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GROWTH AND MORTALITY IN CHILDREN IN AN AFRICAN VILLAGE

BY

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This communication describes the mortality and growth rates of children in a rural Gambian community. The observations were made in the village of Keneba, where since 1949 the staff of the M.R.C. Laboratories, Gambia, have investigated the health of Africans.

Keneba is an African settlement in the West Kiang District of the Protectorate of the Gambia and is typical of the villages situated near the banks of the Gambia river. About 700 inhabitants—Mandinkas—live in dwellings made of mud, grass, or krint (a local bamboo), and maintain themselves almost entirely by agriculture. The food crops—rice, millet (*Pennisetum* sp.), sorghum, maize, and findo (*Digitaria* sp.)—and the only cash crop —groundnuts—are grown throughout the wet months from late May to October. The men usually grow the dry-land crops, rice cultivation being regarded almost exclusively as a responsibility of the women. Cattle, the trypanosome-resistant N'dama, are kept; throughout the period discussed in this communication they totalled just over 100. They are practically never killed for food, and, although milk is welcomed by the villagers, yields are low and little is left for human consumption when the requirements of calves have been satisfied. Goats and sheep are kept, but again these contribute little to the village food supplies, the flesh being eaten only during feasts. Fish is abundant in the river and is consumed readily when available, but fishing is regarded as a low-caste occupation and adds little to the food supply.

Most infants are exclusively breast-fed for four to six months after birth. The earliest food supplements are paps, usually made of rice but sometimes of millet or sorghum, and to these fish and sauces of groundnuts and green leaves are gradually added. Boiled rice grains are also added to the diet, usually by the mother feeding the infant a very small quantity of her own portion during meals. When breast-feeding finally stops, usually between the 18th and 22nd months, the toddler is receiving a mixed diet of the adult pattern.

A baseline survey made in April–May, 1950 (McGregor and Smith, 1952), showed that malaria was hyperendemic, the crude parasite rate for all ages being 54.7%; that approximately 36% and 33% of villagers were carriers of microfilariae of *Wuchereria bancrofti* and *Anopheles perstans* respectively; that 2.5% of the population were suffering from trypanosomiasis; and that hookworm and ascaris infections abounded. Against this background of varied and heavy parasitization a recurrent shortage of the harvested cereals was at first noted. From mid-May until late July, when the early crops, maize, and digitaria were harvested, dwindling cereal stocks were reinforced by extensive collection of bush fruits, nuts, berries, and green leaves. Since 1950 the duration of this recurrent "hungry season" has been progressively reduced by gradual extension of rice cultivation, until in the agricultural year 1957–8 it was entirely eliminated, the cereal supplies lasting from the harvest of 1957 to that of 1958.

Measures Against Disease

Since 1950 research has involved certain operations against disease.

Trypanosomiasis.—All cases diagnosed during the initial survey in 1950 were treated, and since then individual cases were treated as they arose. Trypanosomiasis, therefore, has ceased to exist as an important disease in Keneba.

Insect Vectors.—From August, 1950, to May, 1958, all village houses were treated with residual insecticides throughout the period of high mosquito density, May–November. During the first four years benzene hexachloride was used, and thereafter dieldrin. It was found that the use of such insecticides alone did little to reduce the incidence of mosquito-borne diseases.

Malaria.—The use of residual insecticides coupled with the mass administration of antimalarial drugs at intervals of two to three months over the wet months of each year failed to eradicate malaria from Keneba. From August, 1950, to May, 1956, these measures succeeded only in reducing the incidence of parasitaemia from 54% to about 12% (McGregor, 1956). By May, 1957, when administration of antimalarial drugs had been discontinued for at least 12 months, the crude parasite index had risen to approximately 40% of the total population. More intensive measures were then adopted. From May, 1957, to April, 1958, pyrimethamine was administered fortnightly to all inhabitants, and this achieved virtual eradication of malaria, the crude parasite rate in April, 1958, being less than 1%. After April, 1958, antimalarial measures were discontinued in all save some 80 subjects, and the crude parasite rate rose in April, 1959, to 34.5% and in April, 1960, to 37.3%.

