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THYROID AND GROWTH

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INCREASE in length of an organism takes place in the early part of its life, and depends upon many factors. The harmonious relationship among these factors produces the normal and constant increase in length which is terminated when ossification is completed at the epiphyseal zones of growth. With the cessation of ossification and union of diaphyses and epiphyses the morphological and physiological maturity of the organism is usually reached.

The factors concerned with growth and maturity have been pointed out by many authors (1–11). It is difficult to establish a classification of these factors because there is complex interaction among them. This fundamental 'substrate,' by which is meant the inherent capacity of the cells of the organism to divide and produce new elements, has intrinsic characteristics due to hereditary factors which condition growth to a certain extent; and it is the 'substrate' upon which other factors operate.

Some factors, such as diet, may ultimately accomplish increase in mass by supplying the cells with the material necessary for their growth and division. Vitamins and hormones regulate cell metabolism and are responsible for growth and differentiation. We have to distinguish, therefore, between simple increase in mass such as can be produced by excessive diet and increase in mass resulting from growth and differentiation in which vitamins and hormones are involved.

Alteration of the capacity of the substrate can be illustrated clinically in cases of retardation of growth in the course of chronic disease. This interference with growth can be seen in the so-called zones of cessation of growth in the bones (12) and in the acceleration and retardation of ossification (13–17). The existence of a definite minimum effective dose and of a limit of maximum stimulation beyond which there is no further growth effect emphasizes the impor of the substrate.

The experiments of Smith (11) demonstrated sation of growth after hypophysectomy wit sumption of growth following administration c tive growth factor of anterior pituitary origin a secondary cessation of growth on discontinuan such treatment. Also, in accordance with the wo Smith, when thyroid is given together with pitu growth hormone, growth is fostered more tha pituitary alone.

It is our purpose, in this paper, to show sta changes, in individuals of 20 years and under, ically manifested as a function of the thyroid.

In the clinical field, the early descriptions of tinism stressed the retardation of growth as an es tial feature of the syndrome; and as time has pa we have come to appreciate the relationship of fa of growth to thyroid deficiency. This explana has been borne out further by the study of bon velopment in acquired hypothyroidism of the juv type in which it has been shown that there is reta tion of epiphyseal closure and of development in bones of the wrist.

The opposite phase of the question—namely, possibility of clinical overgrowth as the result of peractivity of the thyroid—primarily concerns u this study (18–23).

CLINICAL MATERIAL

We selected ~~all~~ the cases of definitely proved ophthalmic goiter in our juvenile group, that is all s patients of 20 years or under. There were 121 ca 13 boys and 108 girls. For comparison we have serted a few cases of juvenile hypothyroidism wh we have had occasion to observe, 1 boy and 7 g From the 121 cases we exclude the boys for simpli in graphic representation and 3 cases with indepe ent chronic disease, thus leaving 104 cases with

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