Growth velocity and stunting in rural Nepal

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SUMMARY In a community based study, height and weight increments of 441 Nepali children aged 0–6 years were measured before harvest and six months later and compared with centile standards derived from American children. Low mean growth velocities for height were found only in children under 2 years of age, and for weight during the first 18 months. The mean height for age standard deviation score for the 12–23 months age group was already −2.8 at first measurement. The effect of the initial thinness of the child on subsequent height and weight velocity was reciprocal: thin children seemed to catch up weight at the expense of height.

These results suggest that stunting is caused largely by a reduced growth velocity during the nutrition dependent infantile phase of growth, with some additional impairment and delay in onset of the early childhood phase of growth hormone dependent growth, especially in thin children. Nutritional interventions after the second year of life are unlikely to alter the prevalence of linear growth retardation in poor communities. Growth velocity may be more useful than static anthropometry to assess the impact of such interventions.

The aetiology of linear growth retardation in poor communities remains unclear. Ethnic differences are less important than environmental influences, although there is variation in the ability of different genotypes to cope with hardship. Prevalence rates of stunting are associated with variables such as the age and sex of the child, the frequency of episodes of infection and socioeconomic indicators of household poverty. The importance of seasonal variation in growth rates has also been noted in longitudinal surveys. These studies have analysed static height for age values, whereas growth velocity measurements are more valuable than ‘distance charts’ in the assessment of growth, which is a dynamic process. Growth velocity is independent of the height achieved by a child so it is a sensitive indicator of good or bad health regardless of previous growth delay. The normal linear growth curve has recently been described in terms of an infancy-childhood-puberty model, which suggests that linear growth comprises three additive, partly superimposed mathematical components, an infancy component, which represents the postnatal contribution of nutrition dependent fetal growth, a childhood component, which is controlled by growth hormone, and a puberty component, which is dependent on sex steroids and growth hormone.

The aim of the present study was to measure growth velocities of children during a six months period after harvest when growth rates would be expected to be maximal in a community where stunting is common, and to consider the relative contributions of infancy compared with childhood growth retardation to the process of stunting.

Subjects and methods

BAGLUNG DISTRICT
Baglung District covers 2153 square km of a remote rural area in western Nepal where there are no motorable roads. To the north it is bounded by the Himalayan mountains and to the south by deep river valleys and basins. Most of the population (1985 estimate: 232 000) live in the middle hill region below 2500 m where they produce on terraced hills rice, maize, millet, and wheat in a monsoon climate. Nearly all the households depend on subsistance agriculture, and Baglung is a food deficit area with an average annual household grain production of about eight months. The district is divided into 62 geopolitical subdivisions called panchayats.

DESCRIPTION OF SURVEY
The survey covered all 349 households with children less than 6 years of age in a single panchayat, Resa, situated about four hours walk from the district capital, Kalika. It was planned after discussions with the pradhan panch (mayor) of Resa, representa-
tives from each of the nine wards in the panchayat, and staff from the health post in Resa and the Save the Children Fund clinic in Kalika.

All houses with children under 6 years were visited on two occasions, during August 1985 (monsoon) and six months later during February 1986 (dry season). Wards were surveyed sequentially by teams comprising one health post person, one Save the Children Fund health worker, and a local man who could identify the houses.

Before starting the survey staff were trained in interview techniques; given practice with the completion of questionnaires at houses near the Save the Children Fund clinic in Baglung; and taught how to measure weights and heights using the Salter scales, the baby measuring cradle, and the height frame. Weights were recorded to the nearest 0.1 kg and heights to the nearest 0.1 cm. The head of the household and the children’s parents were told the purpose of the survey and asked for their cooperation. If parents or children were away, they were visited again the next day. The team interviewed the household head or one of the parents; recorded household, ethnic group, and health related data on a precoded questionnaire; and examined and measured all children under 6 years of age.

The survey teams calculated the age of each child to the nearest complete month by checking the month and year of birth with two adults, and by using a calendar table that eliminated the local custom of overestimating age by one month or one year.

**DATA ANALYSIS**

**Age distribution**
The age and sex distribution of the child population surveyed was checked against nutrition surveys conducted nationwide in 1975 and a Baglung nutrition survey in 1979. The proportion of children aged 60–71 months in both Baglung survey samples was slightly lower than expected.

**Nutritional measurements**
The height and weight measurements recorded at each visit were expressed as the percentage of a reference median and as standard deviation scores for height for age, weight for height, and weight for age using a statistical programme based on the National Centre for Health Statistics standards. Differences between weights and heights at each visit allowed calculation of weight and height increments over the six month period between August and February when, being the period after the harvest, nutrition would be expected to be optimal (or nearly so) in this community. These increments were compared with centiles and standards drawn from the Fels study in the USA.

**Data handling and statistics**
The questionnaire data were precoded where pos-
sible for ease and accuracy of collection. The data were double checked for obvious errors of translation before entry into the computer database. Statistical calculations were made using the Apple Statview programme.

Results

A total of 546 children aged 0–71 months were seen before (August 1985), and 479 seen after (February 1986) the harvest; the latter group included newborns from the intervening period. Growth increments were obtained in 441 children (81%). Using the Waterlow classification of nutritional status, 296 children (54%) were stunted (< 90% of the height for age reference median) and 17 (3.1%) were wasted (< 80% of the weight for height reference median) in the survey before the harvest; 228 (51%) were stunted and nine (1.9%) wasted in the survey after the harvest. Stunting prevalences were highest before the harvest for all age groups, and among the poorest households and lowest caste children; they rose sharply between 12 and 23 months of age.

