extraction rates in both deciduous and permanent teeth (Table XXX).

3) In the ethnic groups by far the greatest prevalence was that of overbite (upper jaw overlapping lower) found in all children. However, in Bantu, there was a higher prevalence of teeth meeting evenly, namely, three to four times that of Whites. Indians resembled Whites; Coloureds tended to the Bantu pattern.

The most surprising observation was that there was an increase in prevalence of overbite by 12 years. That of evenly meeting jaws decreased. However, by 16 - 17 years, prevalences once again returned to the 7-year pattern, except in the case of Whites, among whom

prevalences continued to increase. Prevalence of prognathous jaws, where the lower jaw juts beyond the upper, decreased with age in all groups. This latter malformation was uncommon.

Prevalences of flat foot (pes valgus)

1) Prevalences of flat foot were greater in Bantu than in White children. Coloured and Indian groups had intermediate values.

Prevalences of leg pains (growing pains)

1) The prevalences of leg 'pains' in White children was found to decrease with age in boys, but to increase in girls, also in Indian children (at 7 and 12 years both sexes were combined - see method). Prevalences were lower in Bantu boys and girls of 16 - 17 years than in Coloured and Indian children. White boys had the lowest prevalences at this age. Pains were usually described as being present in knees and thighs, than in other parts of the legs, and more often after sustained exercise than at rest.

3.4. SPECIAL INVESTIGATIONS ON HIGH AND LOW FISH-EATERS

3.4.1. Dietary Studies

In Table XXXIV the mean daily intakes of vitamin D for Coloured 12 year old children is shown, also ranges, and mean daily intakes of the upper and lowest quartiles.

Table XXXIV: Estimated mean daily intakes of vitamin D in 12 year-old pupils from high and low fish-eating communities, also ranges, and mean daily intakes of upper and lowest quartiles

Group	No.of subj.	Vitamin D I.U./day	Range I.U./day	Upper quartile I.U./day	Lowest quartile I.U./day
High fish-eaters	58	311	39-1078	569	113
Low fish-eaters	93	224	65- 455	354	119

3.4.2. Prevalences of Leg and Other Skeletal Abnormalities

The prevalences of bowing and knock knee of 2,5 cm or more, are shown in Table XXXV in Coloured high and low fish-eaters of 7, 12 and 16 - 17 years.

Table XXXV appears on page 134.

Table XXXV: Prevalences of bowing and knock knee of 2,5 cm or more, in high and low fish-eating Coloured communities

Age	Sex	High fish-eaters No.of subj.	Bowing %	Knock knee %	Low fish-eaters No.of subj.	Bowing %	Knock knee %
7 years	M	44	0,0	38,6	52	3,8	32,6
	F	56	5,4	33,8	48	0,0	37,5
12 years	M	50	10,0	28,0	43	6,9	25.6
	F	36	8,3	52,8	55	1,8	36,4
16-17 years	M	55	18,2	12,7	113	13,2	22,1
	F	60	16,8	39,1	105	9,5	34,2

In Table XXXVI the prevalences of chest abnormalities are given for Coloured schoolchildren.

Table XXXVI: Prevalences of chest abnormalities in 7 and 12 year old children, from high and low fish-eating communities, with bowing, or knock knee of 2,5 cm or more, or straight legs

Area	Leg abnormality	Age yrs.	No.of subj.	Harrison's sulcus %	Flaring of ribs %	Pigeon chest %	Funnel chest %	Flat & narrow %
High fish-eaters	Bowing	7	96	1,2	5,2	0,0	2,1	0,0
		12	85	2,3	1,2	0,0	1,2	8,2
	Knock Knee	7	96	0,0	2,1	0,0	2,1	2,1
		12	85	1,2	4,7	0,0	1,2	5,8
	Straight legs	7	96	2,1	1,2	0,0	2,1	0,0
		12	85	1,2	4,7	0,0	2,3	3,5
Low fish-eaters	Bowing	7	101	3,9	1,9	0,0	7,9	7,9
		12	98	2,0	4,1	0,0	6,1	5,1
	Knock knee	7	101	0,9	0,0	0,0	0,9	1,9
		12	98	0,0	0,0	0,0	1,1	4,1
	Straight legs	7	101	0,0	3,9	0,0	5,9	10,9
		12	98	3,1	0,0	0,0	1,1	2,0

3.4.3. Summary of Results

1) Mean intakes of vitamin D were considerably higher in the high fish-eating community, namely 311 I.U./day, than in the low fish-eating community, namely 224 I.U./day. Ranges of intakes were wide, particularly in the high fish-eating groups where some families were extremely poor. However, on consideration of the upper and lower quartiles, intakes in high fish-eating groups were considerably higher than the recommended daily intake of 400 I.U., and the lowest were equivalent to intakes found elsewhere.

2) No differences could be found in prevalences of leg abnormalities between high and low fish-eating groups. Prevalences were similar to those found in Johannesburg Coloureds, who had even lower mean daily vitamin D intakes. Prevalences of bowing for children aged 7 to 16 - 17 years ranged from 0% to 18,2% for high fish-eaters; 0% to 13,2% for low fish-eaters; and 6% to 20,2% for Johannesburg Coloureds. Prevalences for knock knee were greater in girls than boys, particularly at 16 - 17 years, and were found to be similar to those of the Johannesburg Coloured pupils.

3) No marked differences could be found in prevalences of chest abnormalities in children with bowing, knock knee or with straight legs, nor were they different to Coloured or other children in the Transvaal.

3.5. RADIOLOGICAL STUDIES

3.5.1. Data on X-ray Examinations

Prevalence of rachitic manifestations

In Table XXXVII data derived from X-ray studies on the hands and wrists of groups of urban Bantu pupils of 16 - 17 years, with bowing or knock knee of 2,5 cm or more, or straight legs, are given.

Table XXXVII : Prevalences of active rickets, shortening of ulna, also osteoporosis, in hand and wrist X-rays of Bantu pupils of 16 - 17 years, according to bowing or knock knee of 2,5cm or more, or straight legs, also in total sex groups

Leg abnormality	Sex	No.of subj.	Active rickets %	Short ulna x %	Osteoporosis* (subjective impression) 1 %	2 %	3 %
Bowing	M	14	0,0	7,1	0,0	0,0	0,0
	F	18	0,0	22,2	16,6	16,6	11,1
Knock knee	M	15	0,0	20,0	6,6	6,6	6,6
	F	17	0,0	5,9	5,9	5,9	5,9
Straight legs	M	12	0,0	25,0	8,3	8,3	8,3
	F	20	0,0	15,0	25,0	25,0	5,0
Total	M	41	0,0	17,1	4,8	4,8	4,8
	F	55	0,0	14,3	16,1	16,1	7,1

x - Short ulna - head of ulna does not reach distal articular surface of radius

* - Osteoporosis - the assessment was subjective as without objective densiometry measurements were not entirely satisfactory.

1. Juxta-articular bone ends at inter- and metacarpal-phalangeal joints show diminished density and coarse trabecular striation.
2. As above, and in addition similar appearance in distal radius, ulna and carpus.
3. As above, and in addition transverse cortical width of second (index) metacarpal at widest part of shaft, was less than transverse width of spongiosa and medulla.

Bone dimensions

In Table XXXVIII the mean cortical thickness and cortical score of the second (index) metacarpal are given for groups of urban Bantu pupils of 16 - 17 years, with bowing or knock knee of 2,5 cm or more, or with straight legs.

Table XXXVIII : Mean cortical thickness and cortical scores of second (index) metacarpal in hand X-rays of Bantu pupils of 16 - 17 years, with bowing or knock knee of 2,5 cm or more, or straight legs

Leg abnormality	Sex	No. of subj.	Bone dimensions of second metacarpal D	d	D-d	$\frac{D-d}{D}$
Bowing	M	13	8,3	3,8	4,5	0,54
	F	18	7,7	3,3	4,4	0,57
Knock knee	M	15	8,3	3,9	4,4	0,53
	F	18	7,7	3,0	4,7	0,61
Straight legs	M	13	8,4	4,1	4,3	0,51
	F	19	7,6	3,4	4,2	0,57

D - outer width (mid-shaft); d - medullary width;

D - d - cortical thickness; $\frac{D-d}{d}$ - cortical score.

3.5.2. Summary of Results

1) No indications of an active rachitic process were found in pupils with marked bowing of legs, or knock knee, or with straight legs.

2) Shortening of the ulna (a possible manifestation of healed rickets) was visible in 17,1% of boys and 14,3% of girls. Numbers in groups with and without leg abnormalities were too small to draw firm conclusions.

3) Means of cortical thickness and cortical scores of second (index) metacarpal were closely similar in all groups with or without leg abnormalities.

4) There was evidence of osteoporosis in 4,8% of boys and 16,1% of girls, and a severer degree in 4,8% of boys and 7,1% of girls. As the assessment was subjective, and not based on standardized radiological procedures, no attempt to statistically analyse data between groups with and without leg abnormalities was made.

3.6. SKIN PIGMENTATION DETERMINED BY REFLECTANCE PHOTOMETRY

3.6.1. Skin Reflectance Values at 425 and 685 nanometers

Reflectance values at 425 and 685 nm (filters 601 and 609, respectively) on both the left inner upper arm and on the forehead, are shown in Table XXXIX for groups of urban Bantu 16 - 17 year-old pupils, with bowing or knock knee of 2,5 cm or more, or with straight legs.

Table XXXIX : Skin reflectance of inner upper arm and forehead at 425 and 685 nanometers in urban Bantu pupils of 16 - 17 years, with bowing or knock knee of 2,5 cm or more, or straight legs

Leg abnormality	Sex	No.of subj.		Skin reflectance 425 nm Upper arm	425 nm Forehead	685 nm Upper arm	685 nm Forehead
Bowing	M	14	Mean	4,4	5,9	27,4	23,1
			S.D	1,45	1,94	7,28	7,17
	F	18	Mean	7,0	7,8	25,2	27,0
			S.D	1,56	1,49	5,84	6,25
Knock knee	M	14	Mean	5,0	6,1	25,7	20,9
			S.D	1,75	1,69	8,33	6,81
	F	18	Mean	7,6	7,5	26,9	28,2
			S.D	2,03	1,60	7,09	6,44
Straight legs	M	12	Mean	4,4	6,4	26,9	22,4
			S.D	1,22	2,79	6,55	5,95
	F	18	Mean	6,4	7,1	26,2	26,6
			S.D	1,39	1,64	6,98	7,75

3.6.2. Summary of Results

1) The colour of skin as shown by reflectance values, at either the upper arm (little exposure), or on the forehead (continued maximum exposure) revealed no difference between groups of pupils with or without leg abnormalities.

3.7. BIOCHEMICAL STUDIES

3.7.1. Serum Alkaline Phosphatase, Calcium and Inorganic Phosphorus

In Table XL values are given for mean serum alkaline phosphatase, calcium, and inorganic phosphorus values, also their standard deviations, in groups of urban Bantu pupils of 16 - 17 years with bowing and knock knee of 2,5 cm or more, also for those with straight legs.

Table XL appears on page 143.

Table XLI gives values, as above, for Coloured pupils of 12 years in the Cape Province.

Table XLI appears on page 144.

Mean serum alkaline phosphatase for 7 and 10 - 12 year old children are given in Table XLII.

Table XLII appears on page 145.

3.7.2. Summary of Results

1) Mean serum alkaline phosphatase values (in K.A. units/100 ml)

were similar in pupils of 16 - 17 years who had bowed legs, knock knees or straight legs. There were no significant differences; but data were higher than the usual levels reported for this age group. Values in the three groups were, boys, 28,4, 31,2, and 27,8, respectively, and girls, 21,8, 19,8 and 21,4, respectively. Normal values in the literature are 3-10 K.A./100 ml in women, and 5-12 K.A. units/100 ml in men.

2) Apart from Bantu children, whose values were a little higher than those of Indian, White and Coloured children, mean serum alkaline phosphatase values in the ethnic groups were closely similar at 10 - 12 years. For Whites normal values (in K.A. units/100 ml) for this age group are given by Round (1973) as 17,1 - 17,9 for girls, and 16,1 - 18,1 for boys.

3) Values for serum calcium and inorganic phosphorus lay within the normal range in Bantu pupils, irrespective of whether leg abnormalities were present or not. Normal values are reported to be 9,6 for calcium and 3,6 for inorganic phosphorus, mg/100 ml.

