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Growth decelerations among under-5-year-old children in Kasongo (Zaire). I. Occurrence of decelerations and impact of measles on growth

KASONGO PROJECT TEAM[1]

The occurrence of growth decelerations in children was studied in more than 16 000 two-to-four-month time intervals, ending at ages 6 through 59 months, during a multi-round survey in Kasongo. Decelerations were measured as changes in standard deviation scores which were compared with international and local weight-for-age, weight-for-height, arm-circumference-for-age and arm-circumference-for-height reference data. Decelerations were strikingly frequent, particularly at younger ages. An important number of decelerations was related to infection with measles which provokes initial wasting; later, recuperation was partly through increase of weight and arm circumference, and partly through delayed stunting. The measurement of growth decelerations has the advantage over isolated weight-for-age or arm-circumference-for-age measurements in that the impact of age-dependency-induced errors is largely eliminated. The relative merits of the local and international reference curves are discussed.

Certain infections—for example, measles and diarrhoeal diseases—can harm a child's nutritional status; they may cause interruptions in growth and eventually a permanent growth deficit or even death (*1*). Such growth decelerations could therefore be a warning sign.

The assessment of a child's growth evolution has been carried out in African countries with the aid of charts of the "Road to Health" type (*2*). Some well-documented case studies show how the death of a child may be preceded by a growth deceleration and how diseases interfere with the growth curve. However, there is little community-based evidence available to justify the generalization of prognostic inferences from the assessment of a child's growth evolution in the operational context of maternal and child health (MCH) clinics.

The first part of this article examines some aspects of the epidemiology of growth decelerations among under-5-year-old children in Kasongo, Zaire, and particularly the influence of measles on the occurrence of such decelerations. The second part examines the relevance of the detection of growth deceleration to the prediction of the risk of dying and the operational implications for the monitoring of growth changes among children attending MCH clinics.

SUBJECTS AND METHODS

Kasongo is the main town (population, about 30 000) in a rural area of about 180 000 inhabitants in the east of Zaire. Bordering on forest and savanna, it has a tropical climate and a mainly agricultural economy (rice, cotton, groundnuts); the annual income per head is about US$ 200.

A multiround survey was carried out in part of this town to assess the health status, morbidity and mortality of children aged under 5 years. The survey included serial measurements of weight, height, and arm circumference, which were used to construct local reference curves; details are given elsewhere (*3*).

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Assessment of growth changes

The above-mentioned measurements made it possible to assess the growth of a child during a series of time intervals; only the intervals ending when the child was between 6 and 59 months old were acceptable. In order to be eligible for inclusion in the study, these intervals had to be 2 to 4 months long, and the last measurement had to be followed up for at least 100 days.

Between May 1974 and June 1977, a total of 4273 under-5-year-old children were seen at least 3 (and up to 13) times and provided anthropometric information on 21 734 time intervals; 502 (2.3%) of these intervals had to be excluded from the analysis for lack of follow-up: the child had not been seen again, had not reached the fifth birthday, and it was not known whether the child had died during the 100 days after the last measurement. A further 1018 intervals had to be eliminated because they were too short (less than two months) and 3907 because they were too long (more than four months). Thus, 16 242 (74.4%) intervals remained for analysis of the arm-circumference measurements, and somewhat less than this number for the other anthropometric parameters (see Table 1); this corresponds to an average of about 4 three-month intervals per child. For every measurement, the standard deviation score (SD score) (*4*) was calculated by dividing the difference between the actual measurement and the reference mean by the standard deviation of the reference data for that age (or height).

The growth changes (accelerations or decelerations, compared with the reference curve) during the time intervals were measured as the difference between the SD score at the end of the interval and the SD score at the beginning of the interval. These growth changes were expressed in SD score units, a reflection of the change in position relative to the reference curve rather than of the change in anthropometric value.