Bancroftian Filariasis.—In 1951 36% of inhabitants were found to be carriers of microfilariae of

W. bancrofti, most of these being treated with diethylcarbamazine (McGregor, Hawking, and Smith, 1952). Four years later a cure rate of 74% and a reduction in microfilarial density of 89% were observed (McGregor and Gilles, 1956). A further small treatment campaign was conducted in 1955, and the individuals treated were four years later noted to show a cure rate of 89% and a reduction in microfilarial density of 98% (McGregor and Gilles, 1960a). These measures are believed to have greatly reduced the incidence of bancroftian filariasis in Keneba but are thought unlikely to have affected the health of the children reviewed in this communication, because this disease is relatively uncommon in young children.

Hookworm Infestation.—Two large-scale attempts were made in May, 1951, and in May, 1952, to control this disease. The attempts appear to have had almost no effect on the incidence and severity of the condition in the village.

Treatment of Serious Disease.—While the investigations were in progress it was necessary to treat gravely ill patients who asked for help. Some of the children in the present study must have received treatment for disease from which they might otherwise have died. Unfortunately it is not possible from the records to identify these children or to assess the nature and frequency of treatment given. However, the availability of treatment depended on the presence or absence of a doctor at Keneba, and on this basis it may be assumed that seriously ill children could have been effectively treated during only about 30% of the 10-year period of study.

By employing a reliable villager literate in Arabic as registrar the dates of births and deaths were reported precisely, and thus records of the true ages of the young child population were gradually built up. The mortality statistics reported in this paper were derived from these records. Growth rates were measured at annual examinations of all available village children in the months of April or May from 1950 to 1960 inclusive.

During the five-year period 1949–53 there were 195 live births. After eliminating two sets of twins and four single births (the latter children left the village at an early age and no more information about them is available) a group of 187 infants remained. Each member of this group, which represents a nearly complete cohort of Keneba births, has been followed up until death occurred or, with survivors, until the seventh year of life.

Mortality

Of the 187 children born alive, 81 (43%) died before reaching 7 years of age. The neonatal death rate was 54 and the infant mortality rate 134 per 1,000 live births. Mortality remained high during the first four years of life, then fell abruptly in the fifth year and remained low thereafter.

The sex ratio at birth was 1.115. With the numbers involved, this cannot be regarded as signifying an unusually high proportion of male births. Death rates were consistently higher in males than in females at all ages except in the sixth year, when there was only one death, that of a female.

Table I gives the age-specific mortality rates for six-month periods from 3 months to $6\frac{3}{4}$ years of age. There were 12 deaths during the first 3 months, leaving 175 survivors aged 3 months. Table I is constructed in the form of an incomplete life-table, death rates (q_x) at age x being calculated from the numbers of live children entering each age period (l_x) and dying during that period (d_x). It is clear that a peak of mortality occurs between 9 and 14 months of age; the death rate then falls sharply, rises slowly to a secondary peak at age $3\frac{1}{4}$–$3\frac{3}{4}$ years, then falls rapidly to the lowest rates obtained, those after $4\frac{1}{4}$ years of age.

TABLE I.—*Mortality in the First Seven Years of Life*

Age (x)	Number (l_x)	Deaths (d_x)	Death Rate/ 1,000 6 Months (q_x)
3–8 months	175	4	23
9 months–$1\frac{1}{4}$ years	171	20	117
$1\frac{1}{4}$– years	151	5	33
$1\frac{3}{4}$– ,,	146	6	41
$2\frac{1}{4}$– ,,	140	6	43
$2\frac{3}{4}$– ,,	134	8	60
$3\frac{1}{4}$– ,,	126	9	71
$3\frac{3}{4}$– ,,	117	6	51
$4\frac{1}{4}$– ,,	111	1	9
$4\frac{3}{4}$– ,,	110	1	9
$5\frac{1}{4}$– ,,	109	—	—
$5\frac{3}{4}$– ,,	109	1	9
$6\frac{1}{4}$–$6\frac{3}{4}$,,	108	2	19

Table II gives the distribution of births and deaths (both sexes) by month. Nearly two-thirds of the deaths occurred during the wet months, July to October, and 26% occurred in August alone. The slight peak of births during August and September does not explain the high concentration of deaths in the wet season; and the peak of deaths during July–October is just as marked when deaths during the first nine months of life are excluded.

