Linear Growth Retardation and Mortality

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When discussing the relationship between stunting and mortality in developing countries, one has to distinguish two separate issues. (a) Does being stunted, i.e., presenting with retarded linear growth, carry an additional risk of dying? (b) Does stunting, i.e., presenting with deceleration of linear growth, carry an additional risk of dying? Very little is known on either subject.

The data gathered by Keller and Fillmore on the prevalence of protein-energy malnutrition throughout the world (1) show that countries with high juvenile and infantile mortality rates also invariably have high prevalences of stunted children, and those with low prevalences invariably have low mortality rates (Fig. 1). There are, nevertheless, a number of countries such as Yugoslavia, Sri Lanka, Costa Rica, or Panama where low juvenile mortality rates coexist with high prevalences of stunted children.

Thus, poor countries that tend to have high mortality rates also tend to have high prevalences of stunted children.

Very little data are available from a small number of community-based longitudinal studies that can be used to evaluate the relationship between anthropometric indicators and mortality (Table 1).

Only the two more recent studies from Bangladesh (4,6) question the relationship between being stunted and risk of dying, and only Bairagi (6) also addresses the importance of stunting. In this chapter, these observations will be confronted with some of the data from the Kasongo (Zaire) study.

The Kasongo study was a multiround survey whose principal objective was to assess the influence of measles vaccination on the survival pattern of under-5s. More than 32,500 weight, height, and arm-circumference measurements were taken in over 7,000 children. A detailed description of the study population and the way in which the survey was conducted may be found elsewhere (8).

The growth curves\textsuperscript{1} of the Kasongo children show a fairly typical pattern for

\textsuperscript{1}Unless it is specifically mentioned that local or other reference data were used, the NCHS data (9) were used as "reference." Between 24 and 36 months, when length and stature reference data overlap, these were averaged, as were their corresponding standard deviations.
Prevalence of stunted children, i.e., children below the mean minus 2 SD of the reference height for age (23 countries) or less than 90% of the reference height for age (14 countries) at the age of 1 year, and child mortality rate. (From Keller and Fillmore, ref. 1.)

**TABLE 1. Anthropometric indicators whose relationship with mortality has been investigated in nonhospitalized populations**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Single measurements</th>
<th>Measurements of velocity or loss</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>W/A  H/A  W/H</td>
<td>AC/A  AC/H  WtQ  HtQ</td>
</tr>
<tr>
<td>Sommer and Lowenstein, 1975, Bangladesh (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kielmann and McCord, 1978, India (3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chen et al., 1980, Bangladesh (4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kasongo Project Team, 1983, Zaire (5)</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>Bairagi et al., 1985, Bangladesh (6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bairagi et al., 1985, Bangladesh (6)</td>
<td></td>
<td>*</td>
</tr>
<tr>
<td>Kasongo Project Team, 1986, Zaire (7)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*W/A, weight for age; W/H, weight for height; H/A, height for age; AC/A, arm circumference for age; AC/H, arm circumference for height; WtQ, weight quotient; HtQ, height quotient (WtQ and HtQ are the ratios of the weight-age or height-age, i.e., the age at which the weight or height of the child is at the 50th percentile of the Harvard standard, divided by the child's chronological age).
African under-5s, with a deficit in weight for age that becomes evident from 5 to 6 months on. The deficit in height for age starts earlier and is already noticeable at the age of 2 months (Fig. 2). Figure 3 shows that the whole distribution is shifted to the left when compared to the NCHS distribution; the whole population is affected by growth retardation. However, as is evidenced by the weight-for-height curve, which is quasiidentical to the NCHS reference, longitudinal growth deficit and weight deficit in this population are quite proportional; wasting is relatively rare. Figures 4 and 5 show the prevalence of stunted children by age according to the classical Waterlow classification (10) or using the median minus 2 SD of the NCHS reference as criterion: the prevalence increases progressively and peaks during the third year of life; it stabilizes at a slightly lower level after 36 months.

OBSERVATIONS ON THE ASSOCIATION BETWEEN BEING STUNTED AND SUBSEQUENT MORTALITY

The first study providing evidence for an association between being stunted and subsequent mortality was published by Chen et al. in 1980 (4) on the basis of a 2-year follow-up of 2,019 children in Bangladesh. For any given level of weight-for-height deficit at ages 12 to 23 months, a deficit in height for age was associated
with high mortality rates during the two subsequent years. Children who died were 1.2 times as likely to be less than 90% and 2.0 times less than 85% of the Harvard median height for age than the overall study population. Risk of dying increased sharply below a threshold situated around 85% of the reference values.