The NCHS (National Center for Health Statistics, Center for Disease Control, USA) data (*5*) were used as reference data in the calculation of SD scores for weight-for-age (W/A-NCHS) and weight-for-height (W/H-NCHS). One SD score unit for weight-for-age is equivalent to a change from 0.9 kg at age 6 months to 1.9 kg (in girls) and to 2.1 kg (in boys) at age 59 months. The equivalent of 1 SD score unit for weight-for-height data ranges from 1 or 1.1 kg for a 60-cm child up to 1.1 or 1.4 kg for a 100-cm girl or boy, respectively. W/H reference figures are slightly lower than the Kasongo average for small children, and slightly higher than that for taller children (*3*). The local Kasongo weight-for-age (W/A-K) data (*3*) were also used as reference data; the equivalent of an SD score unit then ranges from 1.2 kg at 6 months to 2.0 kg at 59 months.

The SD scores for arm-circumference-for-age (AC/A-K) were calculated using the Kasongo data

Table 1. Percentage frequency of growth decelerations of a magnitude of at least 0.5 or 1 SD score unit during time intervals of 2–4 months

Anthropometric parameter	Percentage frequency of growth decelerations					
	Intervals ending at age 6–24 months		Intervals ending at age 25–59 months		All intervals	
	Decelerations of ⩾0.5 SD score unit	Decelerations of ⩾1 SD score unit	Decelerations of ⩾0.5 SD score unit	Decelerations of ⩾1 SD score unit	Decelerations of ⩾0.5 SD score unit	Decelerations of ⩾1 SD score unit
Weight-for-age (Kasongo)	15.6 (5288)[a]	6.4 (5288)	13.4 (10 775)	5.7 (10 775)	14.0 (16 063)	5.9 (16 063)
Weight-for-age (NCHS)	30.2 (5288)	13.7 (5288)	13.8 (10 779)	5.9 (10 779)	19.2 (16 063)	8.4 (16 063)
Weight-for-height (NCHS)	43.6 (4650)	34.7 (4650)	26.8 (10 419)	13.8 (10 419)	31.9 (15 069)	20.2 (15 069)
Arm-circumference-for-age (Kasongo)	22.4 (5416)	8.4 (5416)	21.5 (10 826)	7.7 (10 826)	21.6 (16 242)	8.0 (16 242)
Arm-circumference-for-height (Kasongo)	24.9 (4684)	9.8 (4684)	22.2 (10 447)	7.9 (10 447)	23.0 (15 131)	8.5 (15 131)

[a] Figures in parentheses indicate the number of intervals on which the calculation was based.

(*3*). The equivalent of 1 SD score unit remains between 1.0 and 1.2 cm for both sexes from age 6 to 59 months. Unpublished data from Kasongo were also used as a reference for the arm-circumference-for-height data (AC/H-K). The equivalent of 1 SD score unit for AC/H-K was on average 1.1 cm, with occasionally 1.0 or 1.2 cm.

Assessment of the influence of measles

During 806 (5.0%) of the studied intervals an episode of measles was recorded. This disease is well recognized by the population and has a local name. In about 75% of cases the mother had brought the child to the health centre or hospital, and in 98% of these the diagnosis was confirmed by a health worker (usually a nurse). It should be noted that if one assumes that every child had measles between the age of 6 and 59 months and that every case of measles had been notified, there would have been notification in 5.6% of all single three-month periods. Measles notification thus appears to be fairly complete.

In a cohort sample of 150 children, the impact of measles on the growth pattern was assessed by comparing their SD scores before they experienced measles with those at various times after the onset of measles. This was done for the aforementioned anthropometric parameters and for height-for-age (H/A-NCHS), using the NCHS data as reference curve. The median age of onset of measles was 18 months.