TABLE II.—*Distribution of Births and Deaths by Month*

Month	Births		Deaths	
	No.	%	No.	%
January	14	7·5	3	3·7
February	12	6·4	3	3·7
March	12	6·4	3	3·7
April	13	7·0	1	1·2
May	15	8·0	7	8·6
June	16	8·6	3	3·7
July	16	8·6	11	13·6
August	22	11·8	21	25·9
September	28	15·0	8	9·9
October	16	8·6	11	13·6
November	15	8·0	7	8·6
December	8	4·3	3	3·7
Total	187	100	81	100

An attempt has been made to link age-specific mortality with month of birth. The births of 45 children who died aged 15 months or older were scattered fairly evenly over all months in the year; it does not appear, therefore, that season of birth has much influence on the chances of death at the later ages. Of 16 children dying within 9 months of birth, five were born in January and five in July, the remaining births being scattered throughout other months. No factor common to the January series was found; they were born in different years and died at different ages. The July series (five deaths under 9 months of age) included four neonatal deaths in July, 1951. Our records do not suggest any explanation.

Of 20 children who died aged 9–14 months, 16 were born when the rains were well established in late July to October. Seven of these 16 deaths took place during August in the following year, when the children concerned were aged 11–13 months.

Table III shows the chances of dying at ages 9–14 months and at ages 15 months to 7 years, children born in January–June being compared with those born in

TABLE III.—*Month of Birth and Chances of Death at Age 9–14 Months and at Age 15 Months–7 Years*

Month of Birth:	January–June	July–December
No. of children alive at 9 months	74	97
Deaths 9–14 months	5	15
Death rate 1,000 6 months ..	68	155
No. of children alive at 15 months	69	82
Deaths 15 months–7 years ..	19	26
Death rate 1,000 6 months ..	24	28

July–December. Children born in the second half of the year—broadly speaking, the rainy season—are more likely to die when aged 9–14 months than those born in the first half of the year. The second part of Table III shows that if this dangerous age is survived the season of birth makes little difference to the subsequent chances of dying.

Growth

Table IV shows the average heights and weights of all children surveyed. Children who died during the course of the survey are included with the survivors for reasons discussed below. Increases of height and weight are

TABLE IV.—*Average Heights and Weights of Keneba Children. Numbers are in Parentheses After the Means*

Age (Yrs.)	Mean Age (Yrs.)	Males				Females			
		Weight (lb.)		Height (in.)		Weight (lb.)		Height (in.)	
		Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
<½	0·3	11·9 (15)	3·0	24·8 (6)	—	11·9 (18)	3·2	—	—
½–	0·7	15·9 (55)	2·8	26·4 (28)	2·2	14·8 (44)	2·4	25·7 (20)	1·8
1–	1·2	18·7 (17)	2·8	28·3 (11)	1·4	17·3 (27)	2·7	27·7 (19)	2·0
1½–	1·7	20·2 (42)	2·4	29·8 (33)	2·2	18·7 (28)	2·1	28·9 (21)	2·0
2–	2·2	22·0 (17)	2·7	30·8 (17)	1·8	21·1 (19)	2·6	30·9 (19)	1·8
2½–	2·7	24·5 (33)	3·0	33·0 (33)	1·8	23·0 (28)	2·5	32·2 (28)	1·9
3–	3·2	26·5 (23)	2·9	34·5 (23)	1·9	27·3 (15)	2·6	34·5 (15)	1·5
3½–	3·7	28·8 (27)	3·1	36·0 (28)	1·4	27·6 (27)	2·5	35·8 (27)	1·4
4–	4·2	31·2 (22)	3·5	37·2 (22)	1·2	30·1 (17)	3·5	36·8 (17)	1·8
4½–	4·7	32·1 (25)	3·5	38·7 (25)	1·8	32·0 (29)	3·0	38·6 (29)	1·7
5–	5·2	35·4 (21)	3·3	40·2 (21)	1·0	34·9 (16)	3·4	40·2 (16)	1·8
5½–	5·7	35·1 (24)	3·5	41·6 (24)	1·5	35·8 (17)	3·5	42·0 (17)	1·5
6–	6·2	40·1 (17)	3·4	43·3 (17)	1·1	38·1 (18)	4·7	42·4 (18)	2·0
6½–7	6·7	39·7 (26)	3·8	44·1 (26)	1·3	38·6 (25)	3·6	44·1 (25)	1·8

