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Editorial

Mechanism of Testicular Descent

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Since its discovery about two hundred years ago the mechanism of testicular descent represents an enigma. In connection with this concept, the study on the influence of epididymal atrophy on the development and maturation of the testis undertaken by a highly respected group of urologists from The Urological School at John Hopkins Hospital in Baltimore, Maryland (in this issue) is an important contribution to the examination of the secondary changes in the postpubertal testis and particularly to the study of the mechanisms involved in testicular descent. Other recent studies with respect to testicular descent favour the epididymis as a guiding force in testicular descent [1–5]. According to Bedford, there is no biological need for the testis to descend [1] and, moreover, he maintains that in some mammals the testes passively accompany epididymal descent, while in others only descent of the epididymis occurs [1]. Relative to this concept, it should be noted that Hadžiselimović et al. [2] experimentally proved the importance of the epididymis in testicular descent. Furthermore, all newborns with cryptorchidism were shown to have short and underdeveloped epididymides [6, 7]. This underdevelopment was shown to be due to a hypothalamus-pituitary-gonadal deficiency already occurring during intrauterine life [7, 8]. In addition it was pointed out that GnRH treatment by itself is successful in approximately sixty percent of both cryptorchid boys and rats [6, 7]. At the termination of treatment, the most prominent changes observable were the normalisation of the length and structure of the epididymis [6–8]. Testicular descent took place not because of an increase in intraabdominal pressure but as a result of increased intratesticular testosterone [6–8].

In their study published in the current issue McCullough et al. [15] challenge the aforementioned observations and in particular they found that the testes descend unaided by the epididymis.

Since the conclusions of McCullough et al. are at variance with the findings reviewed here, it is important that interested readers be aware of other observations in this area. Concordant with the distinguished urologist from The Brady Urological Institute, the editor also found that twelve percent of adult ACI rats had a complete lack of epididymis (Fig. 1A). However, contrary to the findings of the above group, it was noticed that significantly lower incidences of a complete epididymal defect occurred in newborn ACI rats than in adult rats (Table 1). Therefore,

Table 1. Occurrence of complete defect in newborn versus adult ACI rats

	totally lacking epididymis (including caput!)	epididymis	total
Number of newborn ACI rats	2	77	79
Number of adult ACI rats	12	88	100

X^2 $p < 2\%$

Table 2. The vertebro-gonadal distance in relation to the epididymal defect found in 59 gonads of newborn ACI rats (horizontal plane)

	Percent of distribution	I ovarial distance (⩽ 2 mm)	II (>2<2.5 mm)	III ventral abdominal wall (2.5–6 mm)
Complete lack of epididymis	1/59 2%	1	0	0
Partial epididymal defect	6/59 10%	4	2	0
Complete epididymis	52/59 88%	7	15	30