Table 2 shows the ratio of the risk of dying during the two subsequent years for children below and above 85% of height for age. A height for age below 85% was associated with increased risk of dying whether the weight for height was above or below 80%, independently of indicators of maternal nutrition and housing size. Poor height or weight of the mother was not associated with an increased risk of dying for children above 85% (risk ratios of 0.99 and 0.92), whereas it was for children below 85% (risk ratios of 1.58 and 1.25); small housing size was associated with a higher risk for both stunted and other children, but more so for the stunted children, with a risk ratio of 2.29 instead of 1.24. This supports the hypothesis that stunted children are more vulnerable than others to whatever ultimately will be the direct "cause" of death.

Bairagi et al. (6), who studied children in the same population as Chen et al. (4) (actually, about 40 children were included in both studies), confirmed the association of low height for age with subsequent mortality, but without finding Chen's threshold phenomenon.

In principle, the most convincing evidence for the hypothetical association
FIG. 4. Prevalence by age of stunted, stunted and wasted, and wasted children, according to the Waterlow classification.

FIG. 5. Prevalence by age of stunted children using the NCHS median minus 2 SD of height for age as cutoff.
TABLE 2. Ratios of risk of dying within 2 years for children above and below 85% of the Harvard standard height for age

<table>
<thead>
<tr>
<th>All children</th>
<th>2.98</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wt/Ht ≥ 80%</td>
<td>2.88</td>
</tr>
<tr>
<td>Wt/Ht &lt; 80%</td>
<td>1.54</td>
</tr>
<tr>
<td>Shorter mother</td>
<td>3.45</td>
</tr>
<tr>
<td>Taller mother</td>
<td>2.16</td>
</tr>
<tr>
<td>Lighter mother</td>
<td>3.35</td>
</tr>
<tr>
<td>Heavier mother</td>
<td>2.48</td>
</tr>
<tr>
<td>Small housing size</td>
<td>2.29</td>
</tr>
<tr>
<td>Large housing size</td>
<td>4.23</td>
</tr>
</tbody>
</table>

From Chen et al. (4).

FIG. 6. Height distribution of the Matlab study population (solid line) and of subsequently dying children (dashed line). (From Chen et al., ref. 4.)

would be provided by a comparison of the height distribution of the subsequently dying children and the overall population. Such a comparison of distributions is preferable to one of "prevalences" (11). One would expect subsequently dying children to be shorter than the other children of the same age in the population: the height distribution of subsequently dying children should be different—shifted to the left of that of the overall population.

Chen and his co-workers (4) observed this shift to the left (Fig. 6). However, in Kasongo the pattern is different.

Methods

Anthropometric information is available for 137 children who died at the age of 6 months or older (including three deaths at the age of 61 months). Z scores of height for age at the last measurement before death (the average interval between measurement and death being 2.3 months) were calculated for each of these children, using the local mean and standard deviation of height for age. This provides an age-independent distribution that can easily be compared with that of the popu-
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lation, assuming that at any age the latter is a normal distribution (the mean \( = 0 \) and 1 SD = 1). The statistical significance of differences between the distribution of subsequently dying children and that of the overall population was tested using a Kolmogorov–Smirnov one-sample test.

Results

The distribution of the Z scores of height for age at the last measurement made on each child (Fig. 7) is not significantly different from the overall distribution of the population (Kolmogorov–Smirnov \( Z = 0.910; p = 0.380 \)). This is further confirmed by Table 3, which shows that the average Z scores do not differ significantly from the population average of 0.

Thus, the distribution of the Z scores does not corroborate the hypothesis of an association between being stunted and subsequent mortality. The corollary of this distribution is that risk of dying is fairly homogeneously distributed among the Kasongo children of a given age, whatever their attained height.

OBSERVATIONS ON THE ASSOCIATION BETWEEN THE STUNTING PROCESS AND MORTALITY

If an association between the stunting process and subsequent mortality exists, then children who die should have experienced periods of less intensive linear

![Distribution of the Z scores of height for age at the last measurement before death, using the local mean and SD as reference; \( n = 137 \). Average interval between measurement and death was 2.3 months; circles, overall distribution for all children.](image-url)
growth than one would expect in the overall population.

Bairagi et al. (6) examined the association between mortality and growth velocities (g or cm/month over periods of 2 months) in 1- to 4-year-old children from Matlab. They did find an association between mortality and weight velocity, but not with height velocity. However, for a number of reasons their results are difficult to interpret. The small number of deceased children on whom previous anthropometric information was available did not make it possible to control for age, which should affect height velocities measured in that fashion. The survey was conducted in the aftermath of a famine: catch-up growth was observed, as evidenced by a U-shaped relationship between weight velocity and mortality. There were also important seasonal variations in growth. With that background, the fact that height velocities were not significantly lower for subsequently dying children does not provide a formal argument against the hypothesis of an association; it merely provides no formal argument for it.

**Material and Methods**

In order to test for such an association in Kasongo, the process of growth faltering had to be measured in some way that makes it possible to compare the growth of the subsequently dying children and that of the overall population by taking age into account. The difference between the Z scores of two measurements, divided by the time interval between those measurements, provides a convenient standardized expression of the growth increment during this interval as a number of SD units of change per month. If this "standardized monthly velocity" is negative; i.e., if the Z score at the second measurement is lower than the first, there is growth deceleration (as compared to the average monthly increment of the reference population, which by definition is equal to zero); if it is positive, there is growth acceleration. If height for age is being measured, a deceleration can be called stunting.