fairly regular despite rather small numbers in some age-groups, the annual gain being about 4 lb. (1,814 g.) and 3 in. (7.5 cm.). Boys were slightly heavier and taller than girls.

Some statistical properties of the data in Table IV should be noted. A few children were not seen in early life, and some babies and toddlers who were seen could not be measured for height, but weights were nearly always measured. The measurements were made in April or May each year, so that individual children usually appear in alternate six-month age intervals in the Table. The data are not cross-sectional, nor are they strictly longitudinal, but may be regarded as predominantly longitudinal, especially when alternate age-groups are considered. In consequence, the errors in annual weight and height increments deduced from the Table are certainly smaller than would be obtained in a cross-sectional survey of a similar population. A more sophisticated treatment of the growth data, on the lines suggested by Tanner (1951), does not appear to be justifiable.

Figs. 1 and 2 show, for weight and for height respectively, the average growth curves of Keneba "survivors" (children alive at 7 years of age) and "non-survivors," together with those of British children. After infancy the African children were shorter and lighter than the British children, but from about 1½ years of age the annual increments were similar, so that the growth curves are parallel. It is also clear that the average growth curve of the African children who died was close to that of the children who survived, at least after the first year. Survivors aged 8 to 10 months (an age-range in which reasonable numbers are available) had a mean weight of 15.9 lb. (7,212 g.) and a height of 27.1 in. (68.8 cm.), as compared with 14.8 lb. (6,713 g.) and 25.9 in. (65.8 cm.) for children of similar age who died subsequently. The height difference may not be reliable, since it is difficult to measure the length of babies accurately without special equipment, and the differences are not statistically significant (there were 31 "survivors" and 22 "non-survivors" in this age range). But since 11 of the 22 "non-survivors" died before the next survey the differences may indicate some faltering in growth.

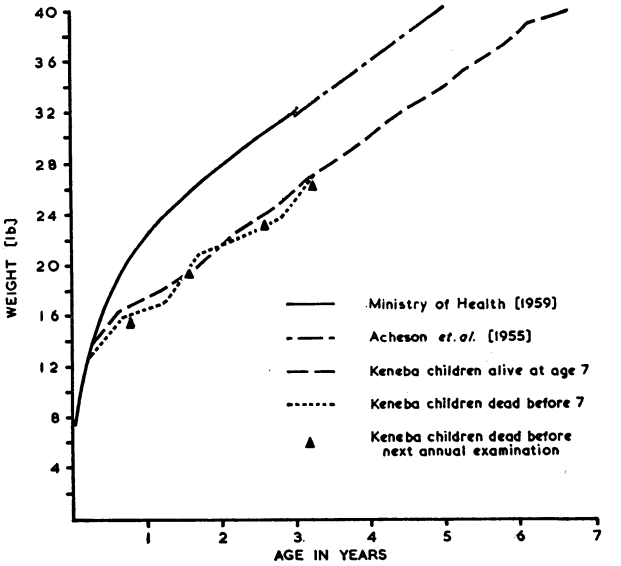

FIG. 1.—Average weight curves of Keneba children alive at 7 years of age, and of those who died before 7 years, compared with British averages for children up to 3 years of age (Ministry of Health, 1959) and over 3 years of age (Acheson *et al.*, 1955).

