PUBERTY

THESIS SUBMITTED IN PARTIAL FULFILMENT

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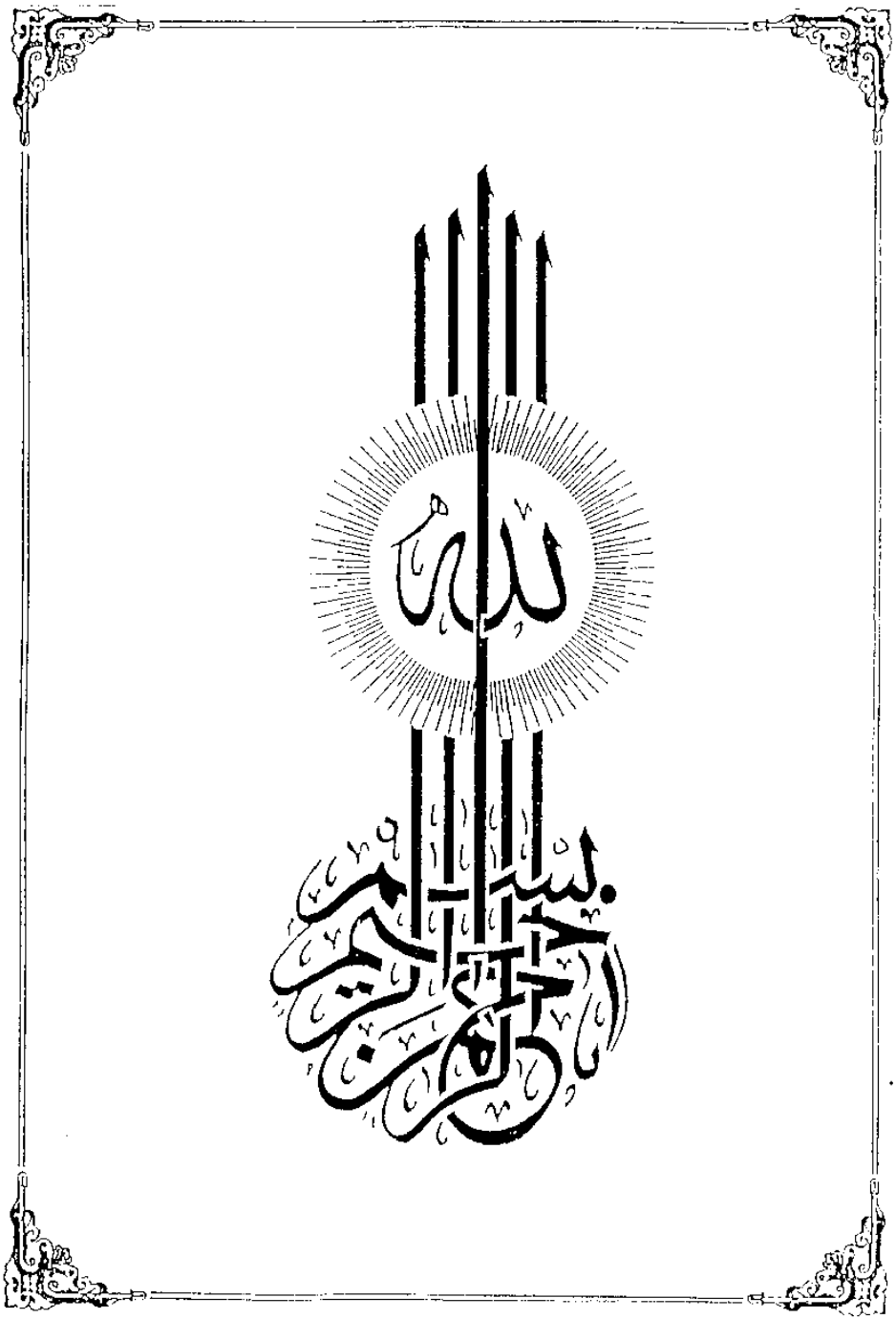
A C K N O W L E D G E M E N T



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C O N T E N T S

I N T R O D U C T I O N

INTRODUCTION

Puberty is the time of greatest sex differentiation since the early intrauterine months. There are changes in the reproductive organs, and secondary sex characters in body size and shape, in the relative proportions of museles, fat and bone and in a variety of physiological functions (Tanner, 1978).

There is great variability both in the ages at which these processes begins and end, and in the sequence in which they occur (Masters, et al., 1979).