Table XL: Means and standard deviations of serum alkaline phosphatase, calcium and inorganic phosphorus in urban Bantu pupils (16-17 years) with bowing or knock knee of 2,5 cm or more, or with straight legs, also percentages above 30 King Armstrong units

Leg abnormality	Sex	No. of subj.		Calcium mg/100ml	Inorganic phosphorus mg/100ml	Alkaline phosphatase K.A. units*	% above 30 K.A. units*
Bowing	M	20	Mean	10,2	3,5	28,4	42,8
			S.D.	0,33	0,62	3,47	
	F	15	Mean	10,3	3,6	21,8	13,3
			S.D.	0,49	0,45	4,36	
Knock knee	M	20	Mean	10,5	4,3	31,2	80,9
			S.D.	0,59	1,09	2,99	
	F	17	Mean	10,3	3,7	19,8	0,0
			S.D.	0,55	0,20	2,99	
Straight legs	M	17	Mean	10,9	3,6	27,8	52,9
			S.D.	0,39	0,81	5,36	
	F	17	Mean	10,4	3,6	21,4	4,9
			S.D.	0,48	0,52	4,35	

* K.A. units - King Armstrong units

Table XLI: Means and standard deviations of serum alkaline phosphatase, calcium and inorganic phosphorus in Coloured pupils of 12 years, from high and low fish-eating communities, with bowing or knock knee of 2,5 cm or more, or straight legs, also percentages above 30 King Armstrong units

Area	Leg abnormality	Sex	No.of subj.		Calcium mg/100ml	Inorganic phosphorus mg/100ml	Alkaline phosphatase K.A. units*	% above 30 K. units
High fish-eaters	Bowing	M	9	Mean	10,2	4,9	23,6	0,0
				S.D.	0,39	0,28	2,79	
		F	4	Mean	10,2	5,1	24,4	0,0
				S.D.	0,66	0,76	3,39	
	Knock knee	M	10	Mean	10,0	4,7	25,1	8,3
				S.D.	0,98	0,56	4,35	
		F	9	Mean	10,5	5,0	26,3	22,2
				S.D.	0,42	0,37	3,73	
	Straight legs	M	11	Mean	10,3	5,0	25,5	18,1
				S.D.	0,65	0,38	5,16	
		F	7	Mean	10,2	5,0	26,6	14,3
				S.D.	0,52	0,27	4,04	
Low fish-eaters	Bowing	M	9	Mean	10,1	4,9	28,2	44,4
				S.D.	0,26	0,46	2,86	
		F	10	Mean	10,6	5,2	28,4	33,3
				S.D.	0,43	0,41	2,98	
	Knock knee	M	8	Mean	9,8	5,1	26,2	10,0
				S.D.	0,41	0,31	4,02	
		F	8	Mean	10,3	5,2	28,8	33,3
				S.D.	0,41	0,42	1,43	
	Straight legs	M	10	Mean	9,9	4,7	27,9	50,0
				S.D.	0,39	0,52	4,20	
		F	9	Mean	10,1	4,9	27,8	22,2
				S.D.	0,27	0,54	3,29	

* K.A. units - King Armstrong units

Table XLII: Means and standard deviations of serum alkaline phosphatase in 7 and 10 - 12 year children, also percentages above 30 King Armstrong units

Group		7 years No.of subj.	Alkaline phosphatase K.A. units Mean	S.D.	% above 30 K.A. units*	12 years No.of subj.	Alkaline phosphatase K.A. units Mean	S.D.	% above 30K.A units*
Bantu Rural	M					25	29,8	3,0	60,0
	F					21	30,1	2,9	52,3
Urban	M					27	26,0	3,9	18,5
	F					26	27,4	4,7	34,5
Coloured Urban	M	17	27,8	2,5	17,6	23	23,9	5,2	16,3
	F	16	28,3	1,9	18,7	22	26,0	4,8	22,7
Indian Urban	M	21	25,0	3,2	4,7	87	26,1	4,1	18,3
	F	20	23,6	2,7	0,0	90	26,4	3,7	17,8
White Urban	M	9	27,1	4,7	22,3	9	24,5	5,5	22,2
	F	3	21,2	2,5	0,0	8	25,0	2,8	0,0

* K.A. units - King Armstrong units

3.8. QUESTIONNAIRE SURVEY ON DEVELOPMENT PATTERNS AND INFECTION RATES

3.8.1. Milestones of Development

The average ages, in months, for 50% of infants reaching each milestone of development namely, sitting, standing, walking and teething, are shown in Table XLIII for each ethnic and sex group, also prevalences of 'late starters' in each group.

Table XLIII appears on page 147.

Figures Depicting Milestones of Development

Figures 5 - 8 depict the milestones of development in each age, sex, and ethnic group for sitting, standing, walking and teething, respectively.

Figure 5 appears on page 148.

Figure 6 appears on page 149.

Figure 7 appears on page 150.

Figure 8 appears on page 151.

3.8.2. Upper Respiratory Tract Infections

The percentage of children reported as having experienced episodes of tonsilitis or adenoids, and those who characteristically snore and breathe through the mouth, in each ethnic and sex group, are shown in Table XLIV.

Table XLIV appears on page 152.

In Table XLV the prevalences in each sex, of bronchitis, pneumonia and whooping cough are shown, also the percentages that have fractured legs or arms.

Table XLV appears on page 153.

Table XLIII: Age range in months for 50 percent of infants reaching various milestones of development, and prevalence of 'late starters'

(from questonnaire answered by mothers)

Group	No.of subj.	Sitting Approx. 50%	Sitting Range mths.	Standing Approx. 50%	Standing Range mths.	Walking Approx. 50%	Walking Range mths.	Teething Approx. 50%	Teething Range mths.
Bantu	860	49,7%	5-6	60,0%	8-10	55,6%	10-12	50,1%	5-7
Coloured	1736	45,5%	5-6	60,3%	8-10	48,6%	11-12	47,0%	5-7
Indian	1796	55,0%	6-7	58,6%	9-11	49,0%	11-12	47,2%	6-8
White	2537	58,5%	5-6	54,2%	8-10	58,5%	11-13	47,9%	4-6

'Late starters'

Group	No.of subj.	Sitting 11+mths.	Sitting %	Standing 15+mths.	Standing %	Walking 18+mths.	Walking %	Teething 13+mths.	Teething %
Bantu	860	12	1,4	9	1,0	27	3,1	8	0,9
Coloured	1736	16	0,9	30	1,7	78	4,4	38	2,2
Indian	1796	70	3,9	66	3,6	131	7,3	89	4,9
White	2537	31	1,2	60	2,3	109	4,3	56	2,2

Figure 5

MILESTONES OF DEVELOPMENT
AGE AT SITTING

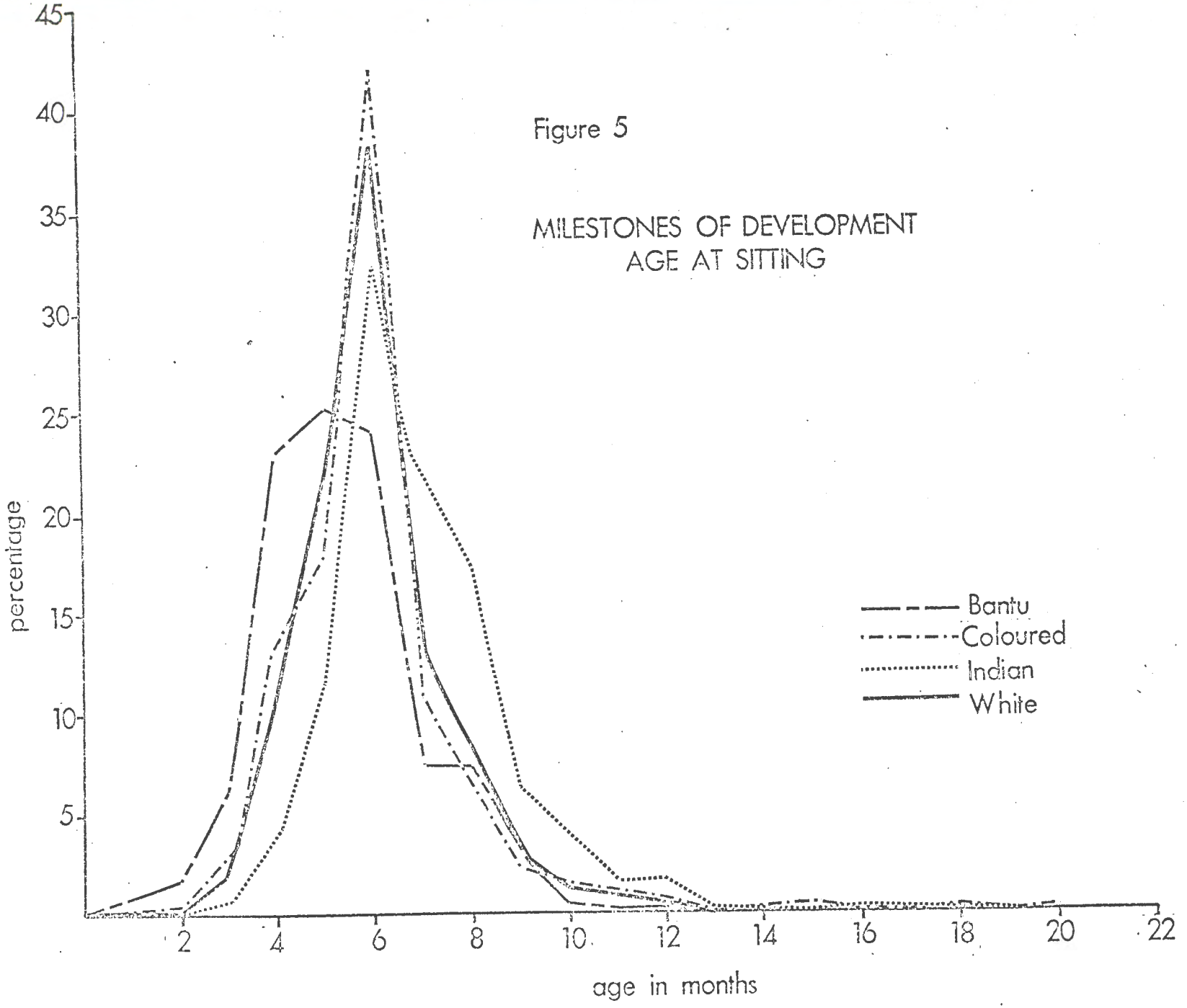


Figure 6

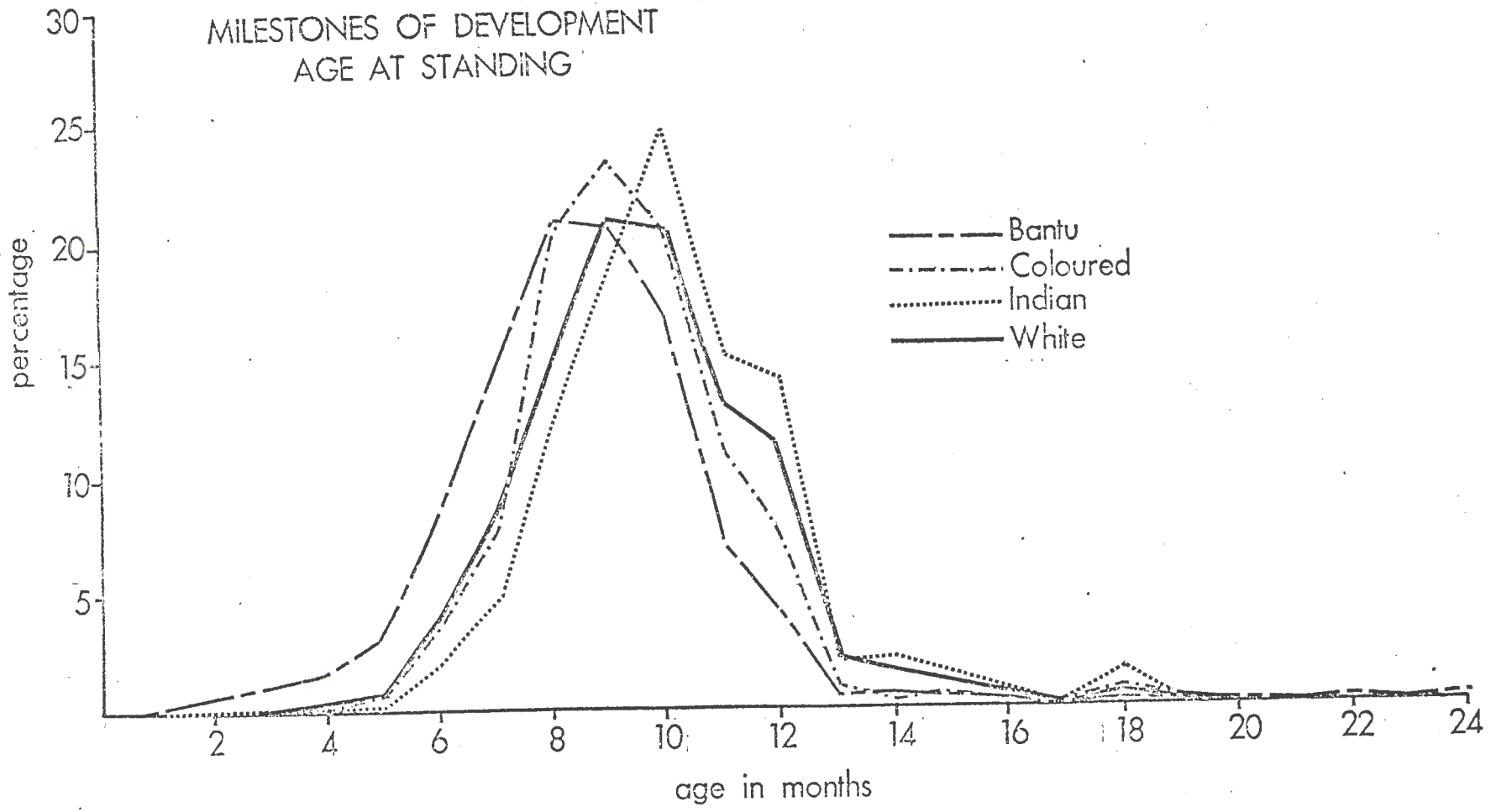

MILESTONES OF DEVELOPMENT
AGE AT STANDING
percentage
30
25
20
15
10
5
2
4
6
8
10
12
14
16
18
20
22
24
age in months
Bantu
Coloured
Indian
White