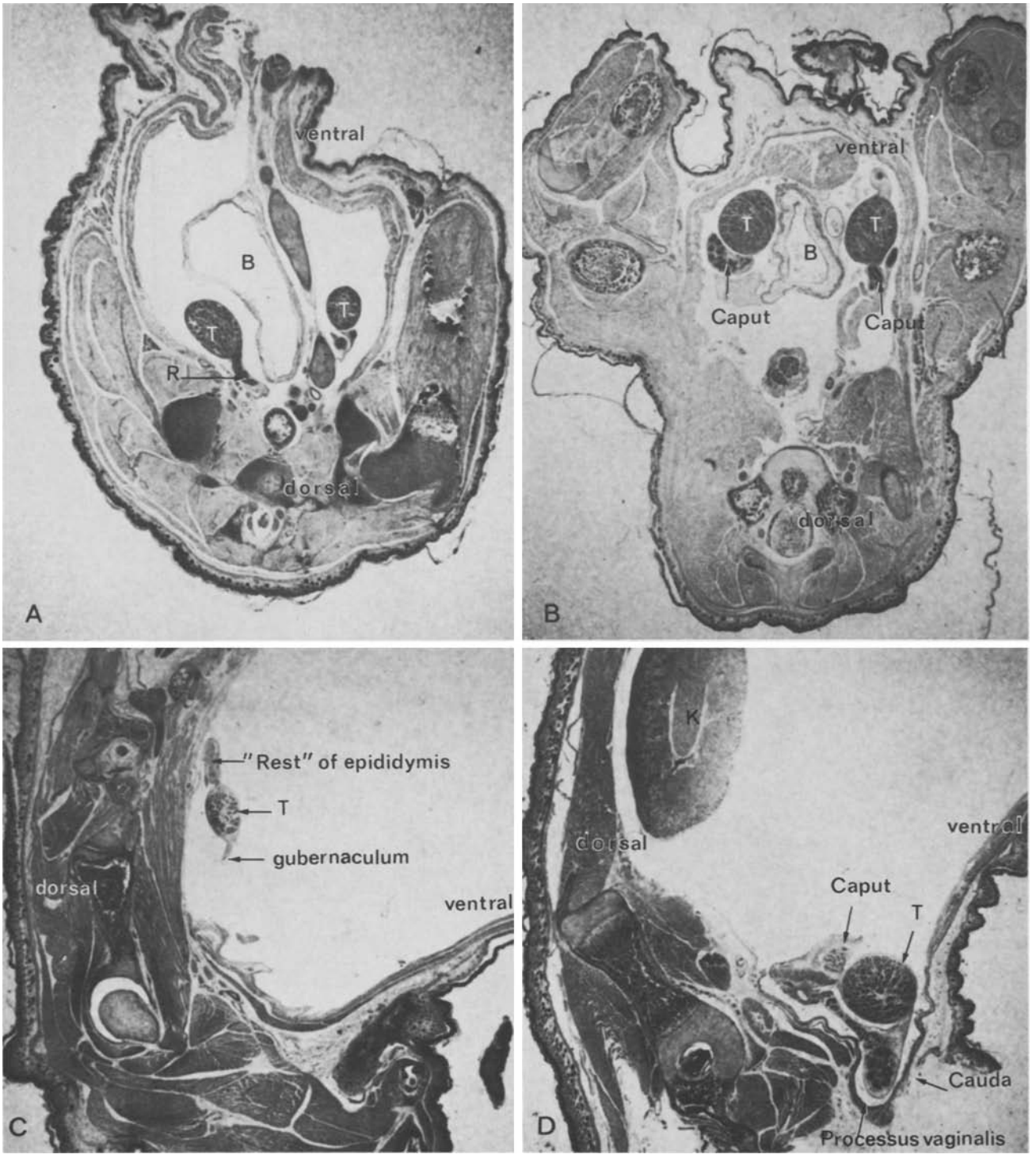

Fig. 1 A-D. Horizontal section (**A**) of the abdomen of a newborn ACI rat without epididymis on the left (↑) and partial defect on the right side. *Note:* testes (*T*) are cryptorchid and situated at dorsal abdominal wall. **B** Horizontal plane of low abdominal cavity of newborn ACI rat with complete epididymis. *Note:* the transabdominal movement of the testes (*T*) is terminated and the gonads are situated at ventral abdominal wall in the proximity of bladder neck (*B*). **C** Cryptorchid newborn ACI rat totally lacking epididymis. Only underdeveloped epididymal stroma (↑) and gubernaculum (↑) inserting directly in atrophic testis (*T*) are recognizable. **D** Sagittal plane: Newborn ACI rat with epididymis taking the testis (*T*) down into the open processus vaginalis (↑). The transabdominal movement is terminated

Table 3. Observations on 17 adult ACI rats with epididymal defect

	Epididymal defect	Complete epididymis	total
Cryptorchid testes	14	2	16
Descended testes	7	11	18

$p < 0.5\%$ Fischer exact test (two-sided)

an early embryological mesonephric ductal arrest cannot be solely responsible for agenesis of the epididymis (Table 1–3, Fig. 1). Thus, the above mentioned difference between newborn and adult ACI rats implies that secondary postnatal changes occur and culminate in the total disappearance of the epididymis. Of special interest is that the degree of transabdominal movement of the testis is proportional to the degree of epididymal differentiation (Table 2). Newborns completely lacking an epididymis were found to be cryptorchid (Fig. 1) while those with a partial defect of the epididymis had impaired testicular descent (Table 2) [9]. A review of the studies previously published concerning ACI rats indicates that one of the contributing authors to the study under review found that some adult ACI rats had both cryptorchidism and a complete lack of the epididymis [10]. However, the article under discussion states that all rats with a complete absence of the epididymis have their corresponding testis descended in the scrotum. Since these findings differ, further research is needed. In the authors own research, 16 out of 100 adult ACI rats were found to be cryptorchid (Table 3), while nine of these sixteen rats displaying cryptorchidism showed a complete defect of the epididymis. Of the remaining seven rats only two had a complete epididymis. It appears that the cryptorchid sides have significantly higher rates of epididymal defects when compared to the descended side (Table 3). In conclusion it appears that the arrest of epididymal development early in fetal life results in cryptorchidism. This is observable in newborn ACI rats (Fig. 1) and in experimentally induced inhibition of Wolfian duct development either because of Vitamin A deficiency [12], chloraminophen [4] or E_2B [2, 13, 14]. In ACI rats with a complete defect of the epididymis, testicular descent may occur in those cases where the secondary changes of the epididymis develop during postnatal life (i.e. late in the process of testicular descent). Furthermore, due to a direct and wide connection between the scrotum and abdominal cavity in rats, it is even possible that a foreign body (silicone ball) placed into the abdomen (after performing unilateral orchiectomy in the newborn rats) drops into the empty scrotum provided there is sufficient room for "descent" [11].

Currently, we do not understand the exact pathological mechanism by which the epididymis in ACI rats is degenerating but it seems obvious that this process is a continuous one throughout life with different degrees of expressivity. Since our results contrast with the findings of McCullough et al., this should not pose a dilemma for those interested in the mechanism of testicular descent but on the contrary it should act as a stimulus for other groups of researchers to prove or disprove the epididymal linked theory of epididymotesticular descent. Much work needs to be done.

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