RESULTS

Growth decelerations are strikingly frequent in Kasongo. The frequencies of decelerations of a magnitude of at least 0.5 SD score unit and of at least 1 SD score unit are shown in Table 1 for W/H-NCHS, W/A-NCHS, W/A-K, AC/A-K and AC/H-K. Fig. 1 plots these frequencies by age at the end of the interval during which the growth change was observed. There are no relevant differences between boys and girls, so both sexes have been re-

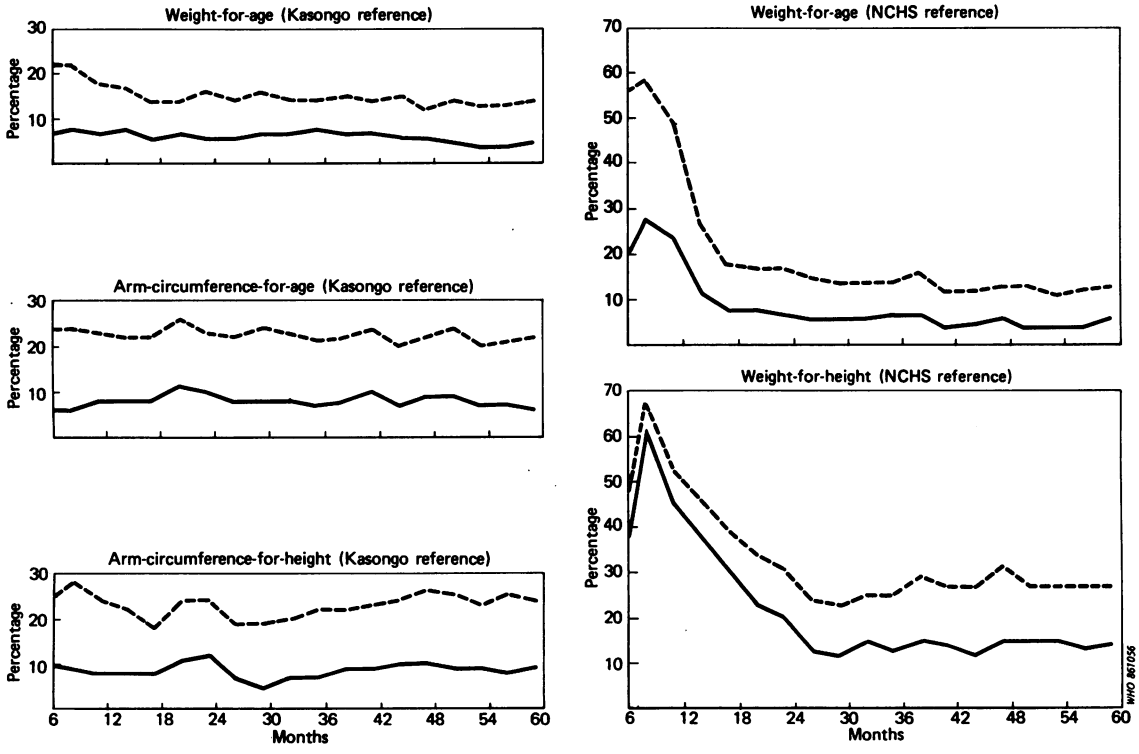

Fig. 1. The relative percentage frequency of growth decelerations of a magnitude of at least 0.5 (broken line) or 1 (full line) SD score unit in relation to the children's age at the end of the time intervals, for the various anthropometric parameters.

Table 2. Percentage frequency of growth decelerations of a magnitude of at least 0.5 or 1 SD score unit during intervals lasting 2–4 months during which a case of measles was notified

Anthropometric parameter	Percentage frequency of growth decelerations			
	Intervals ending at age 6–24 months		Intervals ending at age 25–59 months	
	Decelerations of ⩾0.5 SD score unit	Decelerations of ⩾1 SD score unit	Decelerations of ⩾0.5 SD score unit	Decelerations of ⩾1 SD score unit
Weight-for-age (Kasongo)	26.1 (406)[a]	7.3 (406)	20.4 (392)	8.4 (392)
Weight-for-age (NCHS)	46.1 (406)	20.7 (406)	21.2 (392)	8.7 (392)
Weight-for-height (NCHS)	48.9 (354)	37.3 (354)	31.2 (378)	17.5 (378)
Arm-circumference-for-age (Kasongo)	37.4 (414)	17.1 (414)	36.0 (392)	16.1 (392)
Arm-circumference-for-height (Kasongo)	37.5 (343)	18.1 (343)	37.7 (383)	15.1 (383)

[a] Figures in parentheses indicate the number of intervals on which the calculation was based.

grouped—although, of course, calculations of the SD scores were made separately. For children of nearly every age, the decelerations were observed most frequently for W/H-NCHS, and least frequently for W/A-K.