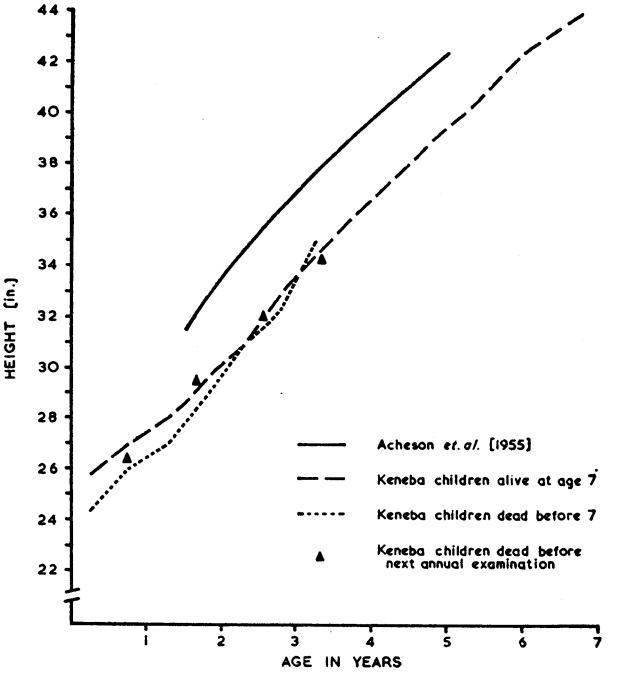

FIG. 2.—Average height curves of Keneba children alive at 7 years of age, and of those who died before 7 years, compared with a British average (Acheson *et al.*, 1955).

The suggestion that growth falters in the second half of the first year of life is supported by Fig. 3. This gives the results of a special survey, in May, 1960, of 106 infants under 1 year of age in Keneba and four near-by villages. The weights are plotted by age and compared with an average growth curve of British babies. During the first few months of life the Gambian babies are as heavy as their British counterparts, but during the rest of the first year do not grow as rapidly. The picture is similar for males and for females separately.

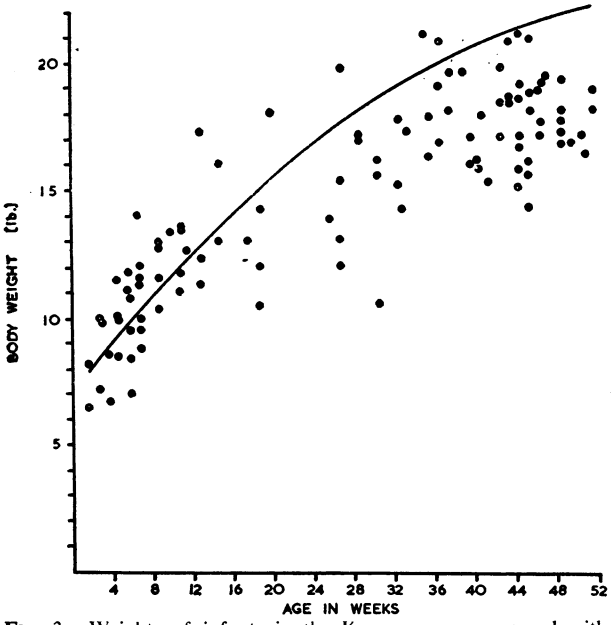

FIG. 3.—Weights of infants in the Keneba area compared with an average weight curve for British infants (Ministry of Health, 1959).

Discussion

The prodigious wastage of life in young children in Keneba—nearly 40% of those in our sample died within four years of birth—occurred during a period when attempts were being made to reduce the incidence of certain diseases. Mortality rates as high or higher occur throughout most parts of rural Africa.

Also, the pattern of growth closely resembles that reported from other parts of Africa (Nicol, 1949 ; Bruce-Chwatt, 1952 ; Welbourn, 1955 ; McLaren, 1960 ; and many others). Growth rates during the first few months of life are comparable with European standards. Then growth falters, and between about 6 and 18 months of age is well below European standards. The leeway is not made up, so that the Gambian children aged 1½ to 7 years of age are lighter and shorter, but have annual increments of weight and height similar to those of British children.*

High mortality and impaired growth in African children are usually explained as being due to malnutrition, disease, and low standards of child care. In the recent literature the importance of malnutrition has been much emphasized ; thus, for example, Wills and Waterlow (1958) have suggested that the death rate at ages 1–4 years might be used as an index of the nutritional state of a country. We were, however, interested to see that, in a recent report from Uganda, McFie (1960) concluded that malaria appeared to be more important than malnutrition as a cause of mortality in children.