For 78 children who died at the age of 6 months or older, information is available for 238 time intervals. It was thus possible to calculate standardized monthly

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2 Apparently height measurements less than 5 months preceding death were available for only 14 children; growth velocity in these children was less than 0.40 cm/month in the period preceding death, compared to velocities ranging from 0.60 to 0.68 cm/month for surviving children.
TABLE 4. Average standardized monthly growth velocities at different ages in Kasongo
(measured in SD score units per month)*

<table>
<thead>
<tr>
<th>Age group</th>
<th>0-5 months</th>
<th>6-11 months</th>
<th>1 year</th>
<th>2 years</th>
<th>3-4 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>H/A</td>
<td>-0.014</td>
<td>-0.025</td>
<td>-0.040</td>
<td>-0.025</td>
<td>-0.004</td>
</tr>
<tr>
<td>W/A</td>
<td>-0.050</td>
<td>-0.125</td>
<td>-0.008</td>
<td>0.012</td>
<td>-0.008</td>
</tr>
<tr>
<td>W/Hb</td>
<td></td>
<td>-0.040</td>
<td>-0.050</td>
<td>&lt;0.004</td>
<td></td>
</tr>
</tbody>
</table>

*H/A, height for age; W/A, weight for age; W/H, weight for height.

Approximation from the average Z scores of weight-for-height curves for ages 0-5, 6-11, 12-23, and 24-59 months.

TABLE 5. Average standardized monthly height-for-age velocities (SD units/month) of subsequently dying children*

<table>
<thead>
<tr>
<th>Age group (months)</th>
<th>0-5</th>
<th>6-11</th>
<th>12-23</th>
<th>24-35</th>
<th>36-59</th>
</tr>
</thead>
<tbody>
<tr>
<td>All intervals:</td>
<td>-0.575</td>
<td>-0.082</td>
<td>-0.061</td>
<td>-0.059</td>
<td>-0.002**</td>
</tr>
<tr>
<td>Velocities measured &lt; 12 months before death</td>
<td>-0.217</td>
<td>-0.099</td>
<td>-0.075</td>
<td>-0.061</td>
<td>-0.008*</td>
</tr>
<tr>
<td>Velocities measured during intervals ending ≤ 3 months before death</td>
<td>-0.268</td>
<td>-0.066</td>
<td>-0.099</td>
<td>-0.090</td>
<td>-0.019</td>
</tr>
</tbody>
</table>

*Significance: *p < 0.05; **NS; all others p < 0.01. All children died between 6 and 61 months. Number of months of observation for the calculation in each age group: (a) 70, 288, 457.5, 228, 158; (b) 44, 118, 116 (two outliers removed), 118, 99; (c) 20, 81, 48 (two outliers removed), 74, 47.

The average standardized monthly growth velocities of height for age, weight for age, or weight for height at different ages. These standardized monthly velocities were averaged for each of the following age groups: 0 to 5, 6 to 11, 12 to 24, 24 to 35, and 36 to 59 months. (If any interval overlapped with one of these age groups, the total standardized growth change was accordingly divided between the different age groups for calculation of the monthly standardized velocities.) This was done (a) using all the available information on subsequently dying children, which totaled just over 100 child-years of observation, (b) for the year preceding the death of each child (a total of 495 months of observation), and (c) for time intervals ending not more than 3 months before death; the average duration of time interval was 2.7 months (a total of 270 months of observation).

For each age group, these averages were compared with the average standardized monthly growth increments of the whole population (Table 4). Statistical significance of differences from the standardized monthly growth increment of the whole population in the same age group was assessed with a one-sided Z test.

The average standardized monthly height velocities of subsequently dying children (Table 5) are negative and significantly more so than those of the overall pop-
ulation (Table 4). This is true at all ages; from the age of 1 year the difference is most marked when only growth increments in the months immediately preceding death are taken into consideration (Table 5, c).

The difference between the standardized monthly height-for-age velocity during the period preceding death and that of the overall population is graphically represented in Fig. 8. Monthly height-for-age velocity of children who will die within the coming months (average 1.7 months after the last measurement) is 19 times slower (i.e., actual stunting) than expected at ages 0 to 5 months, 2.6 times slower at ages 6 to 11 months, and 2.5, 3.6, and 4.7 times larger at ages 1, 2, and 3 through 4 years, respectively.

As shown in Table 6, the decelerations of linear growth during the last months preceding death were also accompanied by larger than expected decelerations of weight for age and weight for height, i.e., actual wasting. In fact, 47.4% of 190 child-months with negative standardized monthly height-for-age velocity also showed a negative figure for weight for height; in 6.3%, this exceeded 0.5 SD units per month, indicating marked wasting. Wasting was much more frequent for those child-months without stunting: 84.8% of 106 child-months; 22.6% with a wasting of more than 0.5 SD units of weight for height per month.