Figure 7

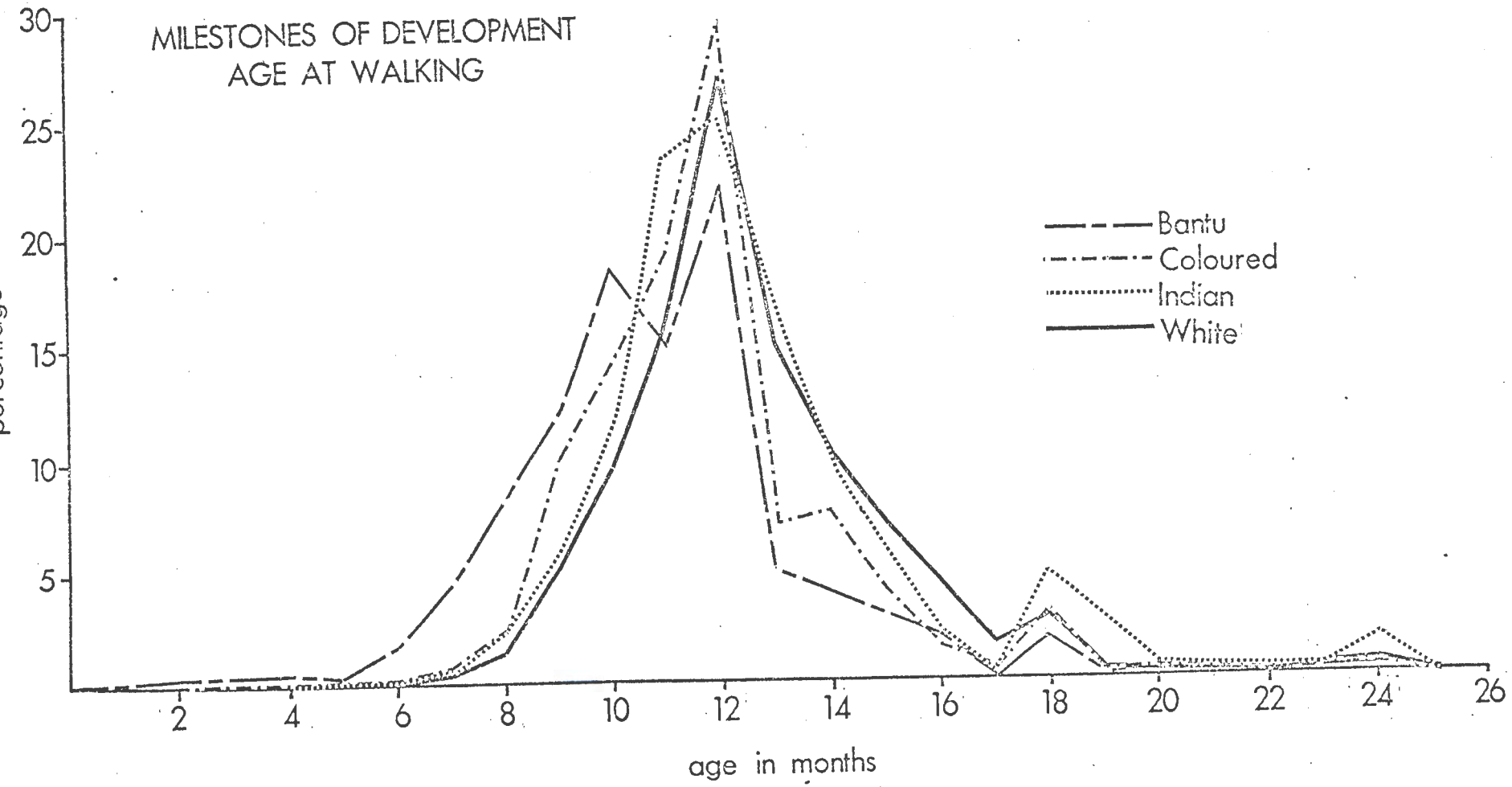

MILESTONES OF DEVELOPMENT
AGE AT WALKING
30
25
20
15
10
5
2
4
6
8
10
12
14
16
18
20
22
24
26
age in months
Bantu
Coloured
Indian
White

Figure 8

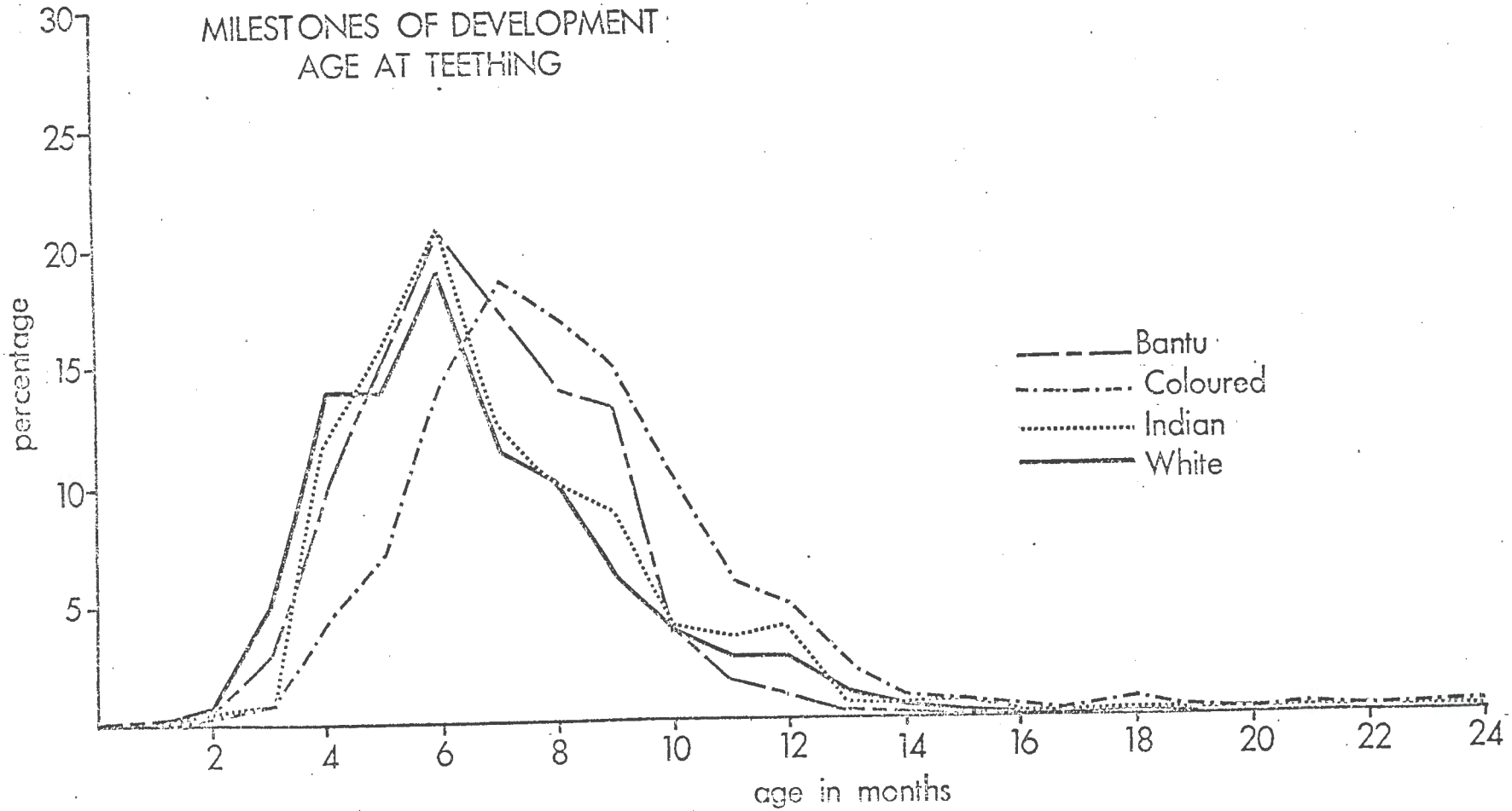

MILESTONES OF DEVELOPMENT
AGE AT TEETHING
30
25
20
15
10
5
percentage
2
4
6
8
10
12
14
16
18
20
22
24
age in months
Bantu
Coloured
Indian
White

Table XLIV: Prevalences of tonsilitis, adenoids, snoring and mouth breathing

Group	No.of subj.	Percentage			
		Tonsilitis %	Enlarged adenoids %	Snoring %	Mouth breathing %
Bantu	933	27,1	4,5	8,3	21,0
Coloured	1860	21,8	5,3	19,8	21,5
Indian	1856	17,3	3,6	14,4	13,0
White	2753	27,5	8,1	22,2	14,0

Table XLV: Prevalences of pneumonia, bronchitis and whooping cough, also fractures of arms and legs

Group	No.of subj.	Pneumonia %	Bronchitis %	Whooping cough %	Fractures Arm %	leg %
Bantu	933	6,9	64,0	32,0	9,4	3,9
Coloured	1860	7,6	15,2	28,5	8,6	4,7
Indian	1856	3,0	9,3	14,3	5,9	1,7
White	2753	5,9	9,9	27,9	10,0	2,5

3.8.3. Summary of Results

Milestones of development

1) Bantu infants sat, on an average, a month earlier (5 months) than Coloured, Indian or White infants (6 months); Bantu girls were more advanced, as they sat earlier (4 months) than contemporary girls of other ethnic groups (6 months).

2) Bantu infants stood at 8 - 9 months on average; girls stood a little earlier than boys; Coloured and White infants stood at 9 months and Indians at 10 months.

3) Most Bantu girls started to walk earlier (10 months) than boys, who started at the same average age of 12 months, as did most other groups, except Indian boys, who started to walk on average at 11 months.

4) Indian infants teethed at an average age of 7½ months, girls at 8, and boys at 7 months, approximately one month later than Bantu, Coloured and White infants (6 months).

5) The average time span for all infants for sitting lay between 3 - 11 months; 5 - 13 months for standing; 8 - 16 months for walking and 3 - 13 months for teething.

6) Only a small proportion of all infants were late 'starters'; approximately 1% sat late, i.e. between 11 - 13 months, except for Indian babies of whom 3,9% were late 'sitters'. More Indian (3,6%) and White (2,3%) infants were late in standing (14 - 16 months) than were Coloureds (1,7%) and Bantu (1,0%). 7,3% Indian, 4% Coloured, 4% White, and 3,1% of Bantu babies walk late (17 - 20 months). Very

few Bantu infants cut their teeth later than 13 months, but 4,9% Indian infants were late 'teethers', the percentage was 2,2% for both Coloureds and Whites.

7) Bantu babies were more advanced in all parameters than those of other groups. Indian babies had the highest proportion of late 'starters'.

Upper Respiratory Tract Infections

1) Snoring was more frequent among White and Coloured children, (approximately 20%), than in the other groups. Almost one quarter Bantu and Coloured children breathe through the mouth, whilst only 13% Indians and Whites did so. Tonsilitis, affected one quarter of children, but the prevalence was lower in Indians. Adenoids were not very common (4 - 8%) with slightly more White children being affected than other groups.

2) Much the same prevalence of pneumonia (3 - 7%) was reported in all groups. Bronchitis was an extremely common condition among Bantu children (64,0%); the infection only affected 9 - 15% of other groups. Whooping cough was common (14 - 33%), but occurred less frequently in Indian children.

Prevalences of fractured limbs

1) Both leg (2 - 5%) and arm (6 - 10%) fractures occurred with similar low frequencies, in all children; there was a lower frequency in Indian children. Arm fractures occurred 2 - 3 times more frequently than leg fractures.

3.9. COMBINED SUMMARY OF RESULTS

A combined summary of the main points is set out below.

3.9.1. Dietary Studies

1) The dietary contribution of vitamin D in all ethnic groups, apart from the Coloured fish-eating community, was low, particularly so in Bantu children.

2) Coloured children resident in fishing villages in the Cape Province, who regularly eat large quantities of fish, had the highest dietary intake of vitamin D, many of whom had daily intakes greater than 400 I.U. - the recommended allowance for children.

3) Public health measures played only a minor role in prevention of rickets in childhood, apart from the use of formulae diets for infants, in which vitamin D is incorporated.

3.9.2. Prevalence of Skeletal Abnormalities: Anthropometric and Clinical Observations

1) Prevalences of bowing and knock knee were more common than expected. Less than half of all children were found to have straight legs.

2) Sex differences in prevalence of leg abnormalities were not apparent in younger groups, however, older boys had more bowing than girls. There was more knock knee among girls than boys.

3) A definite age trend was apparent, in which bowing occurred more frequently at 16 - 17 years than at 7 years; i.e. there was an increase with age. This prevailed in all groups, except Indians.

This same trend did not occur with knock knee, prevalences of which remained fairly constant. Prevalences of straight legs decreased with age; there were fewer older than younger children who had straight legs.

4) Data on severity of abnormalities were similar in the different ethnic groups.

5) Rural-urban differences in prevalences of leg abnormalities were not apparent.

Influencing Factors

Mean anthropometric measurements

1) There were only slight variations in anthropometric data between age-sex-ethnic groups, with White children having the highest values, in measurements of head width, wrist width and girth, also leg widths at knee, calf and ankle. Although head widths were similar in all groups, Coloured and Indian children had smaller heads than Bantu and Whites, i.e. they had smaller head circumferences.

2) Girls had greater mid-thigh circumferences than boys; this was particularly noticeable in older Bantu girls. Younger girls of 7 and particularly at 12 years, not only had longer legs than boys, but were taller. Boys of 16 - 17 years, however, had longer legs and were taller than girls. These latter observations demonstrate clearly the female ascendency, which occurs during the pubertal growth spurt.

3) White children had greater mean body mass and were taller at all ages than children of the other ethnic groups. At 12 years, in

all groups, boys had a lower mean body mass than girls of the same age. This observation again relates to female ascendancy occurring at puberty. Indian girls of 16 - 17 years had the lowest body mass index (mass/height^2). Body mass of Bantu girls increased rapidly after puberty to almost attain that of White girls, but they remained much shorter, therefore their body mass index became greater.

4) Triceps skinfolds were smaller in boys than in girls, and varied with the body mass of individual pupils, being greater in overweight and obese subjects than in non-obese.

The role of obesity on knock knee

1) In obese pupils, knock knee was common, particularly in girls, among whom about half were affected. Mass obviously played an important role in the development of knock knee. It was clear from the group of 16 - 17 year old Bantu pupils studied, that girls with knock knee had significantly greater mean body mass than girls with bowed or straight legs; 47% of girls with knock knee were obese. The situation with boys was different for no boys were obese. Obesity in girls was reflected in their anthropometric measurements, namely, greater mean values for triceps skinfold, also arm and mid-thigh circumferences than boys.

2) In both girls and boys in the obese-with-knock knee category, all, except Indian girls, less noticeably in Indian boys, showed a marked increase of knock knee with age. It should be noted that obesity was almost non-existent in Bantu boys.

Growth curves : adolescent growth spurt

Non-White children, particularly Bantu, reached the growth spurt

approximately one year later than Whites.

Abnormalities of the Chest

Prevalence of chest abnormalities

1) Chest deformities, Harrison's sulcus, flaring of ribs, pigeon chest, funnel breast, and flattened, narrow chests, occurred as often in children with leg abnormalities, as in those with straight legs. These sequelae relate to infant rather than to adolescent rickets.

2) Thoracic abnormalities, such as exaggeration of clavicular curves, also round and raised shoulders, and spinal involvement, occurred more frequently in older children. However, winging of scapulae was present at all ages, tending to be lower in girls, particularly White girls.