The onset of puberty is associated with alterations in the level of circulatory gonadotropic and gonadal hormons (August, 1972).

The period of puberty is a critical period and it is the task of the pediatrician to understand well the problems of that period.

A I M O F T H E E S S A Y

AIM OF THE ESSAY

The aim is to review the physical and endocrinal changes which occur during puberty. Current mechanisms by which puberty may be initiated will be explained.

Aberration of sexual maturation will be discused A light will be thrown on procedures for recognition, definition evaluation of these abnormalities, and treatment if possible.

PHYSIOLOGY OF PUBERTY IN MALE

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Puberty is that period of development which begins with the first sign of pubertal changes and ends with the termination of growth (Prader, 1975).

The earliest recognizable sign of puberty is a more rapid rate of testicular enlargement, this occurs about one year before all other secondary sex changes (Prader, 1975).

The testes has two distinct but related function, both of which are under adenohypophysial and hypothalamic control; these are:

1. Production and storage of viable spermatozoa.
2. Synthesis and secretion of the androgenic hormone, testosterone.

These two major functions of the testes are segregated anatomically with androgen biosynthesis occuring in Leydig cells and spermatogenesis in the seminiferous tubules (Bardin and Paulsen, 1981).

The anterior pituitary participates in the control of both of these functions through its secretions of the gonadotropins, luteinizing hormone (L.H) and Follicle stimulating hormone (FSH). The anterior pituitary is in

turn regulated by multiple parts of the central nervous system which are regulated via the hypothalamic secretion of gonadotropin-releasing hormone (Bardin and Paulsen, 1981).

The hypothalamus and gonadotrapin-releasing hormone:

-Recent studies has emphasized the complexity of the microvasculature between the hypothalamus and the pituitary. The intimate association of the pituitary gland with the central nervous system offered by these blood vessels provides a pathway for releasing hormone from the brain to control anterior pituitary function. Although many parts of the brain may influence reproductive function, the hypothalamus may be viewed as the final common pathway through which the control of gonadotropin secretion and behaviour are mediated (Bergland, 1978).

Testosterone and other sex steroids regulate gonadotropin secretion and reproductive behavior by their action on the brain. A series of studies on male animal using testosterone implant into the brain suggest that the medial basal hypothalamus is important for tonic gonadotropin secretion. Destruction of this area in the brain leads to decresed luteinizing hormone only and testosterone secretion as well as to testicular atrophy (Silverman, 1979).

This portion of the hypothalamus contains a series of peptidergic neurons capable of secreting the hypothalamic hormone (Gn R.H) (Negro-Vilar, 1979).

Leydig cells and testosterone:

The major site of testosterone synthesis in man is the Leydig cells. These are polygonal cells in the interstitial areas which make up about 5% of total testicular volume (Ahmad, 1969).

Leydig cells differentiate and secrete androgene during the seventh week of fetal life in the human (Suteri and Wilson, 1974).

After birth eydig cell continue to secrete androgens for several months, and then revert to a relatively undifferentiated state until they are activated again at time of puberty coincident with the increase in plasma and urinary Gonadotropin-releasing hormone and gonadotropins. Prior to puberty, leydig cells are very responsive to human chorionic gonadotropin (hCG) and the magnitude of testosterone response does not depend upon age. Concomitant with the onset of puberty there is an increased sensitivity of leydig cells to hCG stimulation even before the rise of basal serum testosterone level (Forest, 1979).

Androgen action:

The biologic action of testosterone and its metabolites have been classified according to site of action. All effects that relate to growth of male reproductive system or to development of secondary sexual characteristics have been termed the "Androgenic action" while the effects on the somatic tissue such as liver, kidney, bone and musele have been termed "Anabolic action" (Bardin and Paulsen, 1981).

Spermatogenesis:

It is the process by which spermatozoa are formed. It does not begin till puberty and continous throughout adult life, decline in old age. The seminiferous epithelium contain sertoli cells, each of which extend from the basment membrane to the lumen of the seminiferous tubule. The developing germ cell lie along the glycogen-containing sertoli cells and are nourished by them as they grow from spermatogonia on the basement membrane to become in turn primary spermatocytes, secondary spermatocytes, spermatids and finally spermatozoa (Keele, 1982).

The integrity of the hypothalamic median eminence, a normally functioning pituitary gland and the optimal activity of the testicular leydig cells are essential for

normal development and maintenance of spermatogenesis. All of these structure form an elaborated system of neuro-endocrine integration (Smith and Davidson, 1967).