lasting several years are also needed to see if permanent changes in growth patterns can be achieved.

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1 Milner R. Growth hormone 1985. *Br Med J* 1985;**291**:1593-4.
2 Ross M. Production of medically important polypeptides using recombinant DNA technology. In: Gueriguian JL, ed. *Insulins, growth hormone, and recombinant DNA technology*. New York: Raven Press, 1981:33-48.
3 Rosenfeld R, Hintz R, Johanson A, Genentech Collaborative Group. Prospective, randomized trial of methionyl growth hormone and/or oxandrolone in Turner syndrome. *Pediatr Res* 1985;**19**:620.
4 Frazer T, Gavin J, Doughaday W, Hillman R, Weldon V. Growth hormone-dependent growth failure. *J Pediatr* 1982;**101**:12-5.
5 Van Vliet G, Styne D, Kaplan S, Grumbach M. Growth hormone treatment for short stature. *N Engl J Med* 1983;**309**:1016-22.
6 Grunt J, Howard C, Doughaday W. Comparison of growth and somatomedin C responses following growth hormone treatment in children with small-for-date short stature, significant idiopathic short stature and hypopituitarism. *Acta Endocrinol (Copenh)* 1984;**106**:168-74.
7 Gertner J, Genel M, Gianfredi S, *et al*. Prospective clinical trial of human growth hormone in short children without growth hormone deficiency. *J Pediatr* 1984;**104**:172-6.
8 Marshall W. The relationship of variations in children's growth rates to seasonal climatic variations. *Ann Hum Biol* 1975;2:243-50.
9 Barratt T, Broyer M, Chantler C, *et al*. Assessment of growth. *Am J Kidney Dis* 1986;7:340-6.
10 Preece M. Experience of treatment with pituitary-derived hGH with special reference to immunological aspects. In: Milner RDG, Flodh H, eds. *Immunological aspects of human growth hormone*. Oxford: Medical Education Services, 1986:9-16.
11 Preece M. Creutzfeldt-Jacob disease: implications for growth hormone deficient children. *Neuropathol Appl Neurobiol* (in press).

A better way to detect growth failure

Most doctors are familiar with growth charts for height and weight that have a 10th centile (this means that 10% of normal children have a height or weight as low as or lower than that). Such charts are called distance charts. Fewer doctors, however, seem to be familiar with velocity charts, which show how fast a child is growing. Yet these charts are more important because a single point on a distance chart is not a good way of determining the normality of growth, which is an active process.

The difference between two successive points on a distance chart shows how much a child has grown and can be turned into a velocity by dividing by the time interval. This value can then be entered on to a velocity centile chart,[1] which—just like a distance chart—is marked with centiles. The centiles on a velocity chart are specific to a particular time interval, usually one year. If measurements are taken at shorter intervals an annual growth velocity can be calculated—but the centiles may be as much as twice as wide as those for an annual increment because the measurement errors have been magnified and there may be seasonal effects on growth.

Points on a distance chart for a single child are highly correlated: a child who is small on one occasion will almost certainly be small on the next. A normal child will thus tend to follow a particular centile, and deviations of growth from a centile are difficult to detect by eye. This is one reason for calculating velocity in the clinic because there is a difference when successive points are plotted on a velocity chart: in fact, successive velocities are hardly correlated at all. A child whose velocity was consistently at the 30th centile for years would turn into a dwarf, and one with a velocity consistently on the 70th centile would become a giant. Thus the probability of two successive velocities in a normal child falling on the 25th centile is only around $0{\cdot}25 \times 0{\cdot}25$ (0·0625)—that is, only 6·25% of healthy children will grow as slowly as this over the whole of a two year period.

Because successive velocities are independent one cut off may be taken as an indication for immediate action and another as a warning signal. If a third centile velocity is chosen for immediate action the chances of investigating a normally growing child are only 3%. A suitable warning limit could be at the 25th centile. A child with a velocity below this but not as low as the action limit should be followed up and if the next velocity also falls below the warning limit action should be taken. A chart incorporating this idea is available.[2]

Powerful treatments for altering growth rates are increasingly available, and not investigating healthy children is as important as detecting failing growth. Careful interpretation of growth data will help in both directions.

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1 Tanner JM, Whitehouse RH. Longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Arch Dis Child* 1976;**51**:170-9.
2 Brook CGD. Earlier recognition of abnormal stature. *Arch Dis Child* 1983;**53**:840. [Available as Middlesex hight velocity assessment chart from Castlemead Publications, Ware, Herts SG12 9PY.]

Acquired cystic disease of the kidney: serious or irrelevant?

That the kidneys of some patients with end stage renal failure develop multiple cysts has long been recognised,[1] but only in 1977 did Dunnill and others coin the term "acquired cystic disease of the kidney."[2] They performed necropsies on 30 patients who had been treated with long term haemodialysis (none of them because of primary cystic diseases of the kidney) and discovered that 14 had multiple cortical cysts. Since then many studies have described such cysts[3-15]: these range in size from 1 mm to over 2 cm, occur in both the medulla and the cortex, and may replace much of the parenchyma. But do they matter? Are they a pathological irrelevancy or an important clinical problem? The latest evidence suggests thay they probably do matter.

Acquired cystic disease of the kidney has now been described in patients having haemodialysis or peritoneal dialysis and becomes increasingly common and severe as the duration of dialysis increases. It is more common in male patients and is unrelated to age.[9 16] Whether some primary renal diseases are particularly associated with acquired cystic disease of the kidney is not clear.[2 14] Histological studies show that the cysts communicate with both tubules and glomeruli and may be lined with atypical (possibly neoplastic) cells.[5 11 14] Solid tumours occur, and distinguishing between adenoma and adenocarcinoma is difficult.[2] Suggested aetiologies include a response to ischaemia,[7] occlusion of the tubules by fibrosis or oxalate,[2] the effect of toxins absorbed from