*It is widely believed that complete " recovery " is possible after a temporary retardation of growth. This is probably true in individual instances, but it is not known if the average growth curve of a group would show equally complete recovery. Temporary retardation of growth in young rats appears to cause permanent stunting (Schultze, 1955).

Malnutrition

The faltering of growth at about 1 year of age and the accompanying high mortality are usually ascribed to the replacement of breast milk by foods of low protein content. The literature on kwashiorkor, the syndrome associated with protein deficiency, is now very extensive, but little is known about the incidence and epidemiology of this condition.

In our opinion, primary malnutrition is not likely to be an important cause of high mortality rates and defects of growth in Gambian children. The kwashiorkor syndrome is rare in the area. The breast-fed child is not weaned on to a solitary staple of poor nutritive value, such as sweet potato, plantain, or cassava, but receives paps of rice, millet, or sorghum, which are soon reinforced by ground-nut soups and sauces. There is no reason to think that a diet of breast milk plus such supplements is notably deficient in protein or in any essential amino-acid. The diet during and for a time after weaning may perhaps be insufficient in quantity and difficult for infants and toddlers to digest, but of this there is little evidence at present. The data summarized in Figs. 1 and 2 suggest that babies who died were slightly shorter and lighter than those who survived ; but the differences could easily have occurred by chance, and it is certain that the deaths did not occur selectively among babies who had been notably underweight for a prolonged period.

The diet given after weaning is adequate to support increments of growth as large as those found in British children. In the absence of any information on the growth potential of Gambian children, it is impossible to say whether the fact that they remain smaller than their British counterparts is to be attributed to genetic or nutritional influences, or to both. Though Keneba adults are shorter and lighter than Europeans (McGregor and Smith, 1952), most of them give the impression of being well developed and strong, and they are certainly capable of sustained hard work. The adult diet is sufficient for a high standard of lactation, which is strong evidence in favour of its adequacy in both quality and quantity.

In general the presence of vitamin-deficiency diseases is by no means obvious. Mucosal lesions compatible with lack of riboflavin are, however, common in some years. They tend to appear in the period January–April, and, while children do not escape unscathed, are most noticeable in adolescents and adults. Some individuals may be undernourished or malnourished, or both ; but the general picture suggests that severe malnutrition, when it is present, is more likely to be the consequence than the cause of illness.

Infection

Despite the operations against disease which were detailed above, the pattern of infection in Keneba remains remarkable for its variety and intensity, and communicable diseases are much more often manifest in young children than in older children and adults. Thus the clinical manifestations of malaria, ascariasis, whooping-cough, measles, gastroenteritis, and active trachoma are often seen in the very young but seldom

at later ages.* We do not know how many other diseases follow a similar pattern. Sera from adults living in a village about 8 miles (12.9 km.) from Keneba were found to contain antibodies to the following viruses in the following incidences: poliomyelitis, types 1, 2, and 3, 100% each; West Nile, 100%; yellow fever, 96%; Trinidad dengue, 86%; Zika, 80%; Bunyamwera, 65%; Semliki forest, 61%; Uganda S., 55% (McGregor and Porterfield, in preparation). Although clinical diphtheria has not been noted in the area, diphtheria antitoxin was found in high titre in all sera (Barr and McGregor, in preparation).

Thus, clinical experience and laboratory tests point to the intense transmission of a wide variety of infectious and parasitic diseases. The child is likely to encounter infection early in life, and, if he survives, to show a high level of immunity. It may be that patterns of disease and of immunity are different in other parts of Africa, but the similarity of mortality and growth patterns to those of the Gambia is striking.