Decelerations tend to occur more frequently at younger ages. This tendency is pronounced and statistically significant for W/H-NCHS and W/A-NCHS (χ^2, $P<0.001$). Although significant for AC/H-K and W/A-K, the differences in frequency are much smaller.

Table 2 shows that decelerations occurred more frequently in intervals when measles infection was alleged to occur. This is true both for younger and for older children, and is significant (χ^2, $P<0.001$) for all parameters. It is most pronounced for arm-circumference measurements, where 1 SD score unit decelerations are 2.0 (AC/H-K) to 2.6 (AC/H-K) times more frequent when measles was notified than when it was not.

Fig. 2 demonstrates the deleterious effect of measles on the nutritional status. It displays, for various moments after the onset of measles, the average difference in SD score with the score obtained before the onset of the disease. With the exception of H/A-NCHS measurements, the growth deficit is most marked in the first two months after the beginning of measles.

DISCUSSION

Measles has a definite impact on nutritional status, which is characterized by an initial loss of weight and reduction in arm circumference, the main effect being noted about one or two months after the onset of disease. In the three comparisons with local curves (W/A-K, AC/A-K and AC/H-K), there was no significant growth deficit left after the third month from the onset of measles.

In terms of W/A-NCHS, the growth deficit remained significant 7 months after the onset of measles. The Kasongo W/A growth pattern was characterized by a growing deficit (compared with international standards) which occurred during the 6–24-month age period (*3*). The main explanation for this phenomenon, which is prevalent in Africa, is the interaction between infection and nutrition (*8*). Recuperation of the deficit in terms of W/A-K and the ongoing deficit in terms of W/A-NCHS point to the role of infection, and particularly of measles, in the accumulation of the population's growth deficit.

For W/H-NCHS, the maximum deficit occurred around the second month after the onset of measles. By the seventh month there was partial recuperation through an increase in weight, but there remained a significant growth deficit of an average of 0.6 kg. This lasting deficit is similar to the one demonstrated in Bangladesh (*7*). It is likely that there will be progressive recuperation through delayed stunting rather than through further increase in weight. Indeed, by that time the deficit relative to the local weight-for-age curve should have been filled, while simultaneously, starting from the fifth month on, a significant stunting effect (compared with the NCHS data) appears.

Of course not all the growth decelerations, which, as we have seen, are frequent, particularly at younger

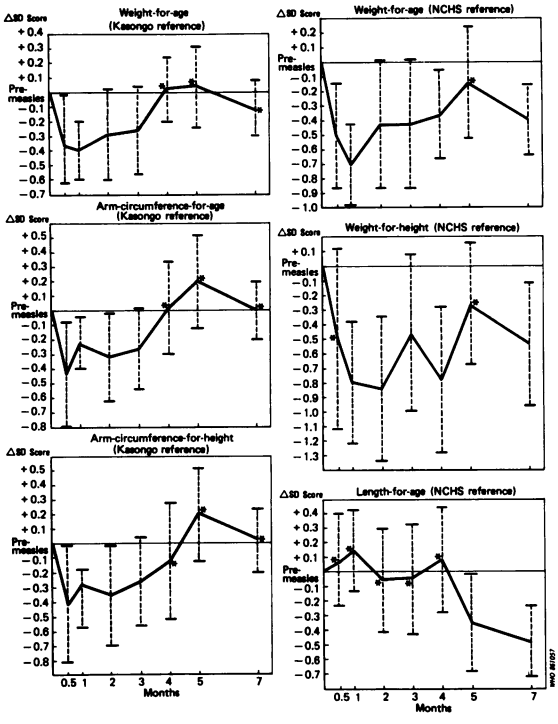

Fig. 2. The average difference, with 95% confidence interval, for a cohort sample of 150 children, between their SD score before and at various times after the onset of measles. The averages are based on a total of 301 measurements: 18, 52, 40, 41, 30, 42 and 78 measurements at respectively 0.5, 1, 2, 3, 4, 5 and 6–8 months after the onset of measles. The differences indicated with * are not significantly different ($P<0.05$, one-sided test) from the pre-measles SD score.

