The Andrea Prader Prize, established with the support of Pharmacia & Upjohn, Stockholm, as a Leadership Award, is given to a member of the European Society for Paediatric Endocrinology (ESPE) in recognition of achievements within the field of paediatric endocrinology. At the last meeting of the ESPE in Brussels (Belgium) in 2000, the prize was awarded to Prof. Charles G.D. Brook.

C.G.D. Brook, Emeritus Professor of Paediatric Endocrinology at University College London, qualified at Cambridge and St Thomas’s Hospital, London. After resident appointments at St Thomas’s and Great Ormond Street Hospitals, he became a Research Fellow and Lecturer at the Institute of Child Health, first with Prof. O.H. Wolff and then with Prof. J.M. Tanner. After a year in the Kinderspital Zürich with Prof. A. Prader, he was appointed to the Middlesex Hospital in 1974 as Consultant Paediatrician with an interest in paediatric endocrinology.

In 1983, he was appointed Senior Lecturer in the University of London, subsequently promoted to Reader and, finally, to Professor of Paediatric Endocrinology in University College London in 1989.

In 1994, he set up the London Centre for Paediatric Endocrinology jointly between Great Ormond Street Hospital and University College London Hospitals at The Middlesex Hospital.

Prof. Brook retired from National Health Service practice and from University College London on 31st March, 2000.

Summary of Andrea Prader Lecture 2000

The Harmony of Growth
‘Differing in size, in note, in weight,
Yet, small or great,
We harmonise’

‘So runs the inscription on the bells of Colchester Town Hall: one could say the same of animal growth’ [1]. The description of the infancy-childhood-puberty model of human growth [2] offered an opportunity to identify the controlling mechanisms of the human growth process.

The infancy component, which runs from fetal life to the first 8 months of postnatal life, is nutrition-dependent and hormones play a small part: the hypothyroid infant, the anencephalic infant and the infant with holoprosencephaly are all of much the same size. The infant of the diabetic mother and the infant overfed from birth is large; placental insufficiency and infantile malnutrition can both cause permanent stunting if they are of sufficient duration and severity.

Growth hormone (GH) deficiency leads a yearling child to follow the infantile curve of growth and the GH-amplitude modulation of childhood growth [3] is now well accepted. The shape of the asymptotic curve relating growth velocity to GH secretion explains the dose-response relationships of growth to exogenous GH [4].

The evidence that short stature per se is disadvantageous to children or adults is wanting [5] so clinicians need to treat GH deficiency but be wary of overenthusiastic exhibition of a substance with powerful metabolic effects [6]. They may just be stigmatizing their patients.
The mechanism of the mid-childhood growth spurt remains elusive, although adrenarche is probably central to its understanding. The hypothalamo-pituitary-gonadal axis is fully functional in the fetus but the mechanism whereby it is inhibited in childhood and reappears at puberty is not known. Experiments inducing puberty with pulsatile gonadotropin-releasing hormone demonstrated its dependence on such secretion [7] and its iterative nature is obvious in clinical practice: small doses of sex steroids induce the gonadotrophic effects of puberty. The differential timing of the puberty growth spurts in boys and girls explains the differences in the adult heights of men and women which depends not on the growth in puberty, which is fairly uniform, but on the amount of childhood growth which precedes it.

Thus do the phases of growth harmonize to effect adult stature?

References