Abnormalities resulting from other skeletal involvement

1) Ethnic differences in crowding or spacing of teeth, which were associated with prevalences of types of 'bite' appeared to have a positive bearing on prevalence of caries. Malformation of teeth and jaw structure are usually regarded as occurring in infancy or even *in utero*. However, the changing shape of jaws reflected in the 'bite' prevalences, particularly at 12 years, was unexpected and would appear to indicate a possible wider skeletal involvement in vitamin D deficiency states during the pubertal growth spurt, than was thought likely.

2) There was a greater prevalence of crowding of teeth in lower than in upper jaws in all groups. Coloured, Indian and White children had more crowding than Bantu.

Upper respiratory tract infections and their bearing on skeletal abnormalities

1) About a fifth of children had enlarged tonsils and/or adenoids, and their associated conditions of mouth breathing and snoring. This enlargement blocks the easy passage of air through the nose, with the consequent loss of this important aid in separating the lateral walls of the nose from the septum, which is of particular importance where bones are soft, as occurs in rickets. This 'contraction' of the maxillae leads to malformation, also crowding of teeth. Crowding of teeth, in the upper jaw, was prevalent in White, Indian and Coloured children, but Bantu had lower prevalences.

2) Upper respiratory tract conditions in infancy influence the moulding of the chest wall, or cage, particularly where softening of the bones is present. The greater commonness of bronchitis, also whooping cough, in Bantu children, is probably partly responsible for the greater prevalences of Harrison's sulcus and flaring of the ribs observed in these children at most ages, than in other groups.

3) Vitamin D-resistant rickets is characterised by the ease with which bones are fractured. Since the prevalences of fractures in arms and legs were found to be iow in all ethnic groups it may be inferred that vitamin D-resistant forms of rickets were uncommon in the general schoolchild population.

3.9.3. Radiological Studies

1) X-ray studies conducted on Bantu pupils of 16 - 17 years failed to detect any evidence of an active rachitic process in groups of pupils with bowing, knock knee or straight legs. Healed rickets is

Not readily detectable on X-ray, but one possible manifestation, a short ulna, was present in a proportion of pupils.

2) There appeared to be an adequate deposition of bone, as shown by similar cortical thickness and cortical scores of second (index) metacarpal, in groups of Bantu pupils with and without leg abnormalities.

3) Osteoporotic manifestations in hand and wrist bones were found in a proportion of pupils. This was unexpected, and therefore an assessment could only be made subjectively. The significance of this observation was not readily apparent.

3.9.4. Skin Pigmentation

1) Exposure to greater amounts of ultraviolet radiation did not have a significant effect on prevalences of leg abnormalities.

2) No differences were discernable, by reflectance photometry, in skin colour of the Bantu groups, who had bow legs, knock knee or straight legs, i.e. greater pigmentation in Bantu children did not influence prevalences of these abnormalities.

3.9.5. Biochemical Studies

1) No differences in mean values for serum alkaline phosphatase, calcium or inorganic phosphorus were found in Negro pupils of 16 - 17 years, or in Coloured children of 12 years, irrespective of either marked bowing, knock knee, or straight legs.

2) There were no significant differences in serum alkaline phosphatase values, between high and low fish-eating Coloured children. Values were somewhat higher where children consumed less vitamin D, i.e. low fish-eating groups.

CHAPTER 4

D I S C U S S I O N

4.1. BRIEF COMMENTS ON RESULTS

Before proceeding to the more detailed discussion, and to facilitate appreciations of the parameters, the salient features that have emerged from the study of leg and chest abnormalities in schoolchildren, and which are usually associated with, or are described as sequelae of rickets, are enumerated and commented upon:-

1) Both leg and chest abnormalities were common in all schoolchild populations studied.

2) There were no clear cut ethnic differences in prevalences of leg or chest abnormalities.

3) Neither differences in amounts of exogenous nor in endogenous sources of vitamin D appeared to affect prevalences of bowing or knock knee. The inference is that in the South African context, sufficient vitamin D for daily needs is available. In spite of low vitamin D intakes, any extra, in the form of an increased dietary intake, or greater exposure to sunlight, did not influence prevalences to any obvious extent. Neither were related factors, such as skin pigmentation, or rural-urban location, found to be obviously influential.

4) No correlation was apparent between present leg abnormalities, and those of the chest, which are generally regarded as being of rachitic origin. The sequelae in the chest appear to have their origin in infancy, and are not affected by adolescence.

5) It has been demonstrated that changes in the pattern of leg abnormalities, particularly that of bowing were associated with the

pubertal growth spurt. Body mass was found to be an important factor in influencing the commonness of knock knee; in all groups there were higher prevalences in obese subjects.

6) The period of active growth during adolescence occurred earlier in White than in Non-White groups.

7) No radiological evidence of active rickets was found in the limited groups studied.

8) There was no biochemical evidence of active rickets in the groups studied.

4.1.1. Comments and Brief Discussion of Salient Findings

What determines this remarkable change, particularly of bowing, in the curvature of the bones of the lower limbs in apparently normal healthy children? The change appears to be associated with the growth spurt of puberty, as will be discussed below. It is not clear whether this change occurs between, or at, the same ages in all ethnic or both sex groups.

The role of dietary vitamin D on the pathogenesis of leg abnormalities

An obvious question is - could abnormalities be due to a deficiency of vitamin D, arising partly from low or marginal intakes? The answer to this question lies in the results of the critical study carried out on Coloured children in high and low fish-eating communities in the Cape. Vitamin D intakes were found to be more satisfactory in groups of children regularly consuming large quantities of fish, and yet such had as much bowing and knock knee as those who had lower intakes of

vitamin D. A highly relevant study, not feasible to the writer, would be the determination of levels of plasma 25-HCC, recently regarded as the most accurate available biochemical index of vitamin D status.

A possible raised metabolic requirement of vitamin D at adolescence

The possibility is put forward that there may be a metabolic block, inhibition or retardation in the involved synthesis of vitamin D, cholecalciferol, to its active polar metabolite, 1,25-DHCC, occurring either concurrently with, or divorced from an increased metabolic requirement during the adolescent growth spurt. Such a block, implying a markedly increased need, could cause a temporary vitamin D deficiency during this critical period. This could explain the increased tendency of lower limb bones to bend, thus altering their shape, as has been described, according to the type and degree of pressure exerted on softened bone.

The bearing of the pubertal growth spurt on the prevalence of leg abnormalities

The changing shape of the lower limbs occurred at the time of the growth spurt, the onset of which is part of the complex of changes accompanying puberty (Tobias, 1972). It is significant that most groups of girls of 12 years had higher prevalences of bowing than boys of the same age. Part of the explanation may be in the earlier growth spurt experienced by girls (10 - 13 years) compared with boys (12 - 15 years) (Marshall and Tanner, 1969, 1970), and the increased metabolic need of vitamin D during this period. However, the part played by the delay of about one year in the commencement of the growth spurt experienced by Non-Whites, compared with Whites, is not clear.

The excellent study by Round (1973), discussed previously, showed

a serum alkaline phosphatase flare which paralleled the growth spurt. A rapid fall to normal adult levels was complete at the end of the 15th year in girls, but not until 18+ years in boys. 'In both sexes this fall is due to a reduction in bone phosphatase levels.' In discussing this finding, Stamp and Round (1973) considered that in a vitamin-D-deficient population, with a high prevalence of rickets, 'It would not be unreasonable to expect an increased rate of utilization of 25-HCC during the puberty growth spurt', as a significant inverse correlation between plasma 25-HCC and alkaline phosphatase would be expected in such a population. However, they added that 'Caucasian children are not in this category' and came to the conclusion, 'that the alkaline phosphatase flare is a normal physiological event at puberty.' If this latter view is valid, is it not possible that the changing shape of lower limbs is also physiological, or alternatively, could it not be that there is an accompanying pathological softening of rapidly growing bone?

Are leg abnormalities of pathological origin?

The evidence points to a pathological, probably sub-clinical, involvement of bone metabolism, which, whether due to a deficiency of vitamin D, or to some other unknown factor/s, remains to be clarified. The fact is that schoolchildren exhibit changes in the shape of the legs, and that such changes, increase with age. Further, obese subjects have a greater prevalence of knock knee, due presumably to an increased body mass exerting greater pressure on mass-bearing bones. Not all children show these abnormalities, presumably indicating individual differences in either vitamin D requirement, or some other related metabolic requirement.

Whether there is any justification in proposing a metabolic block, inhibition, or retardation in vitamin D synthesis, with or without a concurrent increased metabolic requirement, is a controversial subject. A number of studies from recent literature illustrate the various points of view.

Dietary studies. According to Swan and Cooke (1971b) a dietary inadequacy of vitamin D in a pigmentated population, was not the reason for the nutritional osteomalacia observed in their Asian subjects. These patients, although having an adequate vitamin D intake, and otherwise in good nutritional condition, exhibited osteomalacia. On the contrary, Hodgkin et al. (1973) working on Asian immigrants to Britain considered that not only was there a low dietary intake of vitamin D, but there was also an inadequate exposure to solar radiation.

Genetic and metabolic studies. Ford et al. (1972b) have suggested that there may have been a genetic difference in the ability of their Pakistani rachitic children to synthesise cholecalciferol, or its conversion to its more active polar metabolites, in liver and kidney. This was questioned by Mawer and Holmes (1972), who found that of three Asian patients, with similar degrees of vitamin D deficiency, all were able to form the potent, biologically active metabolite 1,25-DHCC, the authors therefore concluded that the inability to produce such metabolites was not an explanation of the findings on the subject of Ford et al. (1972b).

In their studies on Asian immigrants to Britain, Preece et al. (1973) have found that 25-HCC levels were significantly lower in a group of Asians, with no clinical or biochemical evidence of rickets

or osteomalacia, compared with a White control group. They concluded that the crucial factor was availability of vitamin D, which resulted in a vitamin D deficiency, rather than an inability to metabolise the vitamin.

Parathyroid hormone and its relationship to vitamin D metabolism. In Bantu children with nutritional rickets, elevated concentrations of immunoreactive parathyroid hormone have been reported (Joffe et al., 1972). However, in non-active cases of rickets these workers found no evidence of raised values. Such were noted in rachitic White infants by Arnaud et al. (1972). Recently, Galante et al. (1972) demonstrated a direct effect of parathyroid hormone on the metabolism of vitamin D, in which secretion of 1,25-DHCC by the kidney was depressed under the influence of the hormone. 'Clearly, such an effect would be most obvious when vitamin-D intake was marginal or inadequate.' Parathyroidectomy, in cases of severe bone disease in chronic renal failure, has been shown to be beneficial; the latter is believed to 'be due to removal of a block in vitamin-D metabolism' (Anon., 1972a).

Dietary strontium has recently been shown to cause a block in the renal synthesis of 1,25-DHCC from 25-HCC 'The exact mechanism whereby strontium alters the renal metabolism of 25-HCC is unknown.' Indirect evidence is that it may act through parathyroid hormone or calcitonin, or both (Omdahl and De Luca, 1973).

If leg abnormalities arise as a result of an active rachitic process, and are found to be associated with decreased circulating

25-HCC, or to a raised parathyroid hormone level (which would depress the synthesis of the active polar metabolite 25-HCC to 1,25-DHCC) or, to a combination of both these factors, it could be inferred that vitamin D deficiency was the causative factor. Further if this deficiency was associated with an increased metabolic demand for the vitamin, as occurs during active growth, it would further augment bone involvement.

Elucidation will only be forthcoming when detailed serial determinations and observations on relevant age and sex groups, over the critical growth spurt, can be undertaken.

Results, thus far obtained, have demonstrated that a primary vitamin D deficiency is most unlikely to be involved in the aetiology of adolescent leg abnormalities in the South African context. It therefore remains to be shown whether a secondary or metabolic deficiency of the vitamin is involved.

4.2. DIETARY STUDIES

Naturally occurring foods contain very little vitamin D (Williams, 1969; Stamp, 1973; Avioli and Haddad, 1973). The major sources occur in foods of animal origin, particularly in the liver oils, in the flesh of fatty fish (McLaughlin, 1973) and in their liver oils, which are the richest natural sources known. Fish liver oils contain the highest concentrations.

4.2.1. Intakes of Vitamin D

Transvaal. The dietary intake of vitamin D in children of 16 - 17 years was found to be low. Intakes were estimated, using the tables of Fox (1966) and Avioli and Haddad (1973). The estimated mean daily intakes of vitamin D in Transvaal groups were as follows; Bantu, 202 I.U., Coloureds, 210 I.U.; Indians, 159 I.U.; and Whites, 174 I.U. These values are subject to limitations which are inherent in dietary data obtained by questionnaire methods. However, they serve to indicate the pattern prevailing at the present time.

From the information supplied in the survey, it was apparent that children of 12 years ingested approximately two thirds the amount consumed by adults, and those of 7 years, about one half. The implication is that vitamin D intakes are even lower in younger children.

High and low fish-eaters near Cape Town. It was found that vitamin D intakes of 12 year old Coloured children living in the low fish-eating community at Elsies River, were higher than those of Coloured children residing in Johannesburg. The mean intake of 210 I.U./day for Johannesburg Coloured 16 - 17 year pupils would drop to approximately two thirds this value at 12 years, namely, 140 I.U./day. Both these intakes were thus lower than those obtained among the high fish-eating communities at Hout and Kalk Bays. Estimated mean daily intakes for Coloured children at 12 years were, Johannesburg, 140 I.U.; low fish-eaters, 224 I.U.; high fish-eaters, 311 I.U. The ranges of intake were wide.

All groups were thus consuming less than the mean daily amount recommended for children, namely, 400 I.U. (WHO Chronicle, 1972).

However, intakes of many of the high fish-eaters were more than the recommended requirement, with the upper quartile consuming a mean of 569 I.U./day; and the lower quartile 113 I.U./day. In the low fish-eating group, the upper quartile figure was 354 I.U., and for the lower quartile, 119 I.U. daily.

