CHANGING ASPECTS IN CRYPTORCHIDISM

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BY

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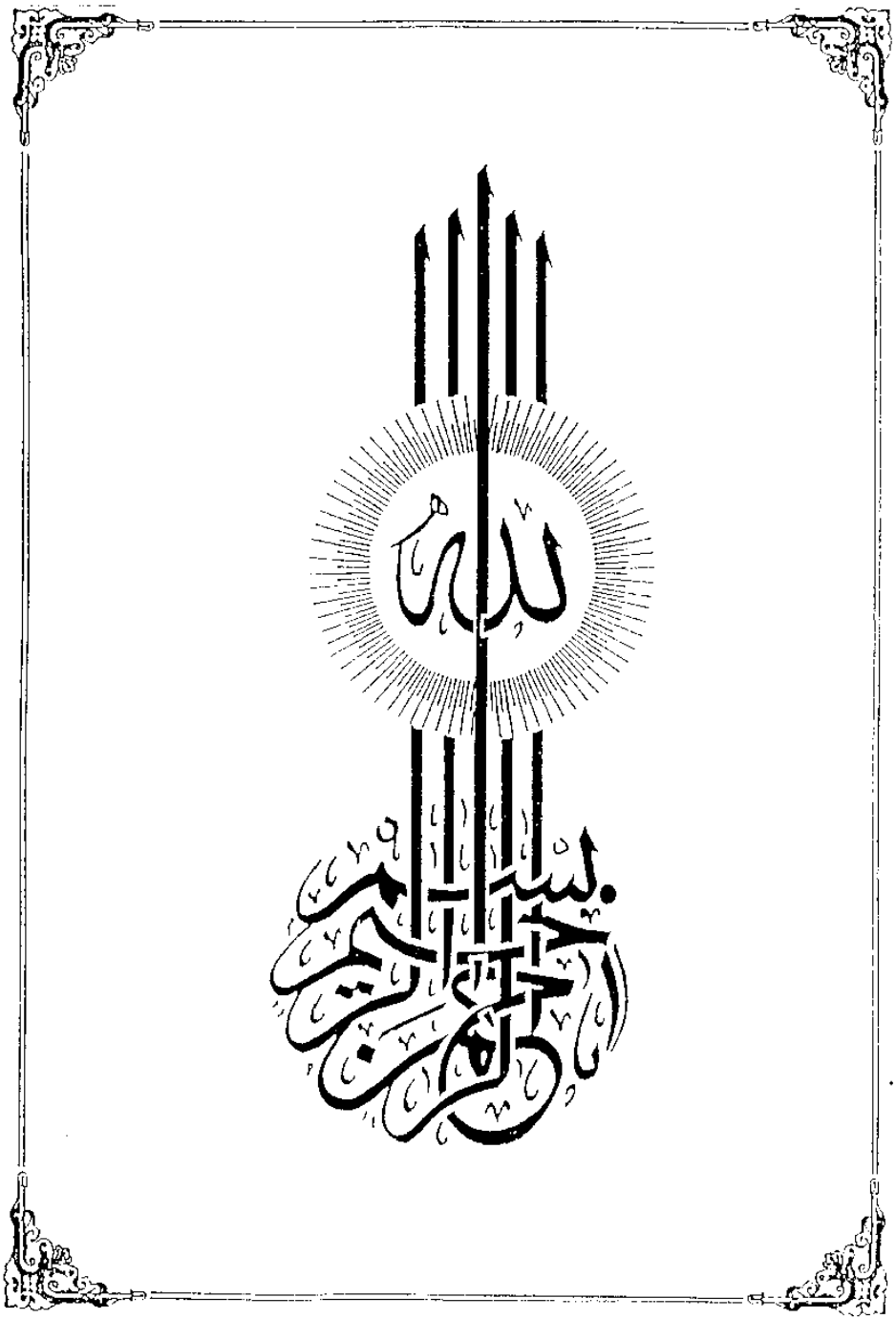