ages, are related to measles. They can roughly be attributed to three causes: measurement errors, pathological processes, and other differences in growth pattern between individual children and the reference curve, the latter being the resultant of a number of individual growth spurts.

Measurement errors certainly are responsible for noise on the information. According to the reproducibility tests and control measurements, errors larger than 500 g or 1 cm arm circumference should be uncommon (*3*); the former are of the order of 0.25 to 0.5 SD score unit, the latter of somewhat less than 1 SD score unit. With this level of measurement reproducibility, errors should interfere little for the larger decelerations (more than 1 SD score unit), and at older ages when, for the weight measurements at least, the standard deviations of the reference curves are larger. For the weight-for-height data, measurement errors might interfere more; there is not only the problem of precision in weight but also in height measurement. There is, however, no reason to suppose that such measurement errors work only in one way, i.e., that they increase the frequency of growth decelerations, and not that of growth accelerations; both types of error probably even up. The measurement errors would thus affect the specificity of the relation between observed decelerations and a possible pathological process rather than the actual frequency of decelerations. Another possible source of noise on the information comes from errors in the estimation of the age of the children. This would result in an erroneous choice of reference value with a consequent error in the calculation of the SD score. The calculation of differences in SD score however is fairly robust and age-independent. In Kasongo, one SD score unit corresponds to a median growth of 4–8 months duration (for W/A-NCHS), 5–18 months (W/A-K), or more than one year (AC/A-K). Errors in the estimation of age therefore have to be very gross before they interfere with the observation of growth changes of the magnitude of 0.5–1 SD score unit. One operationally important implication is that if decelerations are measured instead of the position of a child in the distribution, one of the biggest handicaps for weight-for-age measurements, i.e., age dependency and its related errors, is avoided.

One has then to clarify to what degree the observed growth decelerations are the expression of acute pathological processes that warrant special attention. Their increased frequency in the case of measles shows that at least part of the decelerations is related to such processes.

The difference in the age distribution pattern of the relative frequencies of growth decelerations can be explained as follows. The Kasongo under-5-year-old population as a whole begins to accumulate a deficit from the sixth month onwards. This deficit further increases up to the 24th month, after which it stabilizes (*3*). There are indications that this happens without disrupting the homogeneity of the group; all children experience some growth deficit (compared with the NCHS reference curve) and the position of an individual child within the local distribution of anthropometric parameters carries no consequences in terms of risk of subsequently dying (*3*, *9*). The Kasongo children follow a growth pattern which is different from the NCHS growth pattern. This explains the exceedingly high figures for the frequency of W/A-NCHS decelerations at younger ages, i.e., during the period in which all Kasongo children accumulate a deficit compared with the NCHS curve, as an expression of adjustment to the nutritional environment. The frequency of decelerations for W/A-K in the 6–24-month period, i.e., in

the period of accumulating deficit, is much lower. In the period of stabilized deficit, after 24 months, the frequency of decelerations is about equal for W/A-K and W/A-NCHS: 13.4% and 13.8%, respectively, for 0.5 SD score unit decelerations; 5.7% and 5.9% for 1 SD score unit decelerations. Arm-circumference measurements might be somewhat more sensitive to the impact of infectious processes, as far as can be judged from the frequency of arm-circumference decelerations in the case of measles. The higher frequency of W/H-NCHS decelerations at younger ages is probably related to a delay between weight loss and stunting. As shown before, this delay can be observed for the post-measles evolution of individual children. It can also be observed at population level: the deficit in mean weight-for-age stabilizes around 18 months while the deficit in mean height-for-age continues to increase up to 30 months (*3*).