Newborn infants, older children, and adults in the Gambia have serum gamma-globulin levels well above those of Europeans. From birth the level falls to its lowest point between 3 and 6 months of age, and then rises slowly until adult levels are attained about the fourth year (Gilles and McGregor, 1959; McGregor and Gilles, 1960b). This suggests that a high level of passive immunity is transmitted from mother to baby, and that at about 6 months of age the decline of passive immunity is overtaken by a slowly rising active immunity, which will presumably be "boosted" by repeated infection.

The pattern of malaria in the Gambia certainly supports a theory of changing levels of immunity. The African born in hyperendemic malarious areas shows a remarkable resistance during the first few months of life (Garnham, 1949). Primary attacks are mild and sometimes asymptomatic. Subsequently the severity of attacks appears to depend on age, peak severity being at about 16–18 months (Foord and McGregor, in preparation). Towards the end of the second year considerable immunity seems to have been acquired, and malarial attacks in the third year become milder though moderately dense parasitaemia may persist (McGregor *et al.*, 1956).

Though transmission of diseases of high endemicity probably continues throughout the year in the Gambia, there is a marked seasonal trend, especially for diseases transmitted by insect vectors. Thus in malaria the frequency of primary attacks is much higher in the wet months, when mosquito density is high. The high mortality of children in the wet months (Table II) corresponds to the peak period of insect-borne disease.

The relationship of the state of immunity to the time of maximal infection may be important in determining whether the individual succumbs. The most advantageous circumstance would be when repeated infections take place under a gradually diminishing "umbrella" of inherited immunity, so that active immunity is acquired smoothly without the occurrence of severe illness. Such conditions may pertain for infants born in the first half of the year; they meet the hazards of their first rainy season before passive immunity is lost, and are well protected by active immunity in their second wet season. By contrast, babies born in the second half of the year will mostly meet severe infections shortly after birth and will be able to deal with these by virtue of a high level of passive immunity. But this passive immunity is being lost during the ensuing dry months and they are not being repeatedly reinfected, so that they meet their second wet season in a vulnerable state. The mortality rates in Table III are consistent with this explanation.

Special reference has been made to malaria because this disease has been most intensively studied. Malaria is probably an important cause of mortality in Keneba babies and toddlers, but it is by no means the only one. Respiratory and alimentary infections are common, and there are occasional epidemics of measles and whooping-cough. The rising trends of mortality between the end of the second year and the end of the fourth year, if not a chance fluctuation due to small numbers, may represent a secondary pattern of infection by, and acquired immunity to, a group of diseases different from that responsible for the peak of mortality around the end of the first year. At present, however, we have no suggestion to offer. Records of causes of mortality do not exist and would be difficult to establish. It may even be difficult to arrive at a diagnosis of illness, since the causes are multiple. These children are exposed to a bewildering variety of infections from protozoa, helminths, bacteria, and viruses, the onslaught of which must represent an enormous obstacle to survival. Primary malnutrition or undernutrition may help to lower resistance, but the amount of resistance offered to infection is more striking than the susceptibility. Children often withstand simultaneously a number of infections, any one of which, untreated, might reasonably be expected to kill a European child.

Care of Infants and Children

The amount of care afforded by the mother almost certainly influences growth and mortality in young children. The adult female in Keneba is expected not only to bear and to rear children but also to produce a main food crop, rice, almost unaided. It is customary, indeed essential, if the food supplies of the family are to be safeguarded, for these women to continue their strenuous agricultural activities during the greater part of pregnancy and to recommence them with minimum delay after delivery.

In the early months of life the infant accompanies the mother whenever she leaves to attend her crops, but the toddler is often left in the village in the care of someone either too old or too young to work. Such practices must be expected sometimes to affect young children adversely, but at present little information exists which permits an assessment of their importance. Again, the effect upon the child of incapacitation of the mother either by accident or illness is not clear although it is probably considerable. Ignorance is also important. Keneba parents often seem unaware of the presence of serious illness in their children, and are surprised when it is pointed out to them. Perhaps this lack of awareness represents acceptance of illness in the young child as a natural and expected phase of development. The population knows nothing about the causes of most diseases, or of the ways in which infections are spread. Superstition is strong and money may be expended to purchase a charm for an ailing child, but little is done to provide adequate nursing or special feeding. An ailing child is seldom encouraged to eat or provided with nourishing titbits, so that illness may be the cause rather than the result of inanition and malnutrition.

