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شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

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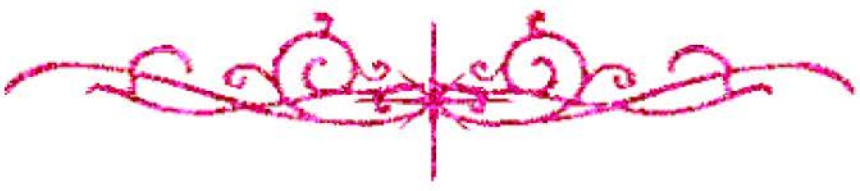

بالرسالة صفحات لم ترد بالأصل

قـالـوا سبحانـك

لاَ عِلمَ لنا إلا ماَ علَمتنا إنكَ أنت العَليمُ الحَكيم

سورة البقرة آية ٣٢

صدق الله العظيم

EVALUATION OF GROWTH HORMONE DYNAMICS IN INSULIN DEPENDENT DIABETIC CHILDREN

Thesis

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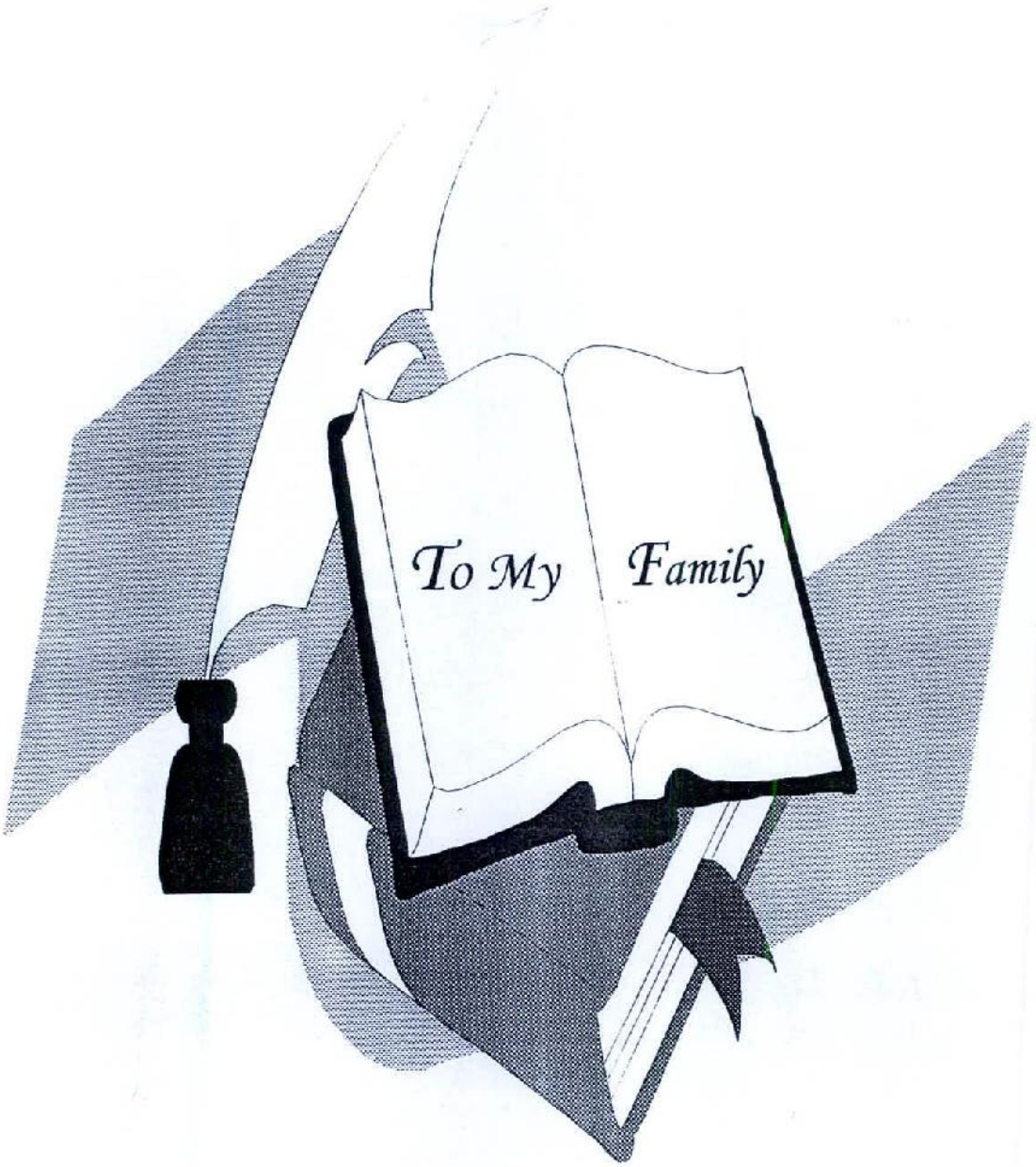
To My Family