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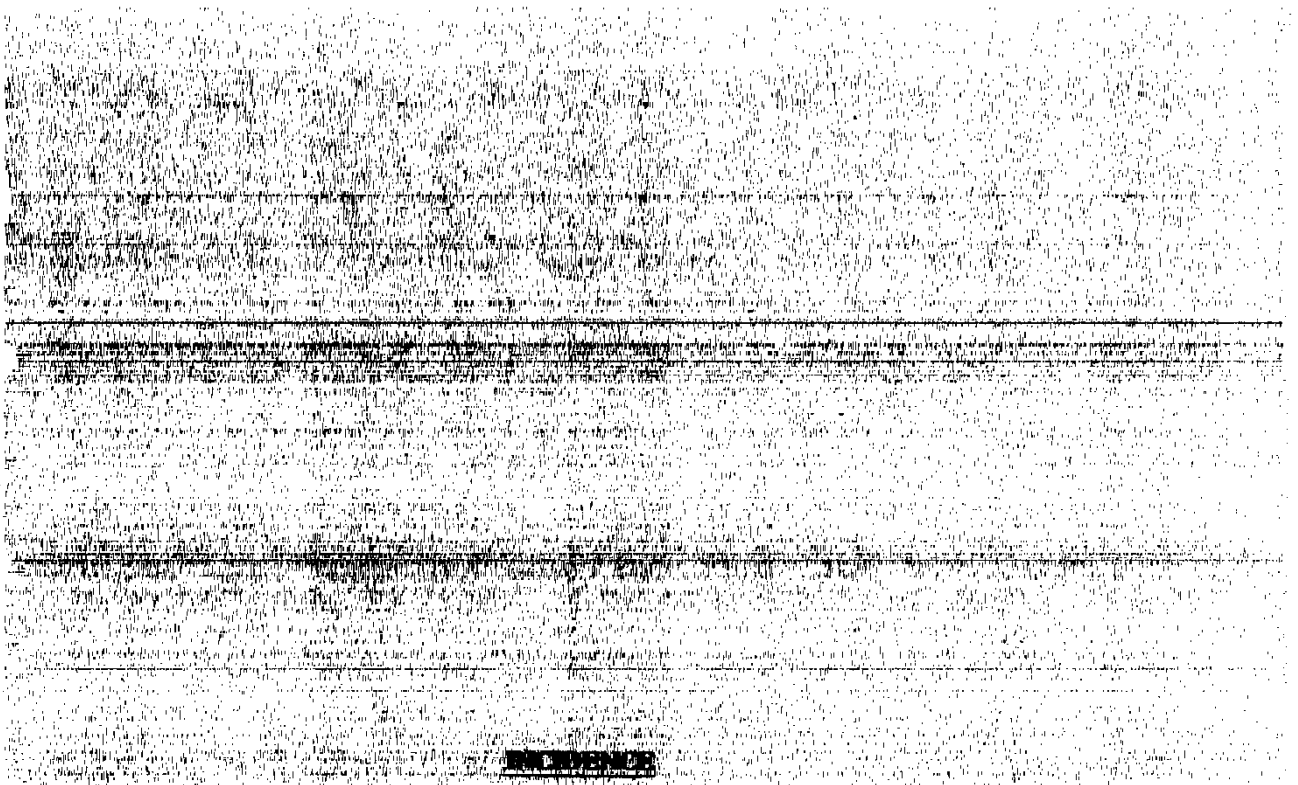
INTRODUCTION

INTRODUCTION :

The undescended testis is a common problem facing most of the pediatric surgeons all over the world. Its incidence is about 26.5% in prematures and 5.4% in the full term infants.

Recently, a lot of progress has been achieved in this subject. The aim of this essay is to discuss this problem and the recent advances about it.



INCIDENCE OF CRYPTORCHIDISM

Cryptorchidism represents the most common disorder of sexual differentiation in man and in itself is one of the most prevalent disorders of childhood. Its frequency is higher in prematures than in term infants. In a combined series reported by Benson and Lotfi (1967) the incidence was 26.5% in prematures and 3.2% in fullterm infants also this was found by Scorer and Farrington 1975. As shown in Table (1).

Table (1) : Incidence of Cryptorchidism from birth to adulthood.

AGE	INCIDENCE %
* Preterm infant	30.3
* Full term infant	3.4
* One year	0.8
* Adulthood	0.8

(Scorer and Farington , 1972).

In addition, the smaller the birth weight of the infant, the greater the incidence of cryptorchidism (Scorer , 1956) . At the end of the first year of life, approximately 5.4% of prematures and 0.5% of fullterm infants have undescended testes.

Scorer (1956) noted that two thirds of the undescended testes in fullterm males descend into a normal position in the scrotum by 6 weeks of age (3 months for prematures). If descent did n't occur by this time, the testis never descended completely into the scrotum, remaining smaller than the contralaterally descended gonad. This occur by this age becuase of hormonal factors, specifically the hypothalamic pituitary-testicular axis. It has been demonstrated that the plasma level of testosterone reaches almost pubertal levels some time within the first three months of age gradually decreases to prepubertal levels there after (Forest et al., 1974 , Gendrel, et al., 1980).