*Dangerous epidemics arise occasionally. For example, while this paper was in preparation an outbreak of measles killed about 18% of children under 5 years of age in Keneba. The mortality in children aged 5 to 10 years was less than 2%.

Conclusions

For the reasons given, we believe the main causes of high mortality and faltering growth in young children in the Gambia to be infection and poor care. Deficiency of specific nutrients may contribute, but in our opinion malnutrition is more often secondary to illness than primary. The possibility that some children are underfed during the process of weaning cannot be excluded, but the nutritional and clinical pattern described as characteristic of kwashiorkor is rare.

These results and conclusions probably apply to large areas of rural Africa, especially West Africa. They may not apply to areas where the staple food is of notably low nutritive value, nor to urban areas where the ecology is entirely different.

Summary

The births and deaths of children in a rural Gambian village were registered as they occurred, and their heights and weights were measured annually.

Of 187 children born during a five-year period, 43% died before reaching 7 years of age. Death rates were high during the first 4 years of life, and relatively low from 4 to 7 years of age. The peak of mortality occurred around the age of 1 year and during the rainy season.

During the first 6 months of life, when breast milk is the main or only source of food for babies, growth was comparable with the British average. Then growth appeared to falter for a few months, around the period of peak mortality; but there was no evidence that children who died, compared with those who survived, had been notably underweight. From about 1½ to 7 years of age, heights and weights were less than those of British children, but the annual increments were similar.

It is considered that infectious disease and inadequate maternal care are probably the major factors contributing to high mortality and impaired growth. Malnutrition, while by no means absent, seems to be more often secondary to disease than primary.

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GUNSHOT WOUNDS OF THE HEART WITH EMBOLISM

REMOVAL OF MISSILES FROM CARDIAC CHAMBERS WITH AID OF EXTRACORPOREAL CIRCULATION

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This is the story of a boy who was shot in the chest with a shotgun. A mass of pellets entered the heart and then dispersed, causing embolism into the greater and lesser circulations. The remaining loose pellets were removed from the cavities of the heart by open operation with the aid of extracorporeal circulation. We have found no published account of any similar course of events even in the masterly and encyclopaedic paper on foreign bodies in the cardiovascular system written by Barrett (1950).

Case Report

A boy aged 12 was shot from a distance of about 35 yards (32 metres) with a 410 shotgun on May 15, 1961. He collapsed and was taken to Black Notley Hospital in Essex, and admitted under the care of Mr. Ronald Reid. There was a single entry wound in front of the chest to the right of the sternum which was closed with two skin stitches. Two litres of blood was transfused and the right side of the chest aspirated, about a litre of blood being removed. Radiographs of the chest showed many pellets scattered over a wide area. The heart was evidently involved. It was noticed that some of his peripheral pulses were absent.

On May 16 the boy was transferred to St. Thomas's Hospital. On admission he was drowsy and a little cyanosed, but his general circulatory state was satisfactory, with a pulse rate of 100 a minute and blood-pressure, measured with a sphygmomanometer on the left arm, 120/60 mm. Hg. The respiratory rate was 30 a minute. There was a round entry wound, which had been sutured, to the right of the sternum over the third interspace. No exit wound could be found. Physical examination revealed signs of a haemothorax on the right but no evidence of cardiac tamponade. Peripheral pulses could not be felt in the right arm below the brachial or in the right lower limb below the femoral. The peripheral pulses on the left side were normal. Radiographs (Figs. 1 and 2) showed a total of 30 pellets widely distributed. Three lay in the region of the right brachial bifurcation, accounting for the absence of pulses at that wrist. Several others were in the right axillary and subclavian areas and presumably lying in branches of these arteries. Two lay in the left carotid bifurcation area and one in the right. These were apparently impacted in the mouths of the internal carotid arteries, as the superficial temporal pulse was easily felt on each side. A number of other pellets lay in line with the heart. No pellets could be seen in films of the lower limb, so presumably the absence of pulses was due to embolism of radio-translucent material to the right popliteal artery.