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INTRODUCTION

Introduction

Growth

Growth is a complex, dynamic process, which is only partially understood. Growth is characterized both by a coordinated, controlled increase in size and by an increase in physical and mental abilities and functions[1].

It is well recognized that growth is seriously impaired in cases of very poorly controlled diabetes. What is less certain, however, is whether diabetes influences growth or limits adult height in children treated conventionally in whom the disease is fairly well controlled- that is, most cases today[2].

The growth, differentiation and development of the body structures depend both upon intrinsic regulators within the individual and upon extrinsic influences. Among the extrinsic factors are the supplies of building materials and oxygen necessary for growth and the ability of the body to assimilate and transport them. Disease processes impairing the cardiovascular, pulmonary, gastrointestinal or renal systems may exert an unfavourable influence on the growth of the body as a whole or upon some part of it. The pattern of "genotype" laid down in the fertilized ovum determines the plan for the future growth, development and biologic constitution of the individual. This is considered the intrinsic regulator that govern the orderly pattern

of growth and cellular differentiation from the time of "zygote" formation to full adult maturity[3,4].

In order to achieve optimal condition for skeletal growth an adequate intake of nutritional elements and vitamins as well as normal hormonal balance is required. The final body length is thus dependent on the nutritional and hormonal status[5].

The infancy-childhood- puberty growth model suggests that infancy component is primarily controlled by nutritional factors, the childhood component by growth hormone and puberty component by sex steroids. Such a conceptual model though simplistic if taken literally, does allow the effect of different insults that may affect growth to be studied more effectively[6].(Fig.1)

Many hormones influence skeletal and somatic growth besides growth hormone (GH), insulin-like growth factors (IGFs) and insulin. These include thyroxine, androgens, oestrogens and glucocorticoids[7].

A complex cascade of events contributes to normal growth. Two hypothalamic peptides, the growth hormone- releasing factor and somatostatin, produce an ultradian and circadian rhythm of GH, which then binds to its binding proteins (GHBP) and then to its receptors on the cells, affecting the generation of IGF-I, which

in turn binds to its binding proteins and stimulates its receptors in the target cell[8].

There are various opinions as regards the effect of D.M. on growth.

Tattersall et al[9] found that in patients with type 1 diabetes there was a decrease in adult height compared with their identical non-affected twins. Growth delay was also observed by Jivani et al[10] and Edelsten et al[11], while according to Birkbeck[12], Rudolf et al[13], and Jackson[14] children with a fairly high degree of metabolic control showed normal growth rate.

Salardi et al[2] reported that there is some interferences in the growth rate of children and adolescents with diabetes and that they are evident in particular as a decrease in pubertal spurt independent of metabolic control. These interferences do not seem to have a significant influence on adult height[15].

Growth failure in poorly controlled diabetes may not be solely the result of disturbances in GH/IGF-I axis. For example, protein metabolism is altered in hyperglycemic diabetics receiving conventional treatment. Characteristically, hyper-aminoacidemia induced by ingestion of protein is exaggerated in the diabetic because of diminished uptake of amino-acids by muscle[15].