Nevertheless, Table 7 shows that stunting during the period preceding death was more intense than in the general population, even for those children who did not show growth deceleration in terms of weight for height, i.e., who were not wasting, and even for children who presented little or no deceleration in terms of weight for age.

THE IMPLICATIONS

The discussion of the implications of the findings focuses on three aspects: (a) the apparent contradiction between the lack of association between mortality and

![FIG. 8. Standardized monthly height-for-age velocities (SD units/month) at different ages; base line, reference population (zero by definition); open arrows, average for all children; solid arrows, average for subsequently dying children.](image-url)
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TABLE 6. Average standardized monthly velocities of weight for age and weight for height of subsequently dying children

<table>
<thead>
<tr>
<th>Age group (months)</th>
<th>0-5</th>
<th>6-11</th>
<th>12-23</th>
<th>24-35</th>
<th>36-59</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight for age</td>
<td>0.171</td>
<td>0.190</td>
<td>0.115</td>
<td>0.186</td>
<td>0.075</td>
</tr>
<tr>
<td>Weight for height</td>
<td>0.056</td>
<td>0.087</td>
<td>0.163</td>
<td>0.124</td>
<td>0.066</td>
</tr>
</tbody>
</table>

*Averages based only on intervals ending ≤ 3 months before death.

TABLE 7. Average standardized monthly height-for-age velocity of subsequently dying children (a) without weight for height deceleration, (b) with decelerations of less than 0.25 SD units of weight for age per month, and (c) without weight-for-age decelerations

<table>
<thead>
<tr>
<th>Age group (months)</th>
<th>0-5</th>
<th>6-11</th>
<th>12-23</th>
<th>24-35</th>
<th>36-59</th>
</tr>
</thead>
<tbody>
<tr>
<td>No W/H deceleration</td>
<td>0.632</td>
<td>0.328</td>
<td>0.270</td>
<td>0.147</td>
<td>0.078</td>
</tr>
<tr>
<td>W/A deceleration of less than 0.25 SD units</td>
<td>0.218</td>
<td>0.193</td>
<td>0.169</td>
<td>0.094</td>
<td>0.046</td>
</tr>
<tr>
<td>No W/A deceleration</td>
<td>0.018</td>
<td>0.354</td>
<td>0.147</td>
<td>0.123</td>
<td>0.048</td>
</tr>
</tbody>
</table>

*Velocities measured during intervals ending ≤ 3 months before death. All children died between 6 and 61 months of age. Number of children observed: 29, 31, and 21.

being stunted and the association with the process of stunting, as well as the discrepancies between the Matlab and Kasongo findings; (b) the causal nature of the association between stunting and mortality; (c) finally, some remarks on the implications of the results and their interpretation for the monitoring and evaluation of the health status of young children.

**Being Stunted, Stunting, and Dying**

Apparently the association between being stunted and increased subsequent risk of dying is found in Matlab but not in Kasongo. This contrast is even more impressive if one considers that the association in Matlab was demonstrated for risk of dying during the two subsequent years, whereas in Kasongo the interval between measurement and death was much shorter, an average of 2.3 months. One would expect the association to be stronger for short-term than for long-term mortality.

On the other hand, there is definitely an association between the stunting process and subsequent mortality in Kasongo. This association is most marked if one considers the period immediately preceding death (subgroup c in Table 5). It is present even in the absence of growth decelerations in terms of weight for height and weight for age. Those children who did not experience stunting in the months preceding death often experienced wasting.

The lack of association between mortality and attained height in Kasongo is not
really surprising. Similar observations have been made for weight for age and
height and, to a lesser extent, for arm circumference measurements (5). This lack
of association could be explained by the specificity of the socioeconomic and cul-
tural situation in Kasongo, where all children are basically exposed to a similar
physical and nutritional environment. This would be responsible for a shift to the
left of the growth curve of the entire population. Thus, a child on the upper fringe
of the local distribution presents a growth retardation (with associated increased
mortality risk), just as a child on the lower fringe does. Consequently, anthropo-
metric measurement will not discriminate between children with greater or smaller
environment- or nutritional-status-associated mortality risk.

An environment that causes a shift to the left for the entire population would
also explain the apparent contradiction with the Matlab data. Whereas Kasongo
children, on both the upper and lower fringes of the distribution, would be ex-
posed to the same environment and the same risk of dying, the Bangladeshi child
on the upper fringe of the height distribution for a certain age would probably be
exposed to a different environment than a child on the lower fringe and would
therefore be less exposed to the risk of dying. For the Matlab children this is cer-
tainly true to a certain extent: attained height and weight not only reflect the recent
famines but also are correlated with indicators of socioeconomic status (6). The
association between attained height and mortality in the Matlab studies, and espe-
cially Chen's demonstration of a cut-off point with increasing risk (4), is possibly
related to a more important social stratification with nutritional corollaries than in
Kasongo and to the fact that the overall nutritional situation appears to be consid-
erably worse and disequilibrated in the Matlab study population.