Infants. No attempt was made to estimate the vitamin D intake in infants. The majority of White and Indian babies were probably protected from deficiency as they received adequate daily amounts of vitamin D in proprietary milk foods. Only 5% White, and about 15% Indian babies were breast fed for longer than 6 months. Bottle feeding with infant formulae, known to be fortified with vitamin D, was given to about three quarters of White and about half the Indian babies; a small proportion were fed fresh cow's or condensed milk. In the case of Coloured infants, about one half were being breast fed for longer than 6 months; about one third received feeds made from infant formulae. More than three quarters of Bantu babies were breast fed for longer than 6 months; about one quarter of urban babies were given infant formulae, the rest were offered condensed milk or cow's milk feeds.

4.2.2. Data used for the Estimation of Vitamin D Intakes

As has already been described, the major dietary sources of vitamin D are foods of animal origin, with fish liver oils the richest natural source. In addition, eggs, margarine and fish are also stated to be 'rich sources' (Anon. 1973a). Palmisano (1973) considered that the only important ingested source is fish products. However, McLaughlin (1973) has pointed out that,

'It is the flesh of fatty fish only (herring, salmon, etc., identifiable by the pinky-brown colour) that contains this vitamin - white fish store it in the liver and this is not normally eaten. Shellfish have none.'

Fish caught in South African waters, around the Cape, include several varieties of fatty fish, such as yellow tail, harders (mullet), Cape salmon, mackerel, pilchards, etc. (Manning, 1973 - personal communication). Hake (stockfish) contains negligible amounts.

The following information was used in the estimation of vitamin D intakes. Fox (1966) has given some values for vitamin D concentrations.

Fish -	Stock	negligible
	Canned fish and pilchards	450 I.U./100 g
Beef		400 I.U./100 g

In Table XLVI data according to Avioli and Haddad (1973) on vitamin D concentrations in I.U./100 g (unless otherwise stated), in some common foods, are given.

Table XLVI: Vitamin D reckoned as I.U./100g (Unless otherwise stated)

(Table from Avioli and Haddad, 1973)

Food	I.U./100g
Butter	35
Cheese	12 - 15
Cream	50
Egg Yolks	25 I.U./average yolk
Halibut	44
Herring	
Fresh, raw	315
Canned	330
Liver	
Beef, raw	9 - 42
Calves, raw	0 - 15
Lamb, raw	17 - 20
Pork, raw	44 - 45
Chicken, raw	50 - 67
Mackerel	
Fresh, raw	1100
Milk	
Cows	0-3 - 4 I.U./100 ml.
Human	0 -10 I.U./100 ml.
Oysters	5 I.U./3-4 medium sized
Salmon	
Fresh, raw	154 - 550
Canned	220 - 440
Sardines	
Canned	1150 -1570
Shrimp	150

4.2.3 Comparative Studies

The low intakes of vitamin D in groups of South African children were similar to estimated daily intakes of 189 I.U. reported for Pakistani children living in Glascow (Ford et al., 1972b). Native Scottish children probably received less than 100 I.U. per day from all sources, excluding sunshine (Arneil, 1967; Richards et al., 1968). In New Delhi, Vaishnava and Rizvi (1971, 1973) reported mean intakes of vitamin D to be as low as 30 I.U./day in a group of females of 15 - 20 years who had late rickets or osteomalacia. In Britain, one study indicated that geriatric patients had a mean intake of only 64 I.U./day (Hodkinson et al., 1973). The total minimum requirement for normal adults, however, is stated to be about 70 I.U./day (Palmisano, 1973). Mean daily dietary intakes of vitamin D are low in most population groups, except those in U.S.A. where most commercial milks, baby foods, margarine and breakfast cereals are fortified, and 'the average American may receive several times the daily amount of vitamin D he requires' (Palmisano, 1973). The mean amount of vitamin D in serum in U.S.A. Americans, namely, 27,3 ng/ml (Preece et al.,1973) is reported to be greater than that of the British, namely 16,0 ng/ml (Stamp, 1973).

Reviewing the mode of action of vitamin D, Omdahl and DeLuca (1973) concluded that under normal circumstances 'vitamin D is not required in the diet since adequate ultraviolet irradiation of skin will produce sufficient vitamin D.' In the first direct quantification of the separate contributions of cutaneous and dietary ingestion of vitamin D in man, Haddad and Hahn (1973) showed that, although both ingested and exogenous sources contribute, the latter accounted for

the majority of circulating 25-HCC.

This revival of interest in exogenous sources of vitamin D recalls the early work of Hess and Unger (1922a), and Chick et al., (1923), and others, who amply demonstrated the importance of the curative properties of sunlight. The significance of dietary vitamin D in the prevention of rickets, although established during this period, did not appear to have been influential until the irradiation of dried milk introduced a new and effective method of prevention in susceptible populations.

In the South African context, although dietary intakes of vitamin D are low, there appeared to be adequate available solar radiation, with a mean range of 6 - 9 hours sunshine per diem (Weather Bureau, 1968). Thus, apart from known individual differences in the requirement of the vitamin, it is clear from the studies undertaken that neither dietary vitamin D nor ultraviolet irradiation had an obvious influence on the prevalence of leg abnormalities.

4.2.4. Prophylactic Measures and Public Health Aspects

In South Africa, as in other countries, public health measures for the control of vitamin D deficiency relies mainly on fortification of infant dried-milk formulae, such as S.M.A., Lactogen, Nutrine, etc. Most White and Indian babies are adequately protected by these foods, but fewer Coloured and still fewer Bantu babies.

From the enquiries made, members of the medical profession, and associated professions, e.g. clinic sisters, play a minor role in promoting active vitamin D prophylaxis. In the questionnaire survey,

based on mothers' replies, only 20% of mothers received advice on vitamin D supplementation from doctors, 30% from clinic sisters, and 5% from chemists. About half had received no advice. Just over a half of the mothers questioned had given their infants vitamin D containing syrups. It is doubtful, however, if the majority of mothers knew of the presence of vitamin D in these preparations, perhaps they simply believed that their babies needed 'vitamins'. From enquiries, it was apparent that there is a large proportion of mothers, who either do not know, are unaware, or do not understand the necessity for rickets prevention, nor how the disease may be prevented. Arneil (1964), dejectedly, expressed similar views, 'we have failed to teach mothers what this vitamin is and what the disease is, let alone how to prevent it.'

Concern in Cape Town over the prevalence of rickets in Coloured, Bantu and White infants led Robertson (1969) to introduce fortified dried milk powder for use in the area, so as to provide 400 I.U. vitamin D per reconstituted pint (i.e. approximately 702 I.U./litre). The rickets situation in Cape Town, according to a recent report (Snudgren, 1973 - personal communication) is believed to have improved.

In spite of this early protection of White and Indian infants, prevalences of bowing and knock knee were not obviously different between the four ethnic groups. Unknown to the author the same conclusion had been reached by Garti and Winter (1964) in Israel. (One of the authors, Dr. S. Winter, saw our recent preliminary report in the Lancet on prevalences of bowing and knock knee in South African schoolchildren (Richardson and Walker, 1973) and sent a copy of their

publication to the author). The summary of their paper is as follows:-

> '1000 schoolboys over the age of 10 were examined for the presence of permanent skeletal deformities of rachitic origin, 500 were Jewish boys from private secondary schools in Haifa, from families who took their infants to infant welfare centres and to physicians, and who give vitamin D to their infants. The other 500 were non-Jewish boys from 2 Druse villages and a Haifa Arab school, from families whose children had not received infant welfare services during infancy.
>
> No difference in the incidence of orthopedic deformities in the 2 groups was found. This casts doubt on the relative importance of prescribing vitamin D or the value of infant welfare services in the prevention of skeletal deformities in this country. It is probable that rickets in Israel rarely leaves permanent sequelae.'

Laxity of prophylactic measures resulting in a shortfall in vitamin D intakes prevail in other countries. Several investigations carried out in Scotland over 4 or 5 years led Richards et al. (1968) to report that the consensus of opinion was 'that a considerable number of Scottish infants are not receiving sufficient vitamin D.' In Greece, Lapatsanis et al. (1968) reported a similar situation, in which 65% of infants received no vitamin D, 'and in over 60 per cent of those the doctor was responsible for the omission.' In East Germany, the necessity for rickets prophylaxis was stressed by Harnapp (1969), because it was not insisted upon nor practised consistently.

Recently, Werner et al. (1972) proposed that irradiated liquid milk be introduced into East Germany, as had been the case since 1968 in Canada, where the vitamin D content was 400 I.U./quart, i.e. 354 I.U./litre (Barsky 1969). Recent studies in Morocco by Guignard et al. (1971) showed that only 40 - 50% of 34,800 infants under 2 years received regular vitamin D treatment. Very few children presenting at hospital with rickets in Melbourne, Australia, had had supplementary vitamin D (Mayne and McCredie, 1972). Holmes et al. (1973) asked, was the reason for sub-optimal intakes of vitamin D, 'the paediatricians' fear of producing intoxication in infants?'

4.3. PREVALENCE OF SKELETAL ABNORMALITIES: ANTHROPOMETRIC AND CLINICAL OBSERVATIONS

4.3.1. Abnormalities of the Lower Limbs

Prevalences of bowing and knock knee. As has previously been stressed, it must be borne in mind that percentage differences in readings in 100 consecutive subjects, for gap measurements of 2,5 cm or more were at knee 10,0%, and at ankle 7,5%. Differences were due almost exclusively in the positioning of legs prior to measurement. There were, in some instances, rather wide differences in prevalences within sub-groups of ethnic groups. Bearing these limitations in mind, it was clear that bowing and knock knee, as measured by gaps of 2,5 cm or greater, were present in large proportions of our South African schoolchild populations, ranging from 5 - 24% for bowing from 7 to 16 - 17 years, and 13 - 43% for knock knee, over the same period. Less than half of all schoolchildren had straight legs. As

already indicated, neither ethnic group, skin colour, nor region (urban, rural), nor greater exposure to ultraviolet radiation, appeared significantly influential in determining the presence or absence of bowing or knock knee. Sex differences were apparent, particularly in post-pubertal groups; boys had higher prevalences of bowing and girls had more knock knee. The latter was more common than bowing.

There was a rise with age in the frequency of bowing (2,5 cm or more) in all but Indian girls, who exhibited a fairly constant prevalence. In the same interval there was a fall in frequency of knock knee (2,5 cm or more) in boys, in all except rural Bantu, among whom the proportion remained constant. However, knock knee in girls remained fairly constant, with urban Bantu girls having the highest prevalence.

Similarly, minor degrees of bowing, although not considered in prevalence figures, clearly indicated a trend in the progression of leg abnormalities. More younger children had minor degrees of bowing than in post-pubertal groups. There was also a concurrent decrease in prevalence of minor degrees of knock knee and of straight legs, with age. The importance of this observation is seen in the progression of abnormalities. Briefly, from the cross sectional studies made these moulding changes would appear to take the following courses. (i) In a child with knock knee, or perhaps straight legs, there would be a gradual straightening of legs, followed by a lengthening and accompanied by a bending outward to become bowed. (ii) In a child with a minor degree of bowing a gradual progression to a more marked abnormality would occur. (iii) A child with an already

severe degree of abnormality would remain much the same or increase somewhat. (iv) Finally, where obesity was superimposed on the already knock kneed child, an increased abnormality would result.

Obviously, some factor or factors must be responsible for this age trend, which was not only evident in minor degrees of abnormality, but clearly so in severer degrees, particularly in the case of bowing.

Influencing Factors

Mean anthropometric measurements. As was expected, growth rates, reflected by mean body mass and height, were greater in Whites than in Non-Whites. At 16 - 17 years Indian girls were lightest and Bantu girls the heaviest, but the latter were also short, compared with Whites. Few differences were noted in other anthropometric measurements within each age-sex group, with the exception of data bearing on obesity, such as armgirth, triceps skinfold, and mid-thigh circumference. Girls after puberty were found to have higher values for these parameters than boys. Overweight and obesity increased in prevalence with rise in privilege, also with age, except in Indian girls and Bantu boys.

The role of obesity on prevalence of knock knee. An observation of importance concerned the role of body mass on the prevalence of knock knee. It was found that of the obese pupils as a whole, irrespective of ethnic group, a large proportion had knock knee. At least three-quarters of obese girls and one half of obese boys, by the age of 16 - 17 years, were affected, but in younger children proportions were lower.

This once again underlines the phenomenon of an age trend in the formation of leg abnormalities, in other words an increasing body mass puts greater stress on 'burdened' limb bones which bend under pressure. In the case of knock knee, knees press together and the distance apart at the feet becomes wider and wider. Anatomically this position must be assumed and maintained to counteract the increased mass and to keep the body balanced.

It was significant that of the 16 - 17 year old urban Bantu pupils, those with the greatest severity of knock knee, i.e. the widest gaps at ankles, were girls, almost all of whom were obese, or overweight. Their mean body mass was approximately 10 kg greater than that of girls with bowed or straight legs. Significantly too, urban Bantu girls, who had the highest prevalence of knock knee, also had the highest body mass index namely, mass/ht^2 (Montegriffo, 1971), i.e. these girls were heaviest for their height. In contrast, Indian girls, who were the lightest, with a low body mass index, showed little increase in knock knee prevalence with age. Moreover, Bantu boys, in whom obesity was almost non-existent, had lower prevalences of knock knee than the other ethnic groups. Girls too had greater mid-thigh circumferences, and thus heavier legs than boys. This phenomenon is again one of body mass and clearly bears on the higher prevalence of knock knee generally observed in girls.

Adolescent growth spurt. Growth curve data indicated the earlier maturation of White children compared with Non-White children, occurring at 10 - 13 years in White girls and 12 - 15 years in White boys, compared with Bantu pupils was shown by cortical thickness and cortical scores of

second (index) metacarpal bones, where Bantu, by 16 - 17 years had values of Whites at 14 years (Walker et al., 1973). Within this context girls showed the well known female ascendancy of growth at about 12 years, which again occurred a little earlier in Whites. However, once the pubertal growth spurt was past boys resumed their growth ascendancy and became taller and heavier than the girls.

Anthropometric data revealed the time of the pubertal growth spurt in the various ethnic groups.

The adolescent growth spurt is the second of two periods of active bone growth. The first occurs at 2 - 3 years and the second at 11 - 15 years. Sexual functions develop at this time. Tobias (1972a) views the growth spurt as part of the physiologically complex period accompanying puberty. Rickets is a disease of growing bone; it is thus conceivable that rachitic symptoms could become manifested during this later active growth phase, as is the case in the early years. It has been known for many years that rickets at puberty can result in bowing or knock knee (Ogston, 1888; Clutton, 1906); this has also been described recently (Ford et al., 1972b).