In surgical explorations for cryptorchidism, absence of one or both testes may be encountered in 3 to 5% of cases (Goldberg, 1974) . The incidence of bilateral undescent has been reported from 10-25% of cases (Gross and Jewett 1956 - Snyder and Chaffin 1955) and in 3% of these cases one or both of testes is absent (Scorer and Farrington 1971).

There also appears to be a familial incidence in certain patients , approximately 14% of patients come from families in which other members have the same condition (brothers, fathers and sons) (Walsh, 1984).

The right side is undescended slightly more often (53%-58%) than the left (42- 47%).(Walsh, 1984).

EMBRYOLOGY

EMBRYOLOGY OF TESTICULAR DESCENT AND MALDESCENT

There are few fields in embryology in which biologic misconceptions and frankly inaccurate reporting of previous work have led to such bizarre views as in the discriptions of testicular descent and maldescent.

Recently , several studies have yielded a better understanding of the embryology of testicular descent.

***Early embryology :-**

By the sixth week of gestation, primordial germ cells migrate along the dorsal mesentry of the hindgut to reach the genital ridges which are the medial fold of the urogenital ridge formed at the 4th gestational week as invagination of the posterior wall of the coelomic cavity after degeneration of the pronephros.

By 7th week, under the influence of the H-Y antigen, the indifferent gonad differentiates into foetal testis. (Jordon and Ruddle 1981, Wachtel et al., 1975) . During the 8th week, the foetal testis becomes hormonally active. Müllerian-inhibiting substance is secreted by the fetal Sertoli cells and causes regression of the Müllerian ducts . (Josso 1972, 1973).

At 10 to 11 weeks ' gestation, testosterone produced by the Leydig cells stimulates development of the Wolffian duct to form the epididymis, vas deferens, and seminal vesicle. (Siiteri and Wilson 1974). Differentiation of the external genitalia takes place between 10 and 15 weeks and is dependent on the presence of local 5-alpha-reductase, which converts testasterone to dihydrotestosterone (Walsh et al.,1974).

During the 5th week of gestation, the gubernaculum forms from a band of mesenchyme attached to the caudal end of the Wolffian duct and gonad, extending through the abdominal wall musculature to the genital swellings, which will develop into the scrotum (Backhouse 1981). During the process of ascent of the kidney and elongation of the embryo, the testis remains anchored at the internal inguinal ring by the gubernaculum (Backhouse 1964 and Steinhardt et al., 1985).

In the 3rd month, the processus vaginalis forms as a hernial sac through the weakness in the abdominal wall adjacent to the gubernaculum and gradually extends into the scrotum (Kiesewetter et al., 1969 , Wyndham 1942) . The process of testicular descent then remains dormant until the 7th month of gestation, at which time the gubernaculum, which is a gelatinous

cord filled with hyaluronic acid, increases in size, distending the inguinal canal and the scrotum. The testis then descends very rapidly through the inguinal canal into the scrotum. (Backhouse, 1964; Wyndham 1942). The cauda epididymis, attached to the gubernaculum, precedes the testis in its descent into the scrotum. Following descent , the gubernaculum persists as a small fibrous band at the lower pole of the testis, the gubernacular ligament. Normally the processus vaginalis is compeltely obliterated prior to birth.

***Mechanisms of testicular descent :**

The embryonic development of the testis may be devided into three phases: intra-abdominal (1-7 months), canalicular (7-8 months), and scrotal (8-9 months). The descent of the testis is attributed to the three combined forces of intra-abdominal pressure, intramuscular pressure due to contraction of the muscles draped around the inguinal canal, and the guidance and active contraction of the gubernaculum (Sonneland 1925).

the left testis is belived slightly to precede the right in its decent, which possibly accounts for the fact the unilateral undescent is more frquent on the right (Snyder and Greaney , 1969) .

The hypothalamus produces (GnRH) , which stimulates pituitary production of the gonadotropins (LH) and (FSH). (LH) stilulates the Leydig cells in the testis to produce testosterone, and (FSH) appears to increase the number of (LH) receptors on the Leydig cell member. Although, testosterone is capable of inducing testicular descent experimentally (Hamilton and Hubert 1938) , descent is regulated primerly by dihydrotestosterone (Rajfer 1982) as shown in fig. 1).