An important implication of this observation is that charts of the "Road to Health" type based on the NCHS or similar weight-for-age data will, if used for monitoring the growth pattern, tend to grossly overestimate the frequency of growth decelerations that are an expression of a pathological process, at least in the 6–24-month period. Comparisons with both local curves and NCHS data provide information contaminated with a lot of noise due to measurement errors. At younger ages, however, local curves seem to have the advantage that if there is a growth deceleration, this is more likely to represent an acute pathological process, whereas if the NCHS data are used, a large proportion of the decelerations seems to be due to the adjustment to the environment rather than to these acute processes. One can of course argue that this adjustment is not desirable, and that local curves underestimate the child's theoretical potential. The NCHS data are certainly more useful to detect deviations from the full potential growth pattern. They provide the most accurate information on physiologically relevant decelerations. This deviation from the full potential growth pattern is, in Kasongo, a process common to the whole population group. The comparison with the local curve is more adapted to the identification of deviations from what can reasonably be expected in the given context. Even if this is physiologically less accurate it might be operationally more relevant in as much as it focuses attention on the more vulnerable acute situations where the growth pattern is unsatisfactory, even in the local context.

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RÉSUMÉ

DÉCÉLÉRATION DE LA CROISSANCE CHEZ LES ENFANTS DE MOINS DE 5 ANS À KASONGO (ZAÏRE). I. FRÉQUENCE DES DÉCÉLÉRATIONS ET IMPACT DE LA ROUGEOLE SUR LA CROISSANCE.

Une enquête par passages répétés à Kasongo, Zaïre, a permis d'étudier les décélérations de croissance chez des enfants de moins de 5 ans. Les paramètres anthropométriques considérés sont les suivants: poids par rapport à l'âge (P/A) et par rapport à la taille (P/T), circonférence brachiale par rapport à l'âge (CB/A) et par rapport à la taille (CB/T). Pour chaque paramètre et chaque mesure les écarts-types ont été calculés par rapport aux données de référence du National Center for Health Statistics (NCHS) des Etats-Unis d'Amérique (P/A et P/T) ou locales (P/A, CB/A, CB/T). Les décélérations ont été mesurées par le changement de valeur de l'écart-type entre le début et la fin de plus de 16 000 intervalles de temps, d'une durée de 2 à 4 mois, se terminant à un âge compris entre 6 et 59 mois. La mesure de différences d'une ampleur d'au moins 0,5 ou 1 unité d'écart-type sur une période de 2 à 4 mois est robuste par rapport aux erreurs de déclaration de l'âge des enfants.

Les décélérations de croissance sont fréquentes, surtout aux plus jeunes âges et si on compare aux données de référence du NCHS: des décélérations de plus d'un écart-type ont été observées durant 8,4% (P/A) et 20,2% (P/T) des intervalles, mais cette fréquence dépasse 20% et 40% respectivement pour les intervalles qui se terminent à un âge compris entre 6 et 12 mois. Par rapport aux données de référence locales la fréquence des décélérations d'au moins un écart-type est de 5,9% pour P/A, de 8,0% pour CB/A et de 8,5% pour CB/T.

Le rôle des infections dans la genèse des décélérations de croissance est illustré par le cas de la rougeole. Des décélérations d'un écart-type sont jusqu'à 2,6 fois plus fréquentes dans les intervalles pendant lesquels un épisode de rougeole est notifié. L'analyse d'un échantillon de 150 cas de rougeole, pour lesquels la valeur de l'écart-type était connue avant la rougeole et deux mesures, en moyenne, ont été

faites dans les 6 mois suivant le début de la maladie, montre que la rougeole provoque une chute initiale de la valeur de l'écart-type pour le poids et la circonférence brachiale par rapport aux données de référence. Après 4 mois la perte est suivie d'une récupération par rapport aux courbes locales: celle-ci semble se traduire en partie par une augmentation du poids et de la circonférence brachiale, et en partie par un ralentissement de la croissance linéaire, qui ne survient que plusieurs mois après le début de la maladie.

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