Although definite proof of rachitic involvement cannot be demonstrated by the studies thus far, it seems a likely possibility, particularly in view of the known increased metabolic demands of puberty.

Comparative Studies

Studies similar to those made by the writer and associates have not been made in other countries, but it is appropriate at this point

to briefly review prevalences of rickets as reported in schoolchildren, and also in infants.

Prevalence of rickets in schoolchildren. Although mention was made in the literature by early writers, e.g. Cheadle (1902), Cheadle and Poynton (1913), and many others, to both bowing and knock knee, no attempt appears to have been made to quantitatively measure the abnormalities in random schoolchild populations. The nearest approach is a recent investigation by Holmes et al. (1973). They classified bowing and knock knee by the distance of 2,5 cm apart at knee or ankle, respectively. Their subjects were 188 Asian immigrants living in Rochdale, Lancashire, and 78 White controls; children ranged in age from 3 - 19 years with very small numbers in each age group; adults were aged 20 - 67 years. Only limited information on bowing and knock knee was given, moreover numbers were too small to afford satisfactory comparative values. Briefly, of the 76 Asian children examined, 23 of whom were considered clinically abnormal, 1 had bowing and 14 knock knee giving approximate prevalences of 1,3% and 18,4%, respectively. Prevalences for bowing and knock knee, of 2,5 cm or more, in South African Indian pupils, 7 - 17 years, ranged from 6,1 - 19,4% for bowing and 12,3 - 28,3% for knock knee.

There have been reports that in Northern India and Pakistan vitamin D deficiency was common, particularly among women and young girls. Wilson (1931) found that 54% of girls from a city school had clinical evidence of rickets compared with 18% in suburban schoolgirls. In Murmansk, in Northern Russia, Nemzer and Belogorskii (1965) found 53,3% of schoolchildren aged 7 - 15 years with bone deformities (mainly

chest and head) stated to be caused by rickets, of which 26% were already healed, and 18% were recovering; 9% had florid active disease at 7 - 12 years and a further 9% at 12 - 15 years.

Some early investigations have provided overall prevalences of rickets in schoolchildren. Unfortunately, none refer specifically to abnormalities of the legs. These, otherwise, thorough and careful studies, will now be mentioned; they concern groups of schoolchildren who were examined for residual effects of rickets. In Melbourne, Australia, in the report of the Medical Officer of Health of the Victorian Education Department, 1911 - 1912, it was stated that 335 of 1327 boys (26%) showed deformities of chest and spine, knock knee, bow legs, square heads, beading of ribs, pot belly, enlarged liver and spleen. In New South Wales, Sutton (1920) found that 25% of 'ordinary schoolchildren' showed slight but definite signs of rickets. In London, Dick (1922) reported the presence of rachitic stigmata in about 80% of schoolchildren examined. Included in his criteria were 'stigmata of rickets left in the skeleton, teeth and elsewhere ... Knock knee and bowing outward of the legs are common ... .' In the Australian series the investigators stressed that only by careful examination was the condition observed, as cases were all mild and had thus probably escaped, not only the eye of the practitioner, but certainly the observation of the parent. This view was reiterated by Arneil (1964) who pointed out, 'The practitioners of the country must look for rickets, and they will find cases more often than they do now.' In a study in Scotland, he found that practitioners missed 36 out of 40 cases of severe rickets in White infants, 'although the bowing of legs and progressive impairment were accurately observed in most cases.'

If the active disease is being missed then Truswell (1973) is possibly correct in remarking,

> 'The iceberg analogy is presumably as applicable to vitamin D deficiency as it is to protein-calorie malnutrition or hypertension. For every florid case in hospital there are likely to be several mild-to-moderate cases in the community which are difficult to recognise but which have some increased risk of morbidity.'

Prevalence of rickets in infants and young children. Although the present study concerns rickets of adolescence it is pertinent to provide information on prevalences of rickets as found in infants and young children, studied mainly in hospitals or outpatient departments.

Prevalences of rickets in hospitals and outpatient departments; percentage admissions. The present situation in Johannesburg is stated by Professor J.D.L. Hansen, Professor of Pediatrics of the University of the Witwatersrand (personal communication) as being high. In White infants as many as 35% of admissions to pediatric wards show signs of rickets. Early reports of rickets in Cape Coloured infants (3 - 12 months) in Cape Town (Dancaster and Jackson, 1960), put the figure as more than 30% and possibly as high as 80% in an outpatient group. In Arab countries 40% of children in hospital admissions have been reported as having rickets (cited by Jelliffe, 1955). 35% among outpatients at Irwin Hospital, New Delhi (Ghosh et al., 1962) were likewise affected. In Ethiopian children, Teklaison and Sterky (1973) give a figure of 30% with rickets, in an outpatient clinic in Addis Ababa.

Number of cases admitted to hospital. Reports from hospitals in Johannesburg, South Africa (Joffe et al., 1972) indicated that 10 cases of nutritional rickets in Bantu children were seen in 6 months; and approximately 1 in 20 admissions of Whites to pediatric wards at the Boksburg-Benoni Hospital (Cartwright, 1973 - personal communication). in Glasgow, 10 cases were admitted in 1963, but the figure rose to 36 for the first 10 months of 1964. In Athens at the Aghia Sophia Children's Hospital, 15 - 20 cases are admitted per year (Lapatsanis, 1968). The figure is now 20 cases per year in Melbourne (Mayne and McCredie, 1972). On the other hand Kendall (1972) reported that only cases of vitamin D-resistant rickets occur at Harare Hospital, Salisbury; there were 8 cases seen in three years in African children.

Prevalence in population groups. In Beer-Sheba in the Negev, Costeff and Breslaw (1962) found rickets in 3% of infants examined in a child health centre. In Kaluga Oblast in the Far North, Frenkel et al. (1962) found 30% in rural and 20% in urban infants. In North Indian children Ghosh (1969) reported a prevalence of 2,7%. Richards et al. (1968) reported 9% of Scottish infants to be suffering from rickets. Robertson (1969) in Cape Town found prevalences of 8% in White, 17,3% in Bantu, and 17,2% in Cape Coloured infants, who were examined at immunization sessions, which are attended by over 90% of infants under one year. Lapatsanis et al. (1968) reported rickets in randomly selected infants of the general population, to be as common in a rural as in an urban community in Greece, namely 14,2% and 16,7%, respectively. In East German children, Harnapp (1969) reported 6 - 8% had florid rickets.

In the summary it is clear, from the various studies cited, that the consensus of opinion is that skeletal abnormalities, including those of bowing and knock knee, are rachitic in origin. In the present investigations it was assumed at the beginning that this was the case. However, thus far it has not been possible to incriminate any one of the factors considered likely to influence the development of rickets, namely, low dietary vitamin D intake, inadequate exposure to ultraviolet irradiation, skin pigmentation, and rural-urban location. In other words a primary vitamin D deficiency does not appear to be the causative factor.

The changing shape of legs, however, was clearly evident in the various ethnic groups. These changes have been shown to be associated with the growth spurt of adolescence. It is thus suggested that a secondary vitamin D deficiency may be responsible at least in part for the development of leg abnormalities during adolescence. This does not exclude the role of a physiological mechanism, nor some other, as yet, unknown factor.

4.3.2. Abnormalities of Chest and Spine

Chest Abnormalities

To regularize these studies, collaboration was obtained from a senior clinician, Dr. Paul Keen, now working in the Cancer Research Unit, South African Institute for Medical Research, Johannesburg. He has had many years experience, both in hospital practice, in Swaziland, also as senior surgeon at the Non-European Hospital in Johannesburg, and as Medical Officer of Health in Swaziland. He examined a group

of urban Bantu pupils of 16 - 17 years, namely, 66 boys and 57 girls, clinically for skeletal abnormalities of the chest. The same pupils were examined independantly, by the author (B.D.R.). The agreement in the results was satisfactory and the percentage data in most instances were closely similar. Table XLVII summarizes the two series of observations made.

Table XLVII : Prevalences of chest abnormalities in Bantu pupils of 16 - 17 years as assessed by two observers

Observer	Sex	Beading	Harrison's sulcus	Flaring of ribs	Pigeon breast	Funnel-shaped sternum	Flat & narrow chest
P.K.	Male	4,5	1,5	9,1	1,5	1,5	24,2
B.D.R.		0,0	3,0	12,2	1,5	1,5	38,3
P.K.	Female	0,0	0,0	0,0	0,0	7,0	3,5
B.D.R.		0,0	5,4	5,4	0,0	1,8	7,2

When pupils were divided into groups having bowing, knock knee,or straight legs, it was found that prevalences of pupils with chest abnormalities, such as Harrison's sulcus, pigeon breast, assymetry of chest, etc., were similar in each group. It was concluded, therefore, that the chest stigmata were sequelae which pertained presumably to infant rickets, and were neither related to, nor developed, during adolescence, nor bore a relationship to leg status.

Prevalences of these chest abnormalities could not be compared with those of other investigators as no corresponding data could be

found, although most articles on rickets mentioned the abnormalities cited as criteria. Pigeon breast was found in 0 - 1,5% of South African schoolchildren, and Harrison's sulcus in 0 - 5,5%. These abnormalities have been frequently mentioned by observers, but apart from their clinical description, no other information was given.

In cases of active rickets, concurrent severe upper respiratory conditions, such as pneumonia, bronchitis, and whooping cough, have been described as sometimes giving rise to a marked retraction of the xiphisternum, forming a funnel-shaped abnormality of the chest. This abnormality was seen in children of all ethnic groups and prevalences ranged from 0 - 12%. Prevalences of pneumonia in White, Coloured, Indian, and Bantu children were stated to have been 3 - 7%, and that of bronchitis, 9 - 15% with the exception of Bantu children, in which group more than half were stated to have had bronchitis (64%) during early life. Almost one third of children had had whooping cough. It must be borne in mind that these prevalences were derived from a questionnaire survey, and are based on mothers' remembrance.

In regard of pneumonia and rickets, in infants in the Arctic region of the Far North in Kaluga Oblast (Frenkel et al., 1962) the prevalence of pneumonia fell by half after a special public health drive for rickets prevention was instituted (vitamin D was given to all infants). This remarkable fall in pneumonia prevalence occurred concurrently with a fall in rickets prevalence. Such a definite improvement in the morbidity pattern is surely of sufficient importance to warrant further intensive research. In Israel, Costeff and Breslaw (1962) reported double the prevalence of hospital admissions for

respiratory and gastrointestinal conditions in children with rickets, compared with a control group. What these observations imply is not clear, but there appears to be an association between upper respiratory tract infections and rickets, and therefore, in a proportion of cases, in the prevalence of skeletal abnormalities resulting.

Exaggerated curvature of clavicles. An exaggeration of the normal upward curve of the outer third of the clavicles, giving a handle-bar effect, was very common, affecting about half to two thirds of Bantu, Coloured and Indian children, but was less in White children (about one quarter to one third). It was usually less common in girls than boys, and prevalences rose slightly with age. The reason for the condition was not apparent.

Winging of scapulae. Little attention has been paid to this abnormality in the literature, yet it was present in about two thirds of boys and one half of girls in all ethnic groups studied. Prevalences decreased with age.

Little relevant information on these two abnormalities could be found, apart from their reported association with rickets described by Findlay, (1918), Dick (1922), and Caffey (1961). In the case of winging of scapulae, the only other brief mention was made by Leary (1969), who used it as one of his criteria of rachitic sequelae in Bantu children, but he gave no prevalence figures.

Spinal curvatures

Information on spinal curvatures related to older pupils. Scoliosis occurred in approximately 10% of adolescent pupils.

Observations were visual. No attempt was made to distinguish between functional and non-functional scoliosis, by using a hanging bar and plumb line. Very few subjects had kyphosis; it was present in 5% of White pupils aged 16 - 17 years. Lordosis was more common in Bantu, particularly girls, where about half showed this abnormality. In other ethnic groups it occurred in less than 5% of pupils.

In discussing idiopathic scoliosis, Zorab et al. (1971) wrote, 'Its development is closely related to spinal growth, worsening most rapidly at times of rapid growth and seldom altering after bone maturity.' These authors found an increase in bone collagen turnover above normal throughout adolescence, indicating the rapidity of bone growth.

In a recent leading Article in the British Medical Journal, (Anon., 1973c) scoliosis of unknown aetiology, was said to be common, affecting 3,9 per 1000 White girls.

> 'What is it that can turn a healthy, vigorous, young girl of some 10 years of age into a crippled, deformed child with a scoliosis of more than 100° in less than five years? We do not know ... Despite much study, idiopathic scoliosis remains mysterious.'

Scoliosis, according to the above author, is known to be associated with many rare inherited syndromes, and spinal curvature may be a symptom of more than 50 diseases.

In early literature, Alexander Ogston (1888) discussing rickets, stated that, 'The most common deformities resulting from adolescent

rickets are knock knee, flat foot and lateral curvature of the spine ... scoliosis, from bad positioning and weak muscles was most numerous between 12 and 18. ... I am satisfied that (flat foot) is seldom due to anything other than rickets.' Arbuthnot Lane (1889) wrote of lateral curvature as arising from 'an habitual assumption of rest.' Dick (1922) regarded scoliosis as occurring more commonly in adolescent rickets, than in younger children with active disease. The scoliosis either advanced rapidly at puberty or tended to disappear. A high shoulder, or hip, or winged scapulae often drew attention to the condition. Dick did not, however, claim that scoliosis, knock knee or flat foot were necessarily of rachitic origin, but that the great majority occurred in connection with the rachitic state, as young adolescents do not assume faulty attitudes without reason,

> '... the position of rest is sought and unduly maintained because the child's muscle and ligaments have been weakened by present or pre-existing disease.'

As in the case of skeletal abnormalities of the legs, so also in abnormalities present in the chest, their origin has usually been considered to be rachitic. These latter abnormalities have been shown not to arise during adolescence, as prevalences do not alter over this period of active growth. It has been assumed, therefore, that such abnormalities are linked presumably with an early deficiency of vitamin D. However, without radiological and clinical studies on representative groups of infants (6 - 18 months) in the various racial groups in the general population, this possibility cannot be tested.

