Growth of children with cancer

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SUMMARY

GROWTH OF CHILDREN WITH CANCER

Between 1982 and 1988 the divisions of oncology and endocrinology of the Department of Pediatrics conducted a prospective and longitudinal investigation into the growth of children with cancer. At regular intervals of 3 months and during a period of 2 years, several anthropometric measurements (14 in all) were performed and the development of a number of secondary sex characteristics was studied. In addition an X-ray of the left hand was made each year. 104 patients entered the study at diagnosis and a further 26 at completion of therapy.

The two reliability studies that were conducted revealed some differences in precision among the observers, as well as a number of systematic errors concerning some anthropometric variables. However, the results of the reliability studies are in line with the results of similar studies. Admittently, further analysis was restricted, to the measurement results of height, weight (for height), upper-arm circumference, armspan, sitting height and head circumference.

The anthropometric data were transformed into z-scores with a contemporary, cross-sectional study (Oosterwolde) as reference; this enabled us to compare different groups of patients of varying age and sex. A growth-curve model for the series of 9 z-scores obtained during 2 years was proposed, based on the parabola of closest fit. This enabled us to establish changes of growth rate in patient groups differing from each other, or from a group of healthy children. Data concerning these healthy children were derived from the Nijmegen mixed-longitudinal growth study. The children from Oosterwolde proved to be, on average, larger than the children from Nijmegen. The application of the proposed model to the Nijmegen data, confirmed the theory (except in the case of sitting height). Our method to establish growth-rate deviations, therefore, appeared to be valid.

Analysis of the measurement results concerning 96 patients at diagnosis, showed a normal height, sitting height and midparent height. The analysis did reveal a long armspan (in the case of leukaemia patients and of patients with a solid tumor), but this was not considered to be conclusive evidence for the suggestion that rapid growth and tall stature are associated with cancer. Also, the nutritional status of patients with a solid tumor at diagnosis proved to be less than optimal.

Of the 130 patients who entered the study, 79 could be evaluated for analysis of the longitudinal data. They were stratified into 8 groups on the basis of the

presumed growth-retardation risk of the treatment they got (groups 1-6) and of the possibility of catch-up growth after completion of treatment (groups 7 and 8). Patients treated with high doses of cranial irradiation (for brain tumors) showed a persistent growth retardation for height, sitting height and armspan. Less retardation for these variables was found in patients treated for acute lymphoblastic leukaemia (ALL). Armspan appeared to be more affected by growth retardation than sitting height. No differences were found between ALL patients that did receive cranial irradiation and those that did not. Therefore we think that the retardation is caused by the medication (cytostatics and corticosteroids) and not by the cranial irradiation. A catch-up growth for e.g. height could be established after completion of therapy. Weight for height growth was excessive during treatment, particularly in patients who received dexamethasone. The height growth of patients with a Wilms' tumor was only retarded in the 6 months after diagnosis, which was probably caused by a poor nutritional status at diagnosis. Patients treated with high doses of cyclophosphamide and/or methotrexate showed no retardation at all. No abnormal relationship between height and sitting height could be established in patients who received irradiation of the spine (brain tumor patients and Wilms' tumor patients). Possibly the follow-up period of 2 years is too short.

In 40 children with ALL, followed up to 5 years after diagnosis, 163 bone age (BA) assessments were performed according to the Tanner-Whitehouse II method based on 20 bones. The development of BA in relation to calendar age was retarded during treatment and made good completely after cessation of therapy. The BA retardation thus parallels the height retardation in these children, preserving the potential of unimpaired height growth. No differences were found between patients that did receive cranial irradiation and those that did not. Again we think the cytostatics and/or corticosteroids play a causative role.

The pubertal development was investigated in 43 children who were older than 8 years at diagnosis. Eleven of these children showed a late or interrupted pubertal development. No premature onset of puberty was noted.

The results of the present study indicate that it could be worthwhile for future researchers to study growth changes during very short periods of time using knemometry. This would enable them to differentiate more accurately the potentially adverse effect on growth of the separate components of the treatment given. In order to reveal the pathogenesis of the growth retardation, hormone levels will have to be studied. The possibility of measuring growth hormone in urine samples gives the opportunity to investigate growth hormone release at various times during the treatment of an individual patient.