Fig 1 shows the mean standard deviation scores (SD score) for static nutritional measurements (weight/height, weight/age, height/age) for different age groups at each survey visit. There were significant seasonal differences for weight/height and weight/age scores overall (children were thinner before the harvest), but height/age scores showed no

![Diagram](https://via.placeholder.com/150)

**Fig 2** Mean height and weight increment curves for Nepali children compared with increment centiles for American children in the Fels study. (The Fels velocity standards were also established using an incremental time interval of six months.)

![Diagram](https://via.placeholder.com/150)

**Fig 3** (a) The effect of a child's initial nutritional state (classified by the initial weight for height SD score group) on subsequent mean weight and height velocity in the period after the harvest. (b) Oldham's plot for correction of the phenomenon of regression to the mean, using the mean of the initial and final values of weight/height SD score group as a measure of nutritional state.
seasonal change except in the 36–71 month age group. Although the 0–5 month age group appears to be worse after the harvest than before for both height and weight, this is probably artefactual as a result of the relatively low numbers of infants aged 0–5 months measured after the harvest.

The mean growth increment curves of the survey children by age group compared with incremental centiles established in American children are shown in fig 2. If values below the 25th centile are considered arbitrarily to be abnormal, then height increments were abnormally low until 24 months and weight increments until 18 months of age. Increments returned to near the 50th centile by 22 months for weight and 36 months for height.

The influence of the initial state of the child (assessed by weight/height SD score before the harvest) on subsequent height and weight velocity is shown in fig 3(a). Analysis of raw data showed a highly significant relationship between the initial weight/height SD score and height velocity (line of best fit $y=9.36+2.28x+0.44x^2$, $p<0.001$) and there was an inverse but weaker association with weight velocity (line of best fit $y=2.36+0.13x+0.14x^2-0.05x^3$, $p<0.005$).

This association between initial weight/height SD score and growth velocity is complicated however by two potential sources of error, regression to the mean and the uneven distribution of weight/height scores with age, high values of weight for height tending to occur in the youngest age group when both height and weight velocity are highest. The error introduced into these relationships by the phenomenon of regression to the mean was corrected using Oldham's method of plotting the velocities against the average of initial and final values of weight/height SD scores, as shown in fig 3(b), which still shows a significant relationship between the thinness of the child and catch up height velocity ($y=8.98+1.71x+0.48x^2$, $p<0.001$); fig 4 shows the same relationship for each of the age groups 0–11 months, 12–23 months, and 24–72 months of age, again showing this association, especially in the first two years of life.

**Discussion**

Although growth velocity measurement (like most methods of growth monitoring) is of limited clinical value in primary care in developing countries, it has advantages as an epidemiological tool when compared with static anthropometric measurements. Velocity is more time specific than a distance measure like height for age: a normal velocity indicates current good health in children who are stunted from previous causes, and a reduced growth velocity suggests poor health even in children whose growth has not yet faltered appreciably on a distance chart.

Chen has suggested a need for better, simpler and more sensitive epidemiological markers than infant or child mortality rates to assess the impact of primary health care initiatives in developing countries. Growth velocity measurements fulfil these criteria. With standardised centiles for growth increments now available, they can be used to identify not only vulnerable groups but also vulnerable time periods in a particular community.

The results of this study confirm a previous finding that stunting has an early onset in poor communities. The maximal impairment of height velocity seems to occur during the latter postnatal stage of the nutritional component of linear growth, although increments in the first 6 months of life
cannot be assessed from these data. Waterlow reviewed surveys in similar communities where breastfeeding is widespread, and showed that growth rates (assessed only by weight velocity) falter from 3 months of age compared with those of British children,\(^{17}\) although the measurement of velocity at this time is difficult. Further, the onset of the growth hormone dependent childhood component of growth may be delayed compared with children in developed countries, in whom it begins between 6–12 months of age.\(^{11}\) Karlberg et al showed an average delay in onset of five months for children from an urban community in Lahore, Pakistan.\(^{18}\)

The most vulnerable period for impairment of growth rates is between weaning and 24 months. Mean growth velocities after this age in Nepali children were close to the 50th centile for American children. This observation is important if, for economic reasons, nutritional measures are to be targeted selectively as part of a national or international health policy. Overall, nutritional and health interventions after the second year of life are unlikely to alter appreciably the prevalence of linear growth retardation. Seasonally adjusted mean growth velocity rates may, however, be a good indicator of the impact of such interventions in this age group.

Our community based results also show the clear effect of the initial nutritional state of the child on subsequent height velocity, and help to clarify the complex relationship between wasting and stunting. Thin children gained weight faster than their normal peers but at the expense of their height velocity. This supports the hypothesis that the frequency and duration of preceding wasting episodes influence the likelihood and degree of stunting. In Nepal and most developing countries prevalence rates for wasting are highest in the 12–23 month age group and wasting probably plays a large part in the reduction in mean height velocity at this time.

Walker and Golden have shown in a hospital based study that malnourished children need to reach a critical threshold of 85% weight/height before catch up linear growth will occur.\(^{19}\) The mechanism of growth failure in patients with protein energy malnutrition appears to be a deficiency in the generation of insulin\(^{20}\) and somatomedins,\(^{21}\) especially insulin like growth factor 1, which mediates the growth of epiphyseal cartilage.

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References


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