4.3.3. Abnormalities resulting from other Skeletal Involvement

Prevalences of dental abnormalities

Teeth and maxillae. Bantu pupils had a 3 - 4 times higher prevalence of teeth meeting together, on closure of the jaw, than Whites; Coloureds tended to the Bantu pattern and Indians to the White. In Bantu children the teeth were also less crowded, with a higher percentage exhibiting widely spaced teeth than in other groups. The question naturally arises, is this wide, roomy palate and even closure of teeth of ethnic origin, or are the contracted jaws and crowded teeth, so prevalent in other groups, due to an increasing manifestation of a disease process? - the answer is not known. From an aesthetic point of view the former is to be desired, and could be considered the 'normal' pattern, however, a degree of overbite of possibly 1mm, is now accepted as the desirable or normal pattern (Cleaton-Jones, 1973 - personal communication). There was too, a higher dental caries rate associated with crowding in the groups studied. Regarding dental caries, Retief et al. (1973), in groups of pupils at 16 - 17 years found that D.M.F. indices per mouth (decayed, missing, filled) were highest in Whites, 8 - 11; followed by Indians 6 - 8; Coloureds 5 - 6; and Bantu, 1 - 2. These indices follow the same pattern as that of crowding (both in upper and lower jaws), namely, 76% in Whites; 84% in Indians, 69% in Coloureds, and 38% in Bantu.

The most interesting and unexpected observation concerns the changing shape of jaws as reflected in 'bite' prevalences. Although at 16 - 17 years prevalences appeared to be similar to those found

at 7 years in Bantu, Coloureds, and Indians, prevalences increased in Whites. Percentages, however, at 12 years, were higher than those at 7 years. The prevalence of overbite increased while that of teeth meeting together, also prognathism, decreased. If a vitamin D deficient state is involved, the phenomenon may indicate a wider skeletal involvement during the pubertal growth spurt, than was thought likely or possible.

Closely associated with the maxillae and teeth are the adenoids and tonsils. In view of their supposed role in the formation of rachitic deformities of the bones of the face and jaws, it is interesting to note that Bantu children, with their wider, roomier palates, had approximately half the prevelence of enlarged adenoids than Whites, with their obviously more contracted jaws. Also, enquiries indicated that snoring, in Bantu, had only a third of the frequency in Whites. Although tonsilitis appeared equally prevalent in all groups, White and Indian children tended to mouth breathe more often than Bantu or Coloured children. A further interesting observation, related to the above, is that Bantu children usually had wider, more flaring nares than White or Indian children, who tended towards the more aqualine facies.

Rachitic deformities, particularly of the upper maxilla, have long been described as giving rise to the high palatine arch, and, in the lower maxilla, to the contracted jaw. There was a close association between the high arched palate and crowded and defective teeth. Marfan, in 1907 wrote, 'In 1896 I affirmed that the co-existence of adenoids and rickets is the most potent factor producing the

deformities of the superior maxilla, and today I am able to affirm that rickets is the true cause of the high vaulted palatine arch.' In 1922, Dick noted in passing that the person with the prominent forehead, short upper lip,slightly prominent teeth and receding chin was regarded as being somewhat distinguished, and yet he pointed out, these people had suffered from adenoids and a mild form of rickets. According to Dick, these characteristics resulted from the high prevalence of rickets which occurred over several centuries of history, moulding and changing the bones. He described how British skulls had changed from, 'the wide, roomy palates, and beautiful regular arches of teeth, ground down, but seldom decayed or missing' of the 16th and early 17th Centuries, to those of the 18th and beginning of the 19th Century where,

> 'jaws generally assumed the type of today, where teeth are markedly irregular and prone to decay, the upper set overlapping the lower, while the palate tends to be arched and high and the maxilla as a whole stunted in growth.'

This was reiterated by Caffey (1961); in hypovitaminosis D, he described the same condition being present in the maxillae.

> 'Both the upper and lower maxillas may be deformed when soft during active rickets; some of these deformities have dental significance and lead to malocclusion and malposition of the teeth. The upper maxilla and hard palate become narrow and the anterior segment of the upper jaw protrudes over the lower.'

Here again it is apparent that in the opinion of others there is a definite association between the type of bite, crowding of teeth, caries,

adenoids and tonsils, and rickets. Present investigations have shown the prevalence of these conditions, but their relationship to rickets remains to be clarified.

Flat foot (pes valgus)

In young children of 7 and 12 years the occurrence of flat foot was difficult to ascertain due to difficulties in positioning. A marked ethnic difference, however, was observed in pupils of 16 - 17 years. At this age over one third of Bantu were affected, but only 6 - 7% of Whites; Coloureds and Indians had intermediate proportions, 17 - 23%.

Flat foot has been very definitely and directly associated with rickets, particularly by early writers (Ogston, 1888; Dick, 1922). This condition, not commented upon in later literature, is one of the common deformities which bring children to medical care. Dick (1922) cited a series of flat foot in London children (from Whitman's Orthopaedic Surgery, London, 1919), in which he showed that children under 12 years had a prevalence of 9,8%; 10 - 15 years, of 19,9%, and 15 - 20 years, of 22,7%. These data are very similar to prevalences obtained in studies on Coloureds and Indians; they are somewhat higher than data on Whites, but lower than such on Bantu.

Leg pains (growing pains)

On questioning children, the prevalence of non-specific pains in the legs was found to range from 13 - 50%. It increased with age in both Indian and White pupils, particularly girls, except in White boys, in whom it decreased. Lowest prevalences were found in Bantu pupils, particularly rural groups. Leg pains were described as being

more often present in knees and thighs, less so in calves, than in other parts of the legs, and more often on sustained activity than at rest. Approximately two thirds to three quarters of pupils with leg abnormalities reported pain in the legs, compared with only one fifth to one quarter with straight legs. However, it must also be borne in mind that the subjective element, and differences in pain threshold, which vary from person to person and between ethnic groups, will influence the response to questioning, and thus prevalence figures.

Growing pains in children have recently been described as being caused by rickets (Ford and Dunnigan, 1972; Cooke et al., 1972). The latter authors have been impressed with the disappearance of these symptoms with vitamin D therapy, and have suggested that 'rickets should be considered in the differential diagnosis of "growing pains" not only in Asian but also in West Indian and White children.' In their contribution, Cooke et al. (1973) noted that the alleviation of non-specific symptoms, including pain, were sufficiently striking to be remarked upon by the teachers of the children.

However, in a Leading Article of the British Medical Journal (Anon., 1972b) growing pains were associated with an emotional disturbance, and not with any specific pathological condition. This view was also expressed by Hawksley (1972). Both publications referred to the work of Brenning (1960) who found, on questioning children, not their parents, that limb pains occurred in 12,5% of boys and 18,4% of girls. In an earlier survey, Brenning (1960), using a questionnaire, reported that 13,0% at 6 - 7 years, and 19,8% at 10 - 11 years, suffered from growing pains. In Danish schoolchildren (6 - 19 years) Øster (1972)

reports that 15,5% reported pains in the limbs. In a further contribution Ford and Dunnigan (1972) have reiterated their previous view on 'the dramatic amelioration of symptoms obtained by administering vitamin D and calcium supplements.'

In view of the association of pain with leg abnormalities, all that can be claimed is that in those pupils studied, pain appeared to be definitely linked with these abnormalities in a proportion of children. That it was of psychological origin seems unlikely, as the childrens' answers were spontaneous and not premeditated, and would in no way benefit them in regard of absence from school, sympathy, etc. Whether these non-specific leg pains should or should not be linked with adolescent rickets, must await further study. Controlled clinical trials giving vitamin D, also a placebo, to groups with and without leg pains, would help to clarify the situation.

4.4. RADIOLOGICAL STUDIES

4.4.1. X-rays of Wrists and Hands of Bantu Pupils of 16 - 17 Years

Examination of X-ray plates was undertaken by Dr. J.I. Levy, senior radiologist of the Non-European Hospital, Johannesburg. No active rachitic involvement was found in the hands and wrists of Bantu pupils, irrespective of present leg status. Little evidence of healed rachitic lesions, apart from shortening of the ulna, which is a possible manifestation of rickets, was found. A short ulna, as described in the radiological methods (section 2.2.4.) was present in 17,1% of boys and 14,3% of girls; 9,7% and 3,5% had grade 1 shortening ($\leq$ 2mm), 7,3%

and 7,1% grade 2 (2 - 4mm), and 0,0% and 3,5% grade 3 (≥ 4mm) in boys and girls, respectively. Numbers, however, were too small in the groups with and without leg abnormalities to draw any firm conclusions. Osteoporosis, based on a subjective assessment only, was present in 4,8% of boys and 16,1% of girls, and a more marked degree in 4,8% of boys and 7,1% of girls. Osteoporosis was considered to be present when there was diminished bone density and coarse trabecular striation of phalangeal, radial, ulna and carpal bones, graded as described in radiological methods (section 2.2.4.). The most marked degree included cortical thinning of the second (index) metacarpal.

Wayburne (1968) reported a loss of trabecular bone in South African children with kwashiorkor; but many of them had rickets. The precise interpretation was thus uncertain. Adams and Berridge (1969) found a 'paucity of cortical trabecular bone' in Central African (Baganda) children with kwashiorkor. These children, however, had no radiological or other features of rickets.

4.4.2. Cortical Thickness and Cortical Scores of Second (Index) Metacarpal

In groups of Bantu pupils with marked bowing or knock knee, or with straight legs, no differences were observed, in mean values for cortical thickness of second (index) metacarpal; data were 4,4, 4,6, 4,3 mm, respectively; nor cortical scores, 0,55, 0,57, 0,54, respectively. Thus, in spite of low intakes of calcium, known to obtain in South African Bantu populations, these low intakes appeared to be adequate and had

not prejudiced satisfactory bone formation (Walker et al., 1973).

Values for cortical thickness and cortical scores of 16 - 17 year pupils were higher than those reported for 14 year Bantu pupils, but similar to values on White pupils of 14 years (Walker et al., 1973). These Bantu pupils of 16 - 17 years, due to normal physiological growth and maturation processes, had naturally increased their bone size. A similar degree of maturation had, however, been reached by White pupils of 14 years. This is further evidence of the delay in maturation experienced by Bantu compared with White children. Mean values for cortical thickness and cortical score in girls of 16 - 17 years were the same as those obtained on Bantu mothers of large and small families (average age approximately 37 years). Values for cortical thickness and cortical scores were 4,4 mm and 0,58, respectively, for 16 - 17 year girls, and 4,4 mm and 0,57, respectively, for mothers (Walker et al., 1972). These girls had apparently reached adult values for the bone dimensions studied by 16 - 17 years.

4.5. SKIN PIGMENTATION DETERMINED BY REFLECTANCE PHOTOMETRY

No differences in skin colour could be detected on either the inner upper arm or on the foreheads of groups of Bantu girls and boys of 16 - 17 years, when comparing those with marked bowing or knock knee, with those having straight legs. The two sites were selected as representing areas of little exposure (inner upper arm) and maximal exposure (forehead) to solar radiation. Values, taken at inner upper arm at a wavelength of 685 nm (filter 609), for bowing, knock knee and straight legs, were 25,8, 26,9 and 26,2, respectively

for girls, and 27,4, 25,7 and 26,9, respectively, for boys. Values on the forehead were 27,0, 28,2 and 26,6, respectively, for girls and 23,2, 20,9 and 22,4, respectively, for boys at the same wavelength. Data revealed boys to be somewhat darker than girls. This is true for any population group (Tobias, 1972b).

The pupils studied had a deeper skin pigmentation than other Southern African populations studied by Weiner et al. (1964), namely, Yellow Bushmen, and the Hybrid population 'Rehoboths' of South West Africa; these females had values for inner upper arm, at 685 nm, of 38,8 and 44,9,respectively, and males 37,4 and 41,7, respectively. South African Bantu pupils, however, at 685 nm, had similar skin colour to that of Okavanga Bantu, who had values for upper arm of 23,3 for girls, and 20,0 for boys.

4.6. BIOCHEMICAL STUDIES

4.6.1. Serum Alkaline Phosphatase, Calcium, and Inorganic Phosphorus

In view of the extremely conflicting evidence on the significance of raised alkaline phosphatase values in detecting 'biochemical' rickets, and the recent controversy over the raised values in adolescence, being probably of physiological and not of pathological origin, it was decided not to study groups of younger children, with contrasting leg abnormalities, but to study those whose values had presumably already 'settled down', namely, pupils at 16 - 17 years. At this age the alkaline phosphatase "flare" described by Round (1973) should have subsided, at least in girls, although perhaps less so

in boys.

Values in Bantu pupils of 16 - 17 years. No significant differences were found in mean values of serum alkaline phosphatase, nor in serum calcium or inorganic phosphorus, in pupils with marked bowing or knock knee*, compared with those with straight legs.

Values in Coloured pupils of 12 years : high and low fish-eaters. Coloured pupils of 12 years in the Cape were studied in order to determine to what extent a higher dietary vitamin D intake of the high fish-eaters would affect biochemical values. When divided into groups according to dietary intake of vitamin D, there were no differences in mean serum calcium or in inorganic phosphorus values. Mean alkaline phosphatase value in high fish-eating pupils were similar to the low fish-eating pupils, namely in boys 24,7 and 27,4, and in girls 25,9 and 28,3 King Armstrong units, respectively. Coloured children in Johannesburg of 10 - 12 years, who were used to eating fish infrequently had values similar to those of the high fish-eaters. The Johannesburg Coloured children had much lower vitamin D intakes; such differences thus did not appear to affect the mean alkaline phosphatase values.

Alkaline phosphatase values in pupils of 7 and 10 - 12 years. Mean serum alkaline phosphatase values were obtained on groups of 7 and 10 - 12 year old pupils in the four ethnic groups. Whereas data were similar, Bantu pupils had somewhat higher mean values than other groups.