Within this explanatory framework, the lack of association between being
stunted and mortality does not preclude an association between mortality and
stunting. If the stunting process is associated with mortality but is not restricted to
a subgroup of the population and affects all children, then an associated excess
mortality is plausible even in the absence of discriminatory power of attained
height. On the other hand, in situations where attained height permits discrimina-
tion, such as in Matlab, the absence of association between mortality and stunting
is unlikely.

In any case, the scarcity of available evidence should make us very cautious
about extrapolating results of nutritional screening from one population to the
other and making causal inferences from them.

The Causal Nature of the Association Between Stunting and Mortality

The association between stunting and mortality in Kasongo satisfies a number
of the criteria required to call the stunting process a risk factor for subsequent
death. In order for the evidence to support a causal interpretation, however, a
plausible and intelligible biological mechanism has to be available (12,13). One should thus be able to formulate at least a tentative answer to the question of why a stunting child would be more at risk of dying.

Within this context it is useful to mention what was observed during a study of the influence of measles on the growth pattern of the children in Kasongo (14). Measles appears initially to cause weight loss and reduction of arm circumference. Four to 5 months after the onset of measles, the arm circumference regains its former level, but some deficit of weight for age, as compared to the NCHS reference, persists, and a statistically significant decrease of height for age starts to appear.

These phenomena could possibly be explained as follows. The height retardation would be the delayed effect of the nutritional stress caused by measles as a result of an incomplete recuperation during which recovery of an acceptable weight for height is privileged over ongoing linear growth. Stunting would become measurable in the aftermath of a serious stress. If a child is exposed to another infectious or nutritional stress while still recuperating from the previous one, it makes sense that he/she would be more vulnerable than the average child.

The nature of the association between stunting and subsequent mortality may be explained as follows: the final and direct cause of death in a stunting child would operate on a child still recuperating from recent stress, stunting being essentially a marker of the general vulnerability of the child to a new aggression. In such an explanatory framework, the frequency of infectious aggressions to which a child is exposed in a developing country (16) becomes extremely important and makes the association between stunting and mortality possible.

Unless a biological mechanism can be suggested through which the stunting process—or being stunted—per se would operate to make the child more vulnerable, the available evidence does not point towards an interpretation of stunting as a direct contributory cause of excess mortality. Rather, the association between stunting and mortality appears to be through their common cause, poverty, and its corollary of frequent nutritional and infectious stresses that do not allow for complete recuperation.

If poverty operates in conjunction with social stratification, as it appears to do in Matlab, attained height will reflect this stratification and therefore show an association with the poverty-linked increased risk of dying. If it operates in a less stratified way, as in Kasongo, attained height will not differentiate, within the population, groups more or less exposed to the poverty-linked increased risk of dying. Stresses affecting the child’s health may be differently distributed within these populations, but in both situations, basic mechanisms are similar: a common effect of stress on a child’s health is stunting; a less common effect is death, the latter occurring particularly when stresses occur in rapid succession and health care behavior at the microlevel does not enable the child to recuperate rapidly.

Linear Growth as a Monitor of Health Status of Children

Whatever its causal nature, the association between stunting and mortality makes it tempting to use it for evaluation purposes. A full discussion on the selec-
tion of indicators for monitoring health status of children goes beyond the scope of this chapter. Some remarks can nevertheless be made, keeping in mind that one has to distinguish (as for the evaluation of the nutritional status) (17) between the value of stunting as a tool to monitor the health status of individual children or to monitor and evaluate the health status of populations of children.

A statistically significant association can be demonstrated between stunting and the individual child’s risk of dying. This, however, does not necessarily provide a practical screening instrument for the identification of children at risk. Do static (as in Bangladesh) or dynamic (as in Kasongo) height-for-age measurements provide a significant marginal benefit over screening on the basis of weight or weight-for-height measurements?

In the population studied by Chen et al. (4), a cut-off point at 80% of the reference weight for height permits selecting a group with a mortality risk 1.15 times higher than in the whole group. Cross classifying with height for age (cut-off point 90% of the reference) improves the ratio to 1.78, with a much higher sensitivity, but at the cost of an important loss of specificity. Only 28% of the children are not considered at risk, and a selected child has a probability of dying during the next 2 years of 0.06; this is a higher risk indeed but remains low from a statistical or operational point of view. There is not enough information available to assess the potential improvement gained from, as well as the cost entailed by, the introduction of velocity measurements (height or weight) as a screening tool in that population.

In Kasongo, standardized growth decelerations of weight for age of a magnitude of -1 SD unit or more over a period of 3 months are fairly frequent—about 6% above 24 months, more than 20% between 6 and 11 months—and robust to either errors in measurement or determination of age. They are more promising than static measurements as a tool for the identification of children at risk (7). Looking for linear growth decelerations among children who do not present decelerations in weight growth does not increase sensitivity (for detection of death within the next 100 days) if the measurements are made after the age of 24 months, whereas it does reduce specificity. Between the ages of 6 and 24 months, an increase in sensitivity can be obtained, from 56 to 77%, but again with a reduction of specificity, from 54 to 38%.

The formulation of concrete recommendations in this regard requires additional research on the characteristics of the screening criteria (sensitivity, specificity, predictive value at various cut-off points, etc.) in different situations, on the possibilities of operationalizing velocity measurements in community settings, and on the possibilities of intervention on children identified as at risk. However, the actual choice of an indicator of faltering or faltered growth is probably less important than whether it is used in conjunction with information on the child’s recent or present morbidity and its socioeconomic environment. This might enable us to discriminate those episodes of growth faltering for which an intervention could be implemented, aiming at diminishing exposure and increasing resistance to nutritional or infectious stresses when the child is particularly vulnerable.
What is the value of the prevalence of stunted children as a tool for the evaluation or monitoring of the health of a population of children? The data presented do not permit more than speculation; still, the measurement of height deficits seems to be useful.

If both stunting and mortality are different outcomes—often successive but without direct causal relationship—of a succession of stresses on the children's health, then the measurement of the height of children in a community is an even more valuable tool for evaluation. Indeed, the measurement of the prevalence of stunted children then becomes an operationally fairly feasible, be it indirect, measurement of poverty-related disease frequency and of the inability of rapid recuperation. Stunting may not be a "disease" per se or a "cause of death"; it probably only results from the adaptation of the body to an incomplete recuperation after exposure to various stresses in an unfavorable environment. The measurement of the prevalence of stunted children in a community does not tell us whether this adaptation was obtained with a high or low cost of human lives; we need other indicators for that. However, it provides us with a tell-tale sign that such an adaptation was necessary, because aggressions were not avoided and/or the children were not able to overcome their debilitating effect.

The prevalence of stunted children appears to be a good overall indicator of the health status of a community of children, as are mortality levels. Both have similar limitations and certainly may not be substituted for socioeconomic or health service functioning indicators, which may be more directly relevant for decision making. But measurement of prevalence of stunted children does have some advantages: it is a cheap measurement, and in situations in which a number of frequent "biological" causes of mortality such as malaria or tetanus are under control, it might be a sensitive indicator showing that the effects of poverty on the child's health have not been completely overcome.

ACKNOWLEDGMENTS

This work was supported in part by grants 3.4541.85 of the F.N.R.S. and 22.363 of the N.F.W.O., Belgium.

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**DISCUSSION**

*Dr. Tanner:* I feel rather bad that it rests with me to make some methodological or technological criticism, particularly in respect to this extremely interesting and important paper with which I agree almost entirely, apart from this technical point, which is nonetheless an important one. If you consider the change in Z score with age as a measure of the probability of velocity in terms of distribution (that is, the velocity centile as usually understood), it is biased and therefore inaccurate. It is a very easy one to get wrong. A lot of people have fallen into this trap, but it is perfectly well known and is in the auxology textbooks (1). So you must look at it in terms of velocity itself.

*Dr. Van Lerberghe:* I am not familiar with velocity measurements, and I was uneasy about the way in which to express them. To me, this was an operational measure enabling us to see whether there was a difference between the overall population and the children who died. I agree that it may be a biased measure of the probability of velocity, but I was only looking for something different occurring in the children who died, in the period before they died, compared to the overall population. It is this difference that is important.

*Dr. Tanner:* I agree with you, but if you start comparing the differences between the dying and the nondying between ages, you might draw the conclusion that certain ages are worse than others; then you are in trouble.

*Dr. Martorell:* Dr. Van Lerberghe, in relation to mortality, there is a third study you may want to include, by Peter Heywood in New Guinea (2). He performed anthropometric measurements in over 1,000 children with a follow-up of 18 months. When the initial status in terms of weight for height was adequate, being stunted did not seem to make a difference in terms of mortality. There was, however, a strong interaction between stunting and wast-
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The group with the highest mortality was the one that was stunted and wasted.

You mentioned the influence of maternal size and maternal height on child mortality. I think that is another area we might want to look at to get a full picture of what stunting means in terms of mortality. We studied maternal height and child survival in about 400 Mayan women in Guatemala (3). Maternal height was strongly related to infant mortality. When we divided these women into terciles of height, the infant mortality for the tallest was about 200 per 1,000; for the midtercile, it was about 150 per 1,000; and for the shortest, it was about 100 per 1,000. So there is a strong relationship between maternal height and infant mortality. Could you comment on this?

Dr. Van Lerberghe: Both maternal height and weight have been used by Chen as indicators of maternal nutrition. It is interesting to see that this indicator of maternal nutrition does not make any difference to the child’s risk of dying if the child is not severely stunted. The risk ratios for children above 85% of the reference are 0.99 for height and 0.92 for weight, whereas if you look at children below 85% of the reference, then the risk ratios are 1.58 and 1.25. So there seems to be an association, but again this should really be analyzed in the context of the social stratification within the population. I am not familiar with the Matlab population, but I wonder whether it really is one homogeneous population or whether it is composed of a number of subpopulations in which the environmental associated risk is different. In the Zaire situation, the physical and nutritional environments of all the children in the population appear to be identical; there are no gross differences. We do not have any data on maternal nutritional status, but my guess is that it would not be an indicator of differences in the environment of the child and that it would not interfere very much with child mortality. However, this is totally speculative.

Dr. Tomkins: I should like to hear your opinion on the duration of follow-up. What worries me about the interpretation of these studies is that the Matlab study by Chen (4) was over a 24-month period; the analysis by Bairagi (5) was over a 12-month period. Chen showed a stepwise threshold; Bairagi showed a linear relationship, at least for two of the seasons. Your study was over 100 days, I believe. Is this difference in duration of follow-up important if we are going to make recommendations for the future? I wonder if you could comment on what bias might be introduced by looking at children over different periods of time?

Dr. Van Lerberghe: The choice of length of follow-up depends on a number of things. One of the reasons we chose a relatively short follow-up period is that we were looking for an operational tool to identify the children for whom we could do something on a short-term basis. I have very incomplete data on long-term risk evaluation; you will find some in the chapter. It seems that the association in Kasongo, at least, is no different for long- and short-term follow-up. As far as velocity is concerned, the mixed long- and short-term follow up (in Chen’s paper the long- and short-term follow-ups are also mixed) shows significant differences, but the differences are smaller than for the short-term follow up. That means that the nearer the measurements are to death, the bigger is the difference compared to what was expected.

Dr. Tomkins: I think that this is a real problem, and I am glad that you differentiated between different communities. Another question is whether there could be a bias between these studies because of the different causes of death? If I understand correctly, 54% of the children in your study died from measles. It is very interesting to realize that nutrition itself may not be a particularly severe risk factor in urban-type measles: work from Guinea Bissau (6) and from South Africa (7) emphasized that it might be a severe problem in relatively
well-nourished children. I wonder if you had an opportunity to look at the difference between height for age and subsequent risk for nonmeasles death, as opposed to measles death, because I do not think that 54% of the children in Matlab died from measles. What do you feel about the potential bias introduced by different causes of death?

**Dr. Van Lerberghe:** I cannot answer your question as far as height is concerned, but for weight, weight for height, arm circumference, and arm circumference for height, we did see the same pattern in measles and nonmeasles deaths. It might be different for height.

**Dr. Gopalan:** Don't you think that the level of health care could influence the relationship between stunting and mortality? Of course, it is to be expected that in many of the poor population groups where there is a great deal of stunting, access to health care is also poor, but even in the most extreme cases of kwashiorkor, we now know that fatalities are not inevitable. Even in kwashiorkor, with proper treatment the mortality rate is now very low. The effort that is needed to reduce mortality is clearly very different from the effort needed to reverse stunting. Many developing countries may be able to afford the first but not the second. The pool of stunted children may actually increase because of the reduction in child mortality. It could very well be that health care in one location was inadequate with, as a consequence, stunting and mortality going together, and this may not apply in another location. Could you comment on the level of health care in the area where you were working?

**Dr. Van Lerberghe:** There are a number of questions involved. One could speculate that the level of stunting is a good indicator of whether there is integrated health care or not. When health care is restricted to a few single actions such as vaccination and oral rehydration and maybe two or three other simple things, I don't think that the health care system will thoroughly influence the level of stunting because it will not consider a number of minor aggressions that end up debilitating the health of the child. That is why the prevalence of stunted children is probably a good indicator of dysfunctioning of the health system. On the other hand, the efficiency of the use of anthropometric data within the context of an integrated primary health care system (curative care, preventive care, promotive care, and so on) is bound to increase very considerably for purely mathematical reasons. The predictive value of a screening test, if you use anthropometry as a screening instrument, increases if the prevalence of what you want to predict increases. If the screening is conducted within the context of general curative care, then the chance is high that anthropometry will be related to a pathological situation that requires intervention; this automatically increases the efficiency of the screening instrument. If you have got a good health system, the prevalence of stunted children should go down, and the usefulness of anthropometry should rise.

**Dr. Gopalan:** I don't want to continue this discussion, but I think the statement that the level of stunting is a reflection of the efficiency of the health system may be an oversimplification; it may be a reflection of socioeconomic deprivation; I wish it were that simple. Stunting is not a disease like goiter or keratomalacia; there is no vaccine against stunting. On the other hand, the reduction of mortality may be much more closely related to the efficiency of the health care system.

**Dr. Van Lerberghe:** This is true in extreme situations with very high levels of mortality and very high levels of stunted children. I don't believe that this remains true in an intermediary situation, when single causes of death have been eliminated—I mean those causes of death for which very simple technological measures exist. For instance, if there is a high mortality from tetanus and the health system starts vaccinating pregnant women, then the best indicator for the functioning of the health system will be the reduction in mortality.
level and not the reduction in stunting. If such causes of death have been eliminated, the
causal structure of mortality changes and becomes more diffuse, the causes of death being
a number of different conditions such as respiratory diseases, chronic diarrhea, and so on,
as in Latin America or some parts of Africa. At that point, the prevalence of stunted chil-
dren becomes an indicator of health system functioning and of socioeconomic status, which
are also interrelated. It doesn’t, however, really identify what goes wrong. It just shows
that something is wrong, and one should start looking for the causes.

Dr. Valyasevi: The main causes of mortality, especially in young children in rural areas
in developing countries, are probably diarrhea and infection of the respiratory tract. These
are thoroughly influenced by the availability of health care, as has been observed in Thai-
land. Before infant health care systems were implemented about 10 or 15 years ago, the
infant mortality rate was very high, about 70 or 80 per 1,000. Up to 3 or 4 years ago, the
preventive and promotive aspects of health care were really not emphasized. Nevertheless,
the infant mortality dropped to below 50. This was mainly because of the availability of
health care in the villages, primarily focusing on curative aspects. This seems to support
Dr. Gopalan’s remark that mortality is rather an index of the availability of health care,
whereas stunting probably reflects the socioeconomic situation. At least, this is my expe-
rience.

Dr. Nabarro: I am concerned that we appear to be making international generalizations
about processes whose nature will vary from location to location. Chen and his colleagues
reported the relationship between children’s anthropometric indices and subsequent mortal-
ity in 1980 (4). Their data have been used by a number of writers to explain the nature of
the relationship and its implications for those making decisions about the allocation of re-
sources to undernourished children. However, workers in Papua New Guinea (8) and your
group in Zaire (9) found quite different anthropometry-mortality relationships. There ap-
ppears to be considerable variety in these relationships, depending both on the interval over
which children’s mortality risks are studied and the location in which the study took place.
I would suggest that both the socioeconomic status of the population and the infections that
are experienced by children are important determinants of the relationship. I consider it
premature to suggest that the prevalence of stunting can be used as a universal index of
health service performance or of the impact of development programs.

Dr. Van Lerberghe: Stunting is only a single measure, and as such it has the same limita-
tions as other single measures. It can certainly not be substituted either for socioeconomic
indicators or for indicators of health service functioning. In my opinion, health service
functioning indicators are much more important because they help us to make decisions
adapted to the local situation. Some indicators might be applicable everywhere; however,
one should avoid claiming that single indicators explain everything, because it is evident
that one cannot evaluate a complex situation just by looking at one single measurement.

Dr. Martorell: Knowing the mechanism is helpful for generalizing. For instance, wasting
may be related to mortality partly through biological mechanisms, impaired immunocompe-
tence and so on, in which case generalization is easier. In the case of stunting, I would
agree with you that it probably reflects many aspects of the socioeconomic environment,
and it is difficult to isolate the mechanisms involved; these need to be investigated in each
location. As a result, one cannot generalize easily concerning the meaning of stunting in
different places.

Dr. Van Lerberghe: If such a mechanism could be suggested for stunting, then I would
certainly speak of a risk factor and of some degree of causal association. In the case of
stunting there are so far no such biological explanations available.
Dr. Golden: I would like to reemphasize the importance, in analyzing this type of data, of breaking it down in terms of cause-specific mortality. In her study of mental stimulation of malnourished children in Jamaica, Sally McGregor was horrified to find that children who were stimulated were dying from increased numbers of accidents, such as traffic injury, burns, etc. If we don’t break mortality down to cause-specific mortality, we might end by reaching quite wrong conclusions. Measles deaths are different from diarrheal deaths and from accidents. We must clean the data in terms of the cause of death.

Dr. Van Lerberghe: It is already very hard to find studies where a reasonable number of deaths are analyzed. In the Bairagi study (5), fewer than 20 children have had height measurements in the months preceding death; we cannot start breaking down 20 deaths by cause and analyzing those different categories. In our study, we had 137 deaths that we could analyze in children between 6 and 61 months. That is still a very small group. Now we can break it down into measles and nonmeasles, or maybe into measles, diarrhea, and “nonmeasles–nondiarrhea,” but with 3-monthly visits you cannot get reasonable morbidity data, and you start having difficulties in identifying the causes of death. If we want to identify the causes of death on a prospective basis, we need a multiround survey with visits every 15 days, but then we get an extremely biased study. There is really no way out unless we use sophisticated designs combining prospective cohort studies with case control.

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