4.6.2. Comparison of Data

The ranges of mean values for alkaline phosphatase of 16 -17 year Bantu pupils, in King Armstrong units/100 ml, namely, 27,8 - 31,2

* Addendum. In a recent publication by K.A.V.R. Krishnamachari, and K. Krishnaswamy (Lancet, 1973, 2, 877) on 'Genu valgum and osteoporosis in an area of endemic fluorosis' it was reported that 'levels of serum calcium, phosporus, and alkaline-phosphate activity were normal.'

for boys, and 19,8 - 21,8 for girls, were higher than those reported by Round (1973) for White boys and girls, namely 11,8 at 16 years, 9,1 at 17 years, for boys; and 6,3 and 6,3 for 16 and 17 year old girls, respectively. West Indian boys had raised values (Cooke et al., 1973) similar to those of the South African groups. Table XLVIII gives comparative values for various biochemical parameters in groups of 16 - 17 year old pupils.

Table XLVIII appears on page 205.

4.6.3. Discussion of the Implication of Recent Studies in relation to 25-HCC Levels

Although mean values for serum alkaline phosphatase, calcium and inorganic phosphorus, were not significantly different in groups with severe bowing, knock knee, or straight legs, this does not rule out the possibility of an active rachitic process having occurred during the earlier years. The post-pubertal pupils studied had already passed the rapid growth phase. In order to throw light on the perplexing problem it would be necessary to use a specific test for vitamin D activity and to determine values in each age-sex group, particularly before and during the growth spurt of 8 - 13 years in girls, and 10 - 15 years in boys. Such a specific test has recently been described for use in human plasma; it is a competitive protein-binding assay for 25-hydroxycholecalciferol, (25-HCC). The method employs a chromatographic separation of the vitamin D metabolite, 25-HCC (the major circulating metabolite found in plasma), which is then assayed by a competitive protein-binding technique and counted in a Packard 3375 liquid scintillating counter (Belsey et al., 1971 ;

Haddad and Chyu, 1971; Hahn et al., 1972; Preece et al., 1973). Recently, Stamp and Round (1973) have described serum 25-HCC values as an accurate index of vitamin D nutritional status. As yet, limited studies have reported a mean normal value of 16,0 ng/ml for U.S.A. adults (Preece et al., 1973). The latter authors could detect no 25-HCC in Asian patients with rickets or osteomalacia, as values were below the sensitivity of the method. A group with raised alkaline phosphatase levels had a mean of 2,2 ng/ml, and those with normal alkaline phosphatase levels, 4,7 ng/ml; this was significantly lower than that of the White control group, namely, 12 ng/ml.

Definitive studies would greatly assist in answering the many uncertainties of the aetiology of leg abnormalities.

Table XLVIII: Mean serum alkaline phosphatase, calcium and inorganic phosphorus in comparative groups of 16-17 year old pupils (local and overseas studies)

Group	No.of subj.	Alkaline phosphatase K.A. units* Mean	S.D.	Serum calcium mg/100ml Mean	S.D.	Inorganic phosphate mg/100ml Mean	S.D.
		GIRLS					
S.A. Bantu:							
Straight legs	17	21,4	4,3	10,4	0,48	3,6	0,52
Bowing	15	21,8	4,4	10,3	0,49	3,6	0,45
Knock knee	17	19,8	3,0	10,3	0,55	3,7	0,20
Asian	29	14,9	12,7	9,8	0,67		
White	17	10,0	2,8	10,1	0,44		
W. Indian (Cooke et al., 1972)	9	12,6	2,3	9,9	0,23		
Whites: 16 yrs (Round, 1973)	22	6,3		9,6		3,6	
17 yrs	21	6,3		9,6		3,6	
		BOYS					
S.A. Bantu:							
Straight legs	17	27,8	5,4	10,9	0,39	3,6	0,81
Bowing	20	28,4	3,5	10,2	0,33	3,5	0,62
Knock Knee	20	31,2	3,0	10,5	0,59	4,3	1,09
Asian	44	26,1	21,0	9,6	0,86		
White	26	23,5	14,6	10,0	0,33		
W. Indian (Cooke et al., 1972)	16	32,6	19,5	9,9	0,34		
Whites: 16 yrs (Round, 1973)	23	11,8		9,6		3,88	
17 yrs	17	9,1		9,6		3,6	

4.7. QUESTIONNAIRE SURVEY ON DEVELOPMENT PATTERNS AND INFECTION RATES

4.7.1. Milestones of Development

Normal distributions. Bantu infants reached the various milestones of development, of sitting, standing and walking earlier than contemporary Coloured, Indian or White infants of the same age. Dentition in all occurred at much the same time, except for Indian infants, who teethed a month later on average. The foregoing confirms earlier observations by Geber and Dean (1957), who reported African infants to be more advanced in these respects than White infants of the same age.

Late starters. Of the small percentage who were late starters, there were approximately twice as many Indian as White infants. Although lateness in reaching the various milestones has been linked with rickets, it is highly unlikely that this would influence adolescent rickets. Indeed, from observations made, Indian girls of 16 - 17 years, have a lower percentage of bowed legs, than the other ethnic groups. However, data on Indian pupils of 7 and 12 years were similar to those on other groups at the same ages. Indian boys of 16 - 17 years showed no difference in prevalence of leg abnormalities compared with boys of other ethnic groups.

4.7.2. Prevalence of Upper Respiratory Tract Infections

The only upper respiratory tract infection in which there were outstanding differences in frequency in the ethnic groups was bronchitis, which was said to be exceedingly common in Bantu children. Whooping cough had occurred slightly more frequently in Bantu than in other groups of children; roughly one third had had it. One

result of severe upper respiratory tract infection is an embaressment of breathing, resulting in irregular, and often spasmodic muscular action. A result of this irregularity of muscle action on softened bone, as in rickets, can tend to an abnormal shaping of ribs and other chest bones. Resultant bony changes often heal in their new positions, thus giving rise to the various chest abnormalities described as sequelae of rickets, e.g. Harrison's sulcus, pigeon and funnel breast, etc. These abnormalities are noted somewhat more frequently in Bantu children, but occurred in all other groups. The implication is that they arise from early rachitic involvement; adolescents are not as prone to these infections, and bones of the chest are less likely to be affected. This may well be the case, as schoolchildren exhibiting these abnormalities, do so irrespective of their present leg status, suggesting that these abnormalities are not recent manifestations of the disease.

4.7.3. Prevalences of Limb Fractures

Fractures of both arms and legs were relatively uncommon. In all ethnic groups arms were fractured two to three times more frequently than legs. The implication is that vitamin D-resistant rickets is uncommon in the general population, as repeated fractures are characteristic of this form of vitamin D deficiency.

CHAPTER 5

CONCLUSIONS

5.1. SALIENT OBSERVATIONS

To obviate turning back, the salient observations already reached will be repeated before proceeding to the conclusions.

5.1.1. Dietary Studies

1. Dietary vitamin D intakes on average, were low in all groups studied.

2. Only Coloured children in Cape Town region, who habitually consumed fish daily, could be regarded as having satisfactory intakes.

3. In the Cape Town region, Coloured children who ate plenty of fish regularly, were better nourished than those who lived at Elsies River, where fish, although available cheaply, formed a smaller proportion of the regular diet compared with the preceding group.

4. Prophylaxis with vitamin D preparations was poor in all groups, including that of Whites.

5.1.2. Prevalences of Skeletal Abnormalities : Anthropometric and Clinical Observations

1. (i) Leg abnormalities, bowing and knock knee, were common in all groups of South African schoolchildren. From one tenth to one fifth had bowed legs, whereas about one third had knock knee. Less than half had straight legs.

(ii) Probably the most important finding was that neither ethnic group, degree of exposure to sunlight, level of dietary vitamin D, nor rural-urban location, appeared to markedly influence prevalences of bowing or knock knee.

(iii) Body mass affected the commonness of knock knee; in all groups there was a higher prevalence in obese subjects. Bowing was much less affected by obesity.

2. No clear cut correlation was apparent between leg abnormalities and chest abnormalities, such as Harrison's sulcus, found in about 0 - 5% of children, pigeon breast, 0 - 2% flaring of ribs, in about 10%, etc. These stigmata, when due to rachitic changes, are sequelae of involvement in early life and not of adolescence.

3. Changes in the pattern of leg abnormalities were associated with the pubertal growth spurt. Bowing increased with rise in age, namely, from a range of 4 - 11% at 7 years, to 11 - 26% at 16 - 17 years. The proportion of knock knee, present in about one third of all pupils, decreased with rise in age only in older boys; prevalences remained fairly constant in all groups of girls.

4. The period of active growth occurred earlier in White than in Non-White groups.

5. In spite of the wide nutritional differences in the four ethnic groups, the prevalences of leg abnormalities were similar in all groups.

5.1.3. Radiological Studies

X-ray studies of the hand and wrist of groups of urban Bantu pupils of 16 - 17 years revealed no evidence of active rachitic change.

5.1.4. Skin Pigmentation determined by Reflectance Photometry

Variations in skin colour could not be correlated with patterns of leg abnormalities.

5.1.5. Biochemical Studies

In the groups of pupils studied, i.e. Coloured children of 12 years, and a post-adolescent group of Bantu pupils of 16 - 17 years, no biochemical evidence of active rickets, as reflected by serum concentrations of alkaline phosphatase, calcium and inorganic phosphorus were apparent. In neither individuals, nor groups, did data bear a relationship to presence or absence of leg or chest abnormalities.

5.2. CONCLUSIONS : AETIOLOGY OF LEG AND CHEST ABNORMALITIES

1. In the literature leg and chest abnormalities are described as arising primarily from a deficiency of vitamin D. From the findings just enumerated, it is concluded that a primary deficiency of vitamin D is not responsible. It is postulated that a secondary deficiency of vitamin D is the most plausible explanation. This is suggested in view of the skeletal changes, particularly in relation to bowing, which occur during the phase of active bone growth, with its increased metabolic demands. Possibly there is a temporary block or inhibition within the complex synthesis of vitamin D.

2. A proportion of children did not exhibit abnormalities. While caution must be exercised, this evidence suggests that the changes are not of physiological origin. It cannot be entirely excluded, however,

that many of the changes lie within the normal physiological range, and that a small proportion of abnormalities are of pathological origin.

3. The foregoing suggestions, additionally do not exclude that other factor/s, as yet unknown, may be partly or even wholely responsible for the changes.

CHAPTER 6

SUGGESTIONS FOR FUTURE INVESTIGATIONS

6. SUGGESTIONS FOR FUTURE INVESTIGATIONS

It has been demonstrated that abnormalities of legs are very common in South African schoolchildren. Usually these abnormalities have been regarded as arising from rachitic involvement of bone, presumably implicating a deficiency of vitamin D in early years. The questions arise - to what extent are abnormalities due to (i) a vitamin D deficient state; (ii) variations within a normal physiological process, or (iii) an idiopathic process.

What is needed is as follows,

1) The first urgent necessity is a highly specific method to establish vitamin D status. The very recently improved chromatographic method has been described to determine 25-HCC levels in human plasma, using a competitive protein-binding technique. The method is specific and would give very valuable information. Unfortunately, it is costly and time consuming, which makes large scale epidemiological investigations difficult, indeed almost impossible. Determination of parathyroid hormone levels in random groups of subjects would provide further, although indirect evidence of vitamin D involvement.

2) A prospective study of the type described in the present investigations designed to cover the age period from birth to post-adolescence is highly desirable. Only in this way can any changes that may occur in the skeleton be carefully followed and investigated clinically, radiologically and biochemically. This type of study should, ideally, be on a long term basis, but valuable information could be gathered by cross sectional studies on representative

random groups over the age period stated.

3) A retrospective study employing questionnaires would yield valuable information although it would be based largely on mothers' remembrance. It is possible by this means to determine what has occurred in the past, not only as regards skeletal involvement, but prophylactic measures used.

CHAPTER 7

ACKNOWLEDGMENTS

I am deeply indebted to my promotor, Professor D. van Eeden (Department of Microbiology, Potchefstroom University for Christian Higher Education, Potchefstroom), and to my co-promotor, Dr. A.R.P. Walker (Medical Research Council Human Biochemistry Research Unit, South African Institute for Medical Research, Johannesburg) for their unfailing enthusiasm, leadership and valuable assistance in the preparation of the manuscript.

Grateful acknowledgment is made to the following persons for their co-operation and valued help during the study, also for services rendered.

1) Dr. P. Keen of the Cancer Research Unit, South African Institute for Medical Research, Johannesburg, for his assistance and guidance in clinical observations.

2) Dr. J.I. Levy, senior radiologist, Non-European Hospital, Johannesburg, for examining the X-ray plates.

3) Dr. W.M. Politzer, head of the Biochemistry Department, South African Institute for Medical Research, Johannesburg for processing sera on the autoanalyser for calcium and inorganic phosphorus.

4) Dr. T.H. Stewart, City Health Department, and members of his staff, especially Miss C. Broddle and Mrs. D. Vilakhazi, and her staff, of the Mafolo Tuberculosis Clinic, Soweto, for their assistance in taking the X-ray plates.

5) Dr. L. Been, State Health Department, Bellville, Cape Province, and members of his staff, especially Sisters M.S. Pillay, B.F. Fisher,

and C.J. Johannes for their assistance in the studies on Coloured children in the Cape Province.

6) Miss M. Wadvalla for her valuable assistance throughout the study in the field, the laboratory and in the processing of data.

7) Mrs. B.F. Walker and Miss L. Daya for measurements of bone dimensions and their assistance in many other ways; also Mrs. M.E. Egglestone and Mrs. C.M. Coetzer for their clerical assistance, and to those who helped record data on the many field excursions.

8) To the many Principals and their staff members, who so willingly and co-operatively assisted in studies on pupils in the four ethnic groups.

9) Mrs. L. Green, for typing the manuscript, also Mrs. R. Kruger for the typing of the tables, and to Miss E.J. Walker for preparation of the graphs.

10) Mr. E.M. Bottel, Managing Director of Pitco Ltd., for the use of the photocopying machine.

11) My husband, Mr. J.K. Richardson, for his support and valuable assistance throughout, and in the final preparation of my thesis.

12) Last, but not least, to all the many hundreds of pupils in the four ethnic groups, from Johannesburg, the many Transvaal rural areas, and those in the Cape Province, for participating so willingly in the study.

